

**Bangor University**

## **DOCTOR OF PHILOSOPHY**

### **The use of motor imagery in the treatment of the hemiplegic hand in adults**

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THE USE OF MOTOR IMAGERY IN THE TREATMENT OF THE HEMIPLEGIC  
HAND IN ADULTS

THE USE OF MOTOR IMAGERY IN THE TREATMENT OF THE HEMIPLEGIC  
HAND IN ADULTS

By

Jonathon O'Brien, SROT, M.Sc.

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## Abstract

### Background

Recent work on mental practice using motor imagery (MI) in the treatment of post-stroke motor impairments.

### Aim

To provide evidence regarding effects of MI on hemiplegic intrinsic hand muscles.

### Objectives

Experiment one's objective was to identify if kinaesthetic MI (KMI) activated the hemiplegic lumbrical muscles and reduced overall movement time (MT). Experiment two's objective was to identify if KMI and visual motor imagery (VMI) had different effects on lumbrical activity and MT. Experiment three's objective was to establish if VMI or KMI provoked electromyogram (EMG) activity in the hemiplegic or intact abductor pollicis brevis (APB).

### Design/methodology

Experiment one used optoelectronic motion capture in a single factor independent groups design. Dependent variables (DVs) included joint magnitude, velocity and MT. Experiment two employed optoelectronic capture in a between-within design. DVs included interjoint correlation, joint magnitude and MT. Experiment three used a between-within design, measuring EMG in APB and abductor digiti minimi (ADM). DVs were EMG levels in different conditions.

### Participants

Experiment one involved ten adults with hemiplegia. ( $M_{age} = 74.4$ ,  $SD_{age} = 10.3$ ). Five were male. Experiment two involved 15 adults with hemiplegia ( $M_{age} = 60.6$ ,  $SD_{age} = 12.9$ ). Six were male. Experiment three involved ten adults with hemiplegia ( $M_{age} = 59.8$ ,  $SD_{age} = 10.57$ ). Five were male. It also included ten non-hemiplegic adults ( $M_{age} = 59.8$ ,  $SD_{age} = 9.09$ ). Six were male. All were Colombian.

### Results

In experiment one MT was shorter for KMI ( $p = .031$ ). Experiment two also showed shorter MT for KMI ( $p = .022$ ) and significant negative correlation between two finger joints for VMI ( $p = .01$ ), suggesting improved lumbrical activation.

Experiment three found lower EMG in the hemiplegic APB following KMI ( $p = 0.019$ ), compared with rest.

### **Conclusions**

KMI is more effective in reducing MT and pathologically raised EMG. VMI may improve interjoint coordination.

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**Publications arising from this thesis**

- O'Brien, J., Bracewell, M. & Castillo-Martínez, J.A. (2011). The role of kinaesthetic motor imagery in the promotion of activation in the lumbrical muscles of the hemiparetic hand: a randomised controlled study. *Revista Ciencias de la Salud (Journal of Health Sciences)*, 9, 5-16.
- O'Brien, J. & Bracewell, M. (2010). A brief historical review of motor control theory. *Advances in Clinical Neuroscience and Rehabilitation*, 10, 22-24.

## **Chapter one**

### **General Introduction**

#### **1.1 Process Employed in Literature Search for Chapter One**

##### **1.1.1 Aim and objective of search for chapter one.**

The aim of the literature search for chapter one was to provide references describing the clinical and theoretical background of the thesis. The first objective was to demonstrate that the rehabilitation of the hemiplegic upper limb is problematic in clinical practice. The second objective was to show that the application of motor imagery to the treatment of this condition has been a subject for discussion in scholarly publications. Third, it was desired to show that the motor simulation hypothesis has become a key reference point in scholarly discussions about motor imagery. The final objective was to present a conceptual map of this hypothesis, showing its main component ideas.

##### **1.1.2 A scoping study.**

The literature review for chapter one was based on “scoping” (Arksey & O’Malley, 2005, p. 19). This has been defined as an approach which aims to map a field, rather than being constrained by a limited research question or pre-specified research design criteria (Arksey & O’Malley, 2005). It has been suggested that a scoping study can provide a useful prelude to a systematic literature review. It may, for example, give a broad picture of the range of work which has been done in a field, determine whether a systematic review is feasible and worthwhile, and if there are open questions in the field which might be probed further (Arksey & O’Malley, 2005).

##### **1.1.3 Inclusion and exclusion criteria.**

As a scoping study, the inclusion criteria for chapter one were rather broad. Literature was identified that focused on the rehabilitation of the hemiplegic hand following stroke. Methodology was not taken into account and studies based upon focus group research, predictive modeling and randomised controlled trials (RCTs) were all included. References were also sought on motor imagery. Again, methodology was not taken into account, and brain imaging studies, RCTs, literature reviews and scholarly monographs and multi-author volumes were all included.

All the studies of motor imagery and the rehabilitation of the hemiplegic hand involved adult humans. However, the search was not limited to individuals with stroke, and the participants in some studies were neurologically intact. In addition, the type of outcome measures used in the references was not taken into account. All citations needed to be written in English. Clinical research articles which combined motor imagery with other novel interventions such as brain-computer interfaces were excluded.

Once potentially relevant citations were identified their titles and abstracts were reviewed in greater depth. If it was felt that they met the inclusion criteria, the entire article was accessed.

#### **1.1.4 Critical appraisal of literature.**

In line with the guidelines for scoping studies outlined by Arksey and O'Malley (2005), the methodological quality of the references included in the review was not taken account. A critical appraisal tool was therefore not employed at this point.

#### **1.1.5 Databases and internet sites used.**

The Web of Science database was searched. Specifically, the Expanded Science Citation Index, one component of this database, was used (2001- January 2012). In addition, Google Scholar was searched (all years to January 2012). The NHS Evidence (formerly National Electronic Library for Health) portal was also used to access and search the following databases: MEDLINE (from 1950 to January 2012); the Cumulative Index to Nursing and Allied Health Literature (CINAHL) (from 1981 to January 2012); and PSYCinfo (from 1806 to January 2012). Google Search was also used (November 2007 – January 2012). In addition, the websites of the United Kingdom Stroke Association and the United Kingdom National Audit Office were accessed.

#### **1.1.6 Limits of search.**

Initial searches were limited by topic alone. However, as the review progressed, it was often desired to obtain a specific reference whose frequency of citation suggested was a seminal reference in the field. Such searches were limited by title. In addition, limits specific to particular databases were used. For example,

Science Citation Index Expanded allows results to be filtered by document type and journal category. This is illustrated in table 1:1.

Table 1:1

*Search strategy for Web of Science (Science Citation Index Expanded) from 2001 to January 2012*

Search terms	Database	Search limits		Hits
		Web of Science categories	Document type	
("motor image*" OR "mental image*" OR "mental practi*") AND (Stroke OR hemiplegia OR hemiparesis) AND (hand OR "upper limb" OR "upper extremity")	Science Citation Index Expanded	Rehabilitation Clinical Neurology Behavioural Sciences Neurosciences	Article Review Letter Proceedings Paper	138

### **1.1.7 Review of reference lists.**

Once a reference had been included, its reference list was in turn inspected in order to identify appropriate works. Working through literature review articles on motor imagery in this way revealed that the “motor simulation hypothesis” (Jeannoerod, 2006, p. 133) was a key conceptual point of reference for many researchers. Articles, scholarly monographs and edited volumes which dealt with this subject were therefore identified and their reference lists surveyed in order to identify component concepts contributing to the hypothesis. This process was guided by the idea of the “concept map”, outlined by Hart (1998, p. 155) and identified by that author as a key element of literature review in the social sciences. Hart (1998) suggests that tracing the history of the development of a concept may both deepen understanding and facilitate critique of that concept.

### **1.1.8 Search strategy and search terms used.**

Free text terms used were: motor image\*; mental image\*; mental practi\*; stroke; hemiplegia; hemiparesis; hand; upper limb; upper extremity. The asterix symbol in these terms is a 'wildcard' indicating a term with truncated characters. The presence of wildcards allows for variations in terminology and spelling. The relationship between the terms was specified using Boolean operators. An example of a search conducted in the Web of Science database Expanded Science Citation Index is given in table 1:1.

### **1.1.9 Hand searches.**

The *British Journal of Occupational Therapy* was hand searched each month (November 2007 –January 2012). This was done throughout the thesis and applies to the separate searches for each chapter outlined below.

### **1.1.10 Grey literature.**

Press releases by the United Kingdom Stroke Association were referenced. In addition, National Audit Office reports produced on behalf of the United Kingdom government were used.

### **1.1.11 Critical appraisal tools.**

As shown above, the focus of this section of the thesis was not on the identification of clinical RCTs and the inclusion criteria were deliberately broad. It was therefore not appropriate to employ a critical appraisal tool at this point.

## **1.2 General Introduction and Literature Review**

### **1.2.1 The scope of stroke related problems.**

Stroke is the third biggest killer and the main cause of severe disability in Wales (Stroke Association, 2006). The problem affects many younger people in Wales, with 10,000 strokes a year in those under 55 and 25,000 in those below retirement age (Stroke Association, 2006). The risk of stroke increases with age and therefore as the Welsh population ages the problem is likely to worsen. Across the whole of the United Kingdom there are around 150,000 new cases of stroke each year (Barclay-Goddard, Stevenson, Poluha, & Thalman, 2011). Stroke care in the National Health Service as a whole costs £3 billion per annum, with half of stroke survivors unable to live independently after discharge from hospital (National Audit Office, 2007). It is estimated that the national cost in reduced productivity and disability is



£1.8 billion per annum, with a further £2.4 billion spent on informal care costs (National Audit Office, 2007).

### **1.2.2 Poor recovery of the hand following stroke.**

The motor impairments following stroke are a major cause of disability and evidence suggests that the recovery of the upper limb following stroke is an especially difficult problem. A key problem is hemiplegia, the loss of strength and dexterity mainly affecting one side of the body (Carr & Shepherd). Kwakkel, Kollen, Van der Grond, and Prevo (2003), for example, carried out a 6 month study of 101 stroke patients. The patients followed a programme of intensive physical and occupational therapy. After 6 months the authors found that only 11.6% of patients, measured using the Action Research Arm Test (ARAT), made complete recovery in the upper limb, with 62% failing to recover any dexterity.

Despite the importance of adequate hand function in the promotion of independence following stroke, and the disappointing results outlined above, some writers have argued that the hand and the upper limb generally are often neglected in therapy and that the recovery process is poorly understood (Carr & Shepherd, 1987, 1998; Hemmen & Seelen, 2007). One survey, for example, noted that there is a “paucity of high level” evidence to support any of the dominant approaches used to treat the upper limb and that even experienced therapists have difficulty in clearly articulating their process of clinical decision making in the area (Kuipers, McKenna, & Carls, 2006, p. 108). These accounts seem to be confirmed by one systematic review of the literature which has found little evidence to support any of the several therapeutic approaches for treating the hand that are available in the clinical arena (Langhorne, Couper, & Pollock, 2009). Proponents of different approaches have debated the merits of their preferred methods, and yet evidence to support one or another approach remains scarce (Carr & Shepherd, 1998; Woldag & Hummelsheim, 2002).

### **1.2.3 Motor imagery in the recovery of the hemiplegic hand.**

The technique of mental practice using MI has, however, recently provoked interest and been the subject of several review papers (Jackson, Lafleur, Malouin, Richards, & Doyon, 2001; Mulder, 2007; Nilsen, Gillen, & Gordon, 2010; Schuster et

al. 2011; Sharma, Pomeroy, & Baron, 2006; Zimmerman-Schlatter, Schuster, Puhan, Siekierka, & Steurer, 2008). In addition, an extensive review has recently been undertaken as part of the Cochrane collaboration (Barclay-Goddard et al., 2011). MI is conceived of as a specific case of mental training which focuses on the imagining of human movement (Jackson et al., 2001; Schuster et al., 2011). Authors have described it as a process involving mental representation of a movement without a corresponding motor output (Braun, Beurskins, Borm, Schack, & Wade, 2006; Dickstein & Deutsch, 2007; Mulder, 2007). It has also been stressed that MI is an active, dynamic process, involving a volitional reactivation of a stored motor representation (Sharma et al., 2006; Zimmermann-Schlatter et al., 2008).

#### **1.2.4 MI as a cognitive process.**

Researchers have argued that the internal representation of movement is a cognitive process which takes place within the mechanisms of working memory (Dickstein & Deutsch, 2007; Sharma et al. 2006). The working memory model memory posits the existence of distinct storage sites for visual-spatial and phonological aspects of memory that are controlled by a “central executive” (Baddeley, 2003, p. 829). These storage systems are hypothesized to have very limited capacity. Phonologic information, for example, is thought to decay rapidly, while visual-spatial storage is thought to have a capacity of only three or four objects at a time (Baddeley, 2003). Similarly, the central executive attention system, which is thought to direct attention towards one or the other storage site, is regarded as having a very limited capacity and also being subject to the effects of fatigue (Baddeley, 2003). Crucially for the current discussion, it has been proposed that working memory may also have kinaesthetic and motor aspects (Baddeley, 2003; Jeannerod, 1997; Malouin, Belleville, Richards, Desrosiers, & Doyon, 2004). In addition, the emphasis on the short term nature of information stored in working memory fits well with Jeannerod’s (2006) argument that internal representations of movement are temporary phenomena which are constructed rapidly to meet the needs of a specific task.

As well as drawing on neuropsychological concepts of working memory, authors have also referred to ideas from motor control theory in order to explain why MI may offer an effective treatment option. In particular, reference has been made to

the notion that the patterns of cortical activation triggered by MI may overlap with those which would be involved in the actual planning and execution of movement (Mulder, 2007; Mulder, Zijlstra, Zijlstra, & Hochstenbach, 2003; Stinear, Byblow, Steyvers, Levin, & Swinnen, 2006; Sharma et al., 2006). The work of Jeannerod (2006) is of special importance here. This author has developed a hypothesis of “motor simulation” (Jeannerod, 2006, p.130), which has done much to promote the idea that the interior imaging of a movement has a lot in common with movement planning and execution. The centrality of this concept in discussion about MI in stroke rehabilitation and also the references which will be made to it in this thesis, justify an overview of the development of the key components of Jeannerod’s (2006) idea. This is done in the following section.

### **1.2.5 Development of the motor simulation hypothesis.**

An early exposition appeared in a paper by Decety, Jeannerod, and Prablanc (1989). This study used “mental chronometry”, which in the case of MI research often involves matching the imagined time to perform a movement against its actual execution time (Guillot, Louis, & Collet, 2010, p.118). A close match has been seen as evidence for the existence of shared mechanisms of movement execution and imagination (Jeannerod, 2006). In Decety et al.’s (1989) study twenty healthy individuals were faced with three targets placed at various distances. One group was asked to concentrate on forming a mental image of the target and the route leading to it, while the other group was not. The mental imaging group was also asked to imagine itself walking towards the target. Both groups were then blindfolded and asked to actually walk up to the target. The real and imagined walking conditions were self-timed using a hand held stopwatch. It was found that the actual and imaginary walking times were not significantly different and were closely correlated. It was noted, in addition, that while the mental imagery group did not make any directional errors when walking, the group that had not used imagery consistently veered away from the target. Furthermore, the non-imagery group showed far higher levels of variability in their paths towards the targets. The authors suggested that the mental imagery task may have supported visual memory, explaining the lack of directional error and reduced distance error in the imagery group.

This paper presented an early example of what has emerged as a consistent theme in the literature on MI and stroke rehabilitation: that mentally imaging a movement might promote a similar process of motor learning to that which would normally be associated with physical practice of the skill. Motor learning here is defined as a lasting improvement in the ability to produce a particular skilled movement (Schmidt & Lee, 2005). Furthermore, the finding of similar mental and executed walking times suggested the notion of an overlap of movement planning and movement execution mechanisms which subsequently emerged as a key idea in Jeannerod's (2006) simulation hypothesis.

### 1.2.6 Fitts' Law.

Later work that was to contribute to the development of this body of theory drew on the insights offered through application of Fitts' law to the mental chronometry paradigm. Fitts' law predicts the movement time needed to reach targets of different widths and has provided a keystone for motor control studies for over half a century (Schmidt & Lee, 2005). It has proved useful in MI research and will be referred to in this thesis. It is worthwhile, therefore, to offer a more detailed description of the law.

Fitts (1954) had been concerned to quantify the amount of information required to produce upper limb movements in which the amplitude and accuracy requirements of the task were determined in advance. Here, information is defined in relation to information theory, in which one unit of information is seen as the amount of information needed to halve the amount of uncertainty in a given situation (Schmidt & Lee, 2005). In this method of analysis one unit of information is expressed as a binary digit or *bit* (Schmidt & Lee, 2005). Fitts (1954, p. 266) had calculated an "index of difficulty" ( $I_d$ ) for a movement, which is often given in a modified form from a later paper,

$$I_d = \log_2 \frac{2A}{W} \quad (1) \quad (\text{Fitts \& Peterson, 1964}).$$

In equation one,  $A$  is the amplitude of the movement,  $W$  is the target width and the  $\log_2$  expression enables the result to be expressed in line with the conventions of information theory (Fitts, 1954; Schmidt & Lee, 2005; Slifkin & Grilli, 2004).

Equation one forms the basis for a further equation predicting the movement time (MT) towards a target. This has become known as Fitts' law and has often been expressed using a later variant of Fitts' original equation,

$$MT = a + b [I_d] \quad (2) \text{ (Fitts and Peterson, 1964).}$$

In equation two,  $a$  and  $b$  are constants which represent the y intercept and the slope of the best fit line respectively (Schmidt & Lee, 2005). The key idea of the law is that an increase in the amplitude of the movement, and a decrease in the width of the target, leading to a more difficult overall movement, will result in a longer movement time. The relevance of Fitts' law to the development of the motor simulation hypothesis is discussed below.

### **1.2.7 Motor imagery and Fitts law.**

Decety and Jeannerod (1996) asked fifteen healthy participants to judge MT towards doors of different widths and distances presented within a virtual reality helmet. The participants were asked to form a mental image of the gate and, when the image was switched off, to estimate their imaginary movement time towards and through the gate. The investigators found that the imagined movement time increased with the magnitude of the distance of the gate and the reduction in its width. The authors then went on to calculate  $I_d$  for the movements, using the width and distances of the virtual doors. Following this, they plotted  $I_d$  for each location and width against the reported mental movement times and found that they closely correlated. Thus it seemed that the imagined movement time was a function of  $I_d$ , as would be predicted by Fitts' Law for an executed movement.

The authors commented that one potential criticism of their conclusions could come from the so-called "tacit knowledge" position (Kosslyn, Thompson, & Ganis, 2006, p.61). This maintains that the participants would be simply accessing implicit knowledge about how long such a task should take in the real world, and were actually basing their reported mental movement times on this information. Decety and Jeannerod (1996) countered this possible objection with reference to the curves fitted to the data relating mental movement times, door width and distance. They noted that the distance between the doors increased by the same amount each time and that, when questioned, the participants stated a belief that these distances followed an

“arithmetical law” (Decety & Jeannerod, 1996, p. 132). The researchers therefore argued that, if the participants were indeed making predictions of movement time based on this belief, then the spread of data should have fitted to a linear curve. However, this was not the case, and the data actually fitted to a logarithmic curve, as would be predicted by the Fitts equation. They therefore concluded that there was “performance equivalence between perceptual and imaginal systems” (Decety & Jeannerod, 1996, p. 133). This is a vital component of the simulation hypothesis of MI. It suggests that the imagined movement is constrained by what Jeannerod (2006) has argued are hard wired rules governing motor performance, such as those represented by Fitts’ law, every bit as much as executed movements.

Slifkin and Grilli (2006) explored further the relation of imagined movements and the predictions of Fitts’ equation. They made predictions of  $I_d$  for hitting two targets of equal width placed at varying distances from each other. They then asked participants to make an imagined estimate of the difficulty for another person in hitting the targets. They were given freedom to choose their own scale to estimate the difficulty and only instructed that a higher chosen value would indicate a higher level of predicted difficulty. The researchers found that the participants’ estimates, which were presumed to be made on the basis of imagined movements, closely correlated with the investigators’ predictions from  $I_d$ .

Slifkin and Grilli (2006) conducted a second experiment in which the size of the display was increased. This meant that the absolute magnitude of the targets and the distance between them increased, while the relative values were constant, therefore giving the same  $I_d$ . Again, it was found that the participants’ predictions of difficulty correlated closely with  $I_d$ . A further finding was that a violation of the predictions for MT based on  $I_d$  occurred at either extreme of the task when the participants were predicting movement difficulty. The authors commented that this non-linearity has also been noted in real MT tasks based on Fitts’ Law (Slifkin & Grilli, 2006). They suggested that the participants were likely to have made their difficulty estimates based on a mentally simulated MT. This led them to speculate that, in line with Decety and Jeannerod (1996), imagined movements must have the same rule-based constraints as real movements.

Radulescu, Adam, Fischer, and Pratt (2009) explored earlier findings suggesting that a violation of Fitts' Law occurred when targets were presented together as an array. Here, it had been noted in executed movement that the time towards the final target was shorter than that predicted in Fitts' equation. The authors conducted two experiments with healthy individuals to see if this violation also occurred in imagined movements towards a target. Initially, participants were asked to move their fingers from a plate to a target presented on a computer monitor. The participants were then asked to place their finger on the plate, watch for a go signal, and remove their finger from the plate when they imagined their finger would have reached the presented target destination. The time difference between the appearance of the go signal and the finger leaving the plate was taken as the movement time. Using the real MTs, the authors extrapolated from the data for the targets presented earlier in the array, which did follow Fitts' predictions, to determine MT for the last target. They found that a statistically significant violation of Fitts' law had occurred, with actual movement towards the final target being faster than that predicted (Radulescu et al., 2009). Crucially, they found that this violation also occurred in the participants' imagined MTs. Furthermore, these investigators were able to demonstrate that the violation only occurred when the target was the last in an array, not when the target was furthest from the participant, so that the violation was a function of the relative position of the target. An interesting aspect of this study is that the participants were not aware of these anomalous findings for Fitts' Law based on target location, and could not, therefore, have been making their MT predictions based on tacit, if unconscious, knowledge of the parameters of movement execution (Radulescu et al., 2009). This experiment therefore provided further support for the hypothesis that imagined movements follow at least some of the same rules which govern executed actions.

### **1.2.8 “Enslavement” and “force deficit” in motor imagery.**

The conservation of violations of Fitts' Law in imagined movements seems to provide compelling evidence that imagined movements may share similar constraints as executed actions. Research on a further category of anomalous movement patterns also points towards a similar conclusion. Zatsiorsky, Li and Latash (2000) and Latash

(2008, pp. 132, 135), have examined the phenomenon of “enslavement” and “force deficits” in the movements of the hand. Enslavement describes the process by which one finger moves involuntarily when another finger is voluntarily activated, or alternatively when one finger involuntarily increases force production when force is increased voluntarily in another finger (Latash, 2008). Force deficit refers to the phenomenon in which the peak force produced by all four fingers pressing together is actually less than the peak force produced by a single finger pressing down on its own (Latash, 2008).

Li, Latash, and Zatsiorsky (2004) used Jeannerod’s (2006) hypothesis of motor simulation as a starting point for exploring the phenomena of enslavement and force deficit in finger force production tasks in relation to MI. Force produced in the hand during MI was measured with a series of force sensors. The authors found that MI of the index finger force alone produced an average force equal to 4% of the actual maximal voluntary contraction (MVC) force, while MI of all fingers pressing together produced only an average of only 2.8% of maximal force. These findings suggested that the phenomenon of force deficit, noted in executed movements, was indeed present in imagined movements.

Li et al. (2004) also examined the effect of index finger MI on other, non-specified, fingers. They found that MI of the index led to a force of 2.6N in the non-specified fingers, compared with a force of 1.6N in those fingers in the resting condition. The authors suggested, therefore, that the enslavement phenomenon was also present in MI. Li et al. (2004) related their findings back to the simulation hypothesis and argued they provided evidence of shared mechanisms for imagined and executed movements. A later study conducted along similar lines by Li (2007) has also found evidence for the existence of force deficit and enslavement effects during MI. These studies seem to show that even anomalous findings relating to the timing and force production of executed movements seem to have their homologues in MI. They may present further evidence, therefore, for the hypothesis of motor simulation.

### **1.2.9 Overlapping patterns of cortical activation in MI and executed movement.**

A second major component of the motor simulation hypothesis is evidence



which suggests that the mental representation of an action is associated with activity in similar neural regions which would be involved in the actual performance of the act. Jeannerod and Decety (1995), for example, noted that MI was linked to activity in the pre-frontal cortex, the cerebellum, the basal ganglia and the supplementary motor area (SMA). Decety (1996) was also able to present evidence showing activation of the anterior cingulate gyrus and the bilateral parietal regions during MI of finger movements. In addition, this author showed that the inferior frontal cortex is active on the side contralateral to an imagined hand movement (Decety, 1996). Jeannerod (2001), meanwhile, highlighted findings regarding pre-motor cortical activation during imagined movements, suggesting that this was one of the most salient findings of brain imaging studies of MI. He also referred to data showing extensive regions of activation in the lateral ventral and dorsal aspects of BA 6 during MI of hand movements. This region, he pointed out, was also activated during an observed movement of the hand or while a person is watching an object which can be grasped. He pointed out, furthermore, that the inferior parietal and intraparietal regions were also activated during imagined grasping movements and while looking at a graspable object. In addition, Jeannerod (2001) maintained that imagined movements were linked to activation in the dorsolateral pre-frontal cortex, the orbitofrontal cortex, the cingulate gyrus and BA 44 and 45.

Jeannerod and Decety (1995) also pointed, however, that there were subtle differences in the distribution of neural activation patterns for real and imagined movements. For example, imagined movements led to a more rostral pattern of activity in the SMA than the executed movement. The authors saw this as evidence that this part of the SMA was more implicated in the representation of actions, while the more caudal segment was involved in action execution (Jeannerod & Decety, 1995). Jeannerod (2001) also noted that, while imagined actions had been linked to activation of the primary motor cortex, this might only be about 30% of the level seen in an executed action. Jeannerod (2001) pointed out that other differences in patterns of activation could be seen in the basal ganglia. In this region, MI was linked to increased activation in the head of the caudate nucleus, which is more involved in cognitive functions, while executed movement activated the putamen, which is a part

of the sensorimotor loop (Jeannerod, 2001). Other divergent patterns of activation could be found in the cerebellum, where executed movements were more linked to activation in the anterior region, while imagined movements were associated more with activity in the posterior parts of this structure (Jeannerod, 2001). In subsequent discussion of such findings, Jeannerod (2006) suggested that these divergent patterns of activation might help the imager to distinguish between an imagined and an executed action (Jeannerod, 2006).

The findings reported above regarding the overlapping patterns of activation in executed movements and MI do seem to be supported by more recent work. Szameitat, Shen, and Sterr (2007), for example, used functional magnetic resonance imaging (fMRI) to scan the brains of healthy individuals imaging a series of everyday upper limb movements, including using scissors and buttoning a shirt. In addition, a series of whole body movements, such as dancing and running, were imagined. Whole body movements were associated with activity in the medial primary motor cortex, extending to the SMA, and also more limited activity in the lateral part of the primary motor region. Activation during whole body imagery was also noted in the posterior cingulate and medial frontal gyri (Szameitat et al., 2007). The imagery of upper limb movements was linked to activity in more lateral regions, including the right inferior central and post-central regions. In the left hemisphere, upper limb imagery was associated with activation of a region just below the primary motor hand region. The general pattern was that imagery of whole body movements was connected to activity in the superior and medial parts of the primary motor area, while imagery of the upper limb was linked to activity in the inferior part of that region (Szameitat et al., 2006).

More recently, Fleming, Steinar, and Byblow (2010) used fMRI to explore the role played by the parietal cortex during MI in fifteen healthy individuals. The participants imagined tasks involving the elbow, forearm and hand. It was found that MI was associated with bilateral activation in the inferior and superior parietal lobes, extending medially to the precuneus, and in the precentral gyrus and superior and inferior frontal gyri (Fleming et al., 2010). The supramarginal, angular and superior occipital gyri were only active in the right hemisphere, as were the inferior frontal lobe and the SMA, with no differential left hemisphere activation. The authors stated that

many of these areas would also be activated in execution of upper limb movements, albeit with slightly different patterns of activity. In addition, they pointed to evidence that the superior parietal lobes have been linked to spatial working memory processes, lending support to the hypothesis that MI engages working memory mechanisms (Fleming et al., 2010). The authors also speculated that activation in the inferior frontal region may be linked to the co-activation of inhibitory processes along with MI. It will be recalled that a key component of the definition of MI is the notion of a simultaneous inhibition of the motor command, blocking any movement taking place, an idea that has also been incorporated into Jeannerod's (2006) hypothesis of simulation.

Fleming et al. (2010) conducted a further study in which sham and genuine TMS were delivered at random over the superior parietal lobes during MI of a sequence of upper limb movements. The participants were required to actually place their upper limbs at the final resting position specified in the MI sequence within two seconds. The authors found that the accuracy of the final resting position was significantly lower during the real as opposed to the sham TMS and suggested that TMS was likely to have disrupted the formation of an accurate motor image, perhaps by interfering with working memory processes. Fleming et al. (2010) explicitly related their findings to the clinical field, suggesting that patients with damage to the parietal lobes may have difficulties performing MI and may not be able to benefit fully from its use. Overall, this study provides further evidence both for the working memory component of MI and also for the overlapping patterns of neural activation for real and imagined movements posited by the motor simulation hypothesis.

#### **1.2.10 Vegetative states and MI.**

A further component of the simulation hypotheses was based on evidence from work by Decety, Jeannerod, Germain, and Pastene (1991). These authors compared respiration and heart rate in healthy participants when using a treadmill and during imagery of treadmill use. It was found that heart rate increased proportionally with the imagined speed of walking in the treadmill. Similarly, it was found that total ventilation rates increased in line with imagined walking speed. The uptake of oxygen actually dropped, however, during mental imagery of the task. The authors interpreted

this as indicating that the increased ventilation and heart rates noted during imagery were of predominantly central origin, as they were not linked to heavier metabolic demands on the system (Decety et al., 1991). This suggested, furthermore, that any motor output which might follow from the imagined movements must have been inhibited. The authors concluded that the evidence was supportive of the hypothesis that motor planning and execution shared similar neural mechanisms.

### **1.2.11 “S-states” and MI.**

In 2001 Jeannerod published a review in which he described in more theoretical detail the notion of motor simulation. He argued that all actions had both an executed, overt stage, and also a pre-execution covert phase. He commented that the covert phase was “a representation of the future, which includes the goal of the action, the means to reach it, and its consequences on the organism and the external world” (Jeannerod, 2001, p. S103). In this paper he introduced a concept of ‘S-states’, mental states in which an imagined, non-executed action could be shown to simulate an executed action (Jeannerod, 2001, p.S104). S-states could include actions which were intended but not executed, or actions that were simply imagined. They might also involve the visual inspection of an object which could be grasped or the observation of another performing an act. The category might also apply to dreaming or altered states of consciousness.

A key element in Jeannerod’s (2001) argument was that there exists a mechanism of inhibition during such a state which prevented the imagined action triggering a motor response. He maintained that the evidence of involvement of the primary motor cortex (M1) in S-states meant that the inhibition must occur elsewhere in the central nervous system (CNS), possibly at the level of the brain stem or spine. He suggested that there were two, not necessarily mutually exclusive, candidate mechanisms of inhibition. First, S-states might only cause a subliminal activation, which would therefore preclude a motor response. Second, there might be a corollary inhibition signal at the same time as the below threshold motor command is issued. Jeannerod (2001) argued that the evidence was not conclusive, noting that some researchers had found evidence of increased spinal reflexes in imagined movement, while others note a suppression of reflexes or a lack of change. He therefore remained

cautious in his suggestion that there may be a simultaneous subliminal action command alongside an inhibitory signal (Jeannerod, 2001).

In this paper Jeannerod (2001) also put forward an argument suggesting that the cognitive act of putting the internal representation into a “motor format” led to the patterns of overlapping neural activation which were noted above (p.108). According to Kosslyn et al. (2006), the question of formatting in mental imagery theory is centred on the code in which information is conveyed, making that information “explicit and accessible” (p. 8). In Jeannerod’s (2006) view, therefore, this motor formatting makes the representation feel more akin to an executed action.

In one sense some of the ideas outlined by Jeannerod (2001) are not new. Several authors have developed similar ideas over many years. Since some of these authors and concepts will be referred to in this thesis in relation to MI, it may be helpful to outline some of them briefly here.

#### **1.2.12 Woodworth and Bernstein’s early contribution.**

Woodworth is regarded as a pioneer in the study of voluntary movement, and an early advocate of the view that upper limb movements can be divided into two phases, an “initial impulse” and a period of “current control” (Jeannerod, 1988; Schmidt & Lee, 2005; Woodworth, 1899, p. 42). In this model, while current control was based on visual or muscular feedback from the moving limb, the initial impulse was seen as being centrally directed and included the command to act, the predetermined pattern of successive innervations of appropriate muscles and a directive specifying the time to stop the movement (Woodworth, 1899). The work of Bernstein (1967), has also been regarded as seminal (Jeannerod, 1988), and contains some echoes of Woodworth’s earlier contribution. Like Woodworth, Bernstein (1967) highlighted the role of a central guiding “engram” or “motor image” which contained in advance “the entire scheme of the movement as it is expanded in time” (p. 39). It can be seen that both Woodworth’s (1899) and Bernstein’s (1967) formulations are actually very close to Jeannerod’s (2001) concept of a covert stage of action.

#### **1.2.13 MI and the notion of efference copy.**

Following the Second World War there developed a growing preoccupation among motor control researchers with questions of human-machine interaction. This

led to a new discipline, cybernetics, which focused on the study of communication and control systems (Wiener, 1948). This new emphasis led motor control theorists to draw on analogies from engineering in order to expand their understanding of the guiding processes in human movement. The work of Fitts (1954), already discussed above, was one product of this tendency. Another key concept, which has been incorporated by Jeannerod (2006) into his hypothesis of motor simulation, was developed by Von Holst (1954). This author was concerned to identify mechanisms by which a visual signal produced by moving the eyeball with an external force could be distinguished from a visual signal produced by an active eye movement. He wished to understand how, in the case of the passive movement, the visual world will apparently shift, while in the case of an active movement it will remain static. His solution involved the proposition that when a motor command was issued within the CNS, then a replica, or efference copy, was also issued for storage in the CNS. An active movement would generate sensory feedback, or re-afference, which would cancel the efference copy. The visual world would then remain still. A passive movement, however, would produce feedback, labelled ex-afference, which produced the illusion of the visual world changing position. This concept of efference copy has formed a central part of subsequent conceptualizations of the processes of pre-execution motor planning, and has informed Jeannerod's (2006) motor simulation concept of MI.

#### **1.2.14 MI and motor program concepts.**

Another key idea which has shaped contemporary theories of MI is the notion of the motor program. One of the most widely discussed formulations of this concept came in a paper by Keele (1968), who described it as "a set of muscle commands that are structured before a movement sequence begins" (p. 387). In addition, he argued that the motor program could be activated in short term memory systems without a corresponding movement being executed.

An influential development of the motor program concept was put later put forward by Schmidt (1975). This author suggested that earlier motor program models had relied on the notion of an individual program for each separate movement. Schmidt (1975) argued that this was unlikely due to the sheer volume of cerebral storage capacity this would imply. This author then proposed the notion of a

“generalised motor program” (Schmidt, 1975, p.232). The idea here was that there was a central program for a class of movements. Schmidt (1975) used the example of a pitch in baseball as a possible category of movement which could be governed by such a general program. The central program of the pitching movement would be modified by “response specifications” such as “throw slowly” or “throw fast” (Schmidt, 1975, p. 232).

Schmidt (1975, p. 234) also put forward a concept of motor “schema”. The schemas were abstract representations of classes of movements built up from experience of those movement categories in the past. The schema would contain information about the state of the neuromuscular system and the environment prior to movement commencement. The schema would also contain the appropriate response specifications, as described above, as well as information about the predicted sensory state of the body following the movement. Finally, the schema would contain information regarding how far the desired movement goal was actually met (Schmidt, 1975). Over repeated movements the relationship between these four sources of information would be combined to produce stable schema for that particular class of movements.

Schmidt (1975) also placed memory at the centre of his theory, as schema activation would depend upon recall and recognition memory. It is interesting to note, in addition, that later developments of the working memory model also posited the activation of motor schema as one component of the central executive control mechanism (Baddeley, 2003). Furthermore, Schmidt (1975) recognised the importance of efference copy. This would be a stored central record of the motor program which had been initiated, so that the actor would know that any sensory feedback resulted from an active rather than a passive movement. The efference copy would also allow the feeding forward of the predicted sensory result of the movement, drawn from the schema, which would allow error detection to take place (Schmidt, 1975). The proposed mechanism of such feed forward control would be via the motor program sending a signal to the muscle spindles. Interestingly, this has also recently been suggested as a proposed mechanism for the efficacy of MI training in stroke, as will be seen in this thesis (Rodrigues et al., 2010).

### **1.2.15 MI and internal model approaches.**

The most recent development of the concepts of efference copy and the motor program, which have also been incorporated into Jeannerod's (2006) simulation hypothesis, is the notion of the "internal forward dynamic model" (Wolpert, Ghahramani, & Flanagan, 2001, p. 488). According to this analysis, the motor commands are fed forward and used to predict the sensory consequences of a movement (Blakemore, Wolpert, & Frith, 2000). A reverse process is also posited in which the desired sensory result is translated into the motor command which would produce that result (Wolpert et al., 2001). The internal model is viewed as a "motor primitive" upon which more complex and finely tuned movements can be built (Wolpert et al., 2001, p.492). This view seems close to the notion of the motor schema as expounded by Schmidt (1975). In addition, Wolpert, Ghahramani, and Jordan (1995) and Ramnani (2006) explicitly identified the feed-forward motor outflow with efference copy. Jeannerod (2006) has also built on this idea, suggesting that a motor representation is distributed from the motor cortex to other cerebral regions and that this forms the basis for the efference copy, allowing the feed-forward component of the internal model to operate.

Ramnani (2006) has argued that the cerebellum is a feasible location in which forward models might be stored and he argues that the loop connecting the cortex, pons and cerebellum is likely to play a major role in the shuttling of forward models between the cerebellum and the cortex. In Ramnani's (2006) view the cerebellum would therefore also be central in the process of movement simulation. It will be recalled that the cerebellum has been found to be active during MI and that it also has extensive connections to the pre-frontal cortices, which have also been found to be extensively activated during MI (Jeannerod, 2001; Ramnani, 2006).

Wolpert et al. (2001) suggested that the internal model might provide the basis for mental training, a point which shows how the concept of the internal model relates directly to this discussion on MI. Furthermore, Slifkin and Grilli (2006), in their work on predicted movement times in MI, have also argued that these predictions must be based on the generation of an internal model of the movement which is then fed forward. Jeannerod (2006) meanwhile, has also suggested that the emulation of the



executed action within the internal model may be involved in mental imaging, commenting that “the idea of an emulator is close to what one would expect from a mechanism accounting for representation of an action” (p. 20).

#### **1.2.16 Mirror neurons and motor simulation.**

Jeannerod’s (2006) hypothesis of motor simulation also builds on evidence about a category of neurons that were originally an accidental discovery reported by di Pellegrino, Fadiga, Fogassi, Gallese, and Rizzolatti (1992). These authors found that neurons in the F5 inferior pre-motor region of the macaque were active both during goal directed hand movements on an object performed by the animal and when observing one of the experimenters performing the same class of movement. Movements towards an item of food, or simple presentation of an item of food did not have the same effect. These became known as mirror neurons, and are now thought to exist in human beings (Iacoboni, 2005). Evidence suggests that they are located in pars opercularis of BA 44 and BA 45 and 47, the SMA, the ventral and dorsal parts of area 6, the precentral gyrus and also in the intraparietal sulcus and the angular and supramarginal gyri (Iacoboni, 2005; Iacoboni & Dapretto, 2006; Jeannerod, 2006; Rizzolatti & Fabbri-Destro, 2009). They have been found to respond more intensely when a person observes an action which they themselves can perform and have been posited as playing a role in learning through observation (Iacoboni & Dapretto, 2006; Rizzolatti & Fabbri-Destro, 2009). A central point in relation to MI is that there is an overlap in the regions activated during MI practice and with mirror neuron areas in BA 44, 45 and 47 (Jeannerod, 2006). There may, therefore, be similar action representation processes involved in mirror neuron activity and MI. As Jeannerod (1997) noted, the fact that these inferior frontal neurons play a role in the internal representation of actions to be executed by the self and observed actions performed by others indicates that “intending, imagining, observing/imitating and performing an action share common structural and functional mechanisms” (p. 191). Jeannerod (2006) has also pointed to tentative evidence that mirror neurons might be active when imaging or planning movements.

#### **1.2.17 MI may have a rapid effect on specific muscle groups.**

Jeannerod (2006) suggested that the motor simulation process “rehearses the

short-term, fast and automatic unfolding of a movement” (p. 140). On this basis, it might be reasonably predicted that the effects of MI training may influence quite specific patterns of muscle activation related to the imagined movement. It might also be concluded that any effects flowing from MI treatment would take place rather quickly. These two hypotheses are explored further below.

Researchers deploying brain imaging technology have certainly been able to show very rapid results using only short sessions of MI practice. In Szameitat et al.’s (2007) study, for example, the details of which were discussed above, the actual imaging period was only 24s, repeated over 14 cycles. In addition, Guillot et al. (2009) were able to show fairly distinct patterns of cortical activation in healthy individuals using MI in the visual and kinaesthetic sensory modalities following just four sessions of MI practice lasting only 30 s each.

Studies using transcranial magnetic stimulation (TMS) have shown similarly rapid results. Cicinelli et al. (2006), for example, used this technique to measure the area of cortical excitability for a single intrinsic hand muscle, abductor digiti minimi (ADM), during MI in a group of 17 people with stroke. This was done by measuring motor evoked potentials (MEPs), which are the EMG signals recorded from a target muscle during TMS (Kamen & Gabriel, 2010). An area of cortical excitability was defined by Cicinelli et al. (2006) as one in which TMS provoked two clear MEPs of 50 microvolts ( $\mu\text{V}$ ). The authors do not specify the exact time spent on producing MI. However, the patients were only involved in a single imaging session and each burst of imagery was performed at a rate of three cycles per second.

Cicinelli et al. (2006) found that, even with this short intervention, the MI condition was associated with a highly significant increase in the size of the cortical representation of ADM in the hemisphere affected by stroke, as measured by the number of excitable regions. Indeed, the authors found that the imbalance for the ADM region excitability between the affected and unaffected hemispheres noted during the baseline resting condition more or less disappeared during MI as the excitability of the affected hemisphere began to match that of the non-affected (Cicinelli et al., 2006). This might suggest that MI somehow ‘normalised’ the patterns of cortical activation. In addition, these authors did not find any significant increase in

the level of cortical excitability for a control muscle which was not the focus of MI, pointing to a very specific pattern of raised excitability that was related to the content of the mental training. Interestingly, it was also found that the increase in excitability was related to the expansion of the cortical representation of the ADM, rather than an increase in the intensity of signalling in the already existing map of the muscle as measured at resting baseline. Cicinelli et al. (2006) suggest, therefore, that their MI task worked by increasing the efficiency of the synaptic connections in the region surrounding the map of ADM in the damaged hemisphere. Overall, this study does seem to show that a short, one-off, session of MI might be adequate to promote muscle specific increases in cortical excitability in stroke patients.

Li et al. (2004), in a study already mentioned above, also used TMS to measure cortical excitability during MI of the hand. In this study, TMS was used to measure the resting motor threshold (RMT) of the flexor digitorum superficialis (FDS). RMT was defined by the authors as the stimulus necessary to produce an MEP of at least 50 $\mu$ v for a minimum of 3 out of 6 MEPs in both the MI and resting conditions. The healthy participants in Li et al.'s (2004) experiment were instructed to rest, to use MI of the index finger pressing down on its own, or use MI of all the fingers pressing down together. In the resting condition mean RMT was found to be 40.8% of the maximum output of the TMS stimulator, while during the MI condition for the index finger it was found to be 36.6% of output and for the MI task for all four fingers it was found to be 37.4%. These differences were found to be highly significant statistically. They show that MI led to a marked reduction in RMT and therefore an increase in cortical excitability. Furthermore, Li et al., (2004) were able to show a significantly higher level force production in the hand when MI was compared with the resting period.

Li (2007) used a very similar approach to that described by Li et al., (2004). This author also found a highly significant reduction in RMT during MI of the hand when compared with a resting condition. He also noted a significant increase in force production during MI. Overall, these authors (Li et al., 2004; Li, 2007) suggest that their findings indicate an increase in the excitability of the cortical-spinal system in the MI conditions. In neither study are exact durations given for the MI training. However, it is clear from the authors' description that each condition lasted for a few

seconds only and was only repeated five times (Li et al., 2004; Li, 2007). The results presented seem to suggest, then, that in healthy individuals at least, short sessions of MI training may be sufficient to promote cortical excitability and concomitant increases in force production in an extrinsic hand muscle.

### **1.2.18 MI and motor performance.**

While the studies discussed above seem to show that MI has an immediate effect at the level of cortical activation and excitability, and also on the production of force at the periphery of the motor apparatus, it would still need to be demonstrated that such results might be linked to rapid improvements in motor performance. Cicinelli et al. (2006), for example, point out that the precise link between the increased cortical excitability they noted for the ADM muscle in stroke patients using MI and any putative cortical plasticity which might facilitate actual improvements in movement execution remains an open question. On the other hand, Szameitat et al. (2007) argue that the highly specific patterns of cortical activation that they noted during MI would make it an effective tool in rehabilitation, as it may help in “fine-tuning or strengthening neural pathways” (p. 711).

Some evidence does seem to confirm the hypothesis that short sessions of MI training may have a positive impact on motor performance. Jackson et al. (2001) reviewed evidence, for example, suggesting that a mere ten minutes mental practice can be sufficient to show improvement in a motor skill. Féry (2003), meanwhile, was able to show significant improvements in the performance of a motor task in healthy individuals with only 22 minutes of mental training. Other studies have shown similar positive results with short sessions of MI. Two of these are discussed below.

Mulder et al. (2004) used ten sessions of one minute bursts of MI training, repeated twice over two days, to train 14 healthy individuals to abduct their big toe. It was found that individuals who already had some basic level of competence in this skill showed a significant improvement in performance when compared with a control group who had not undergone any training. These authors also offered an explanation for their results which referred to the idea of “common neural mechanisms” shared by internally represented and executed movements in keeping with the motor simulation hypothesis (Mulder et al., 2004, p. 216).

Malouin et al. (2004) presented results suggesting that such short MI sessions might also promote motor learning in a stroke population. In their study, 12 individuals with stroke used MI training in an effort to reduce the degree of force overload in their hemiplegic lower limb. The participants physically practised one sitting to standing transfer, followed by five mental rehearsals of this action. The precise timing of these sets of treatment is not described. However, given the nature of the task, it is unlikely that they would have lasted more than a few minutes each. Furthermore, the authors do not specify how many repetitions of this cycle took place, although they do explain that this was a one-off session which took place on one day (Malouin et al., 2004). Despite this short intervention, the authors were able to demonstrate significant reductions in the degree of leg overloading in their patient group, which were still present one day after training (Malouin et al., 2004).

The results discussed above do seem to support the prediction that a short, one-off session of MI, might lead to quite rapid changes at the cortical level in both healthy individuals and those with stroke. Furthermore, this might also be linked to equally fast changes in motor performance. In addition, based on the evidence presented, it would seem that such a short one-off session of MI might also promote motor learning in a stroke population, at least for re-training of lower limb function. It does not seem unreasonable, therefore, on the basis of the hypothesis of motor simulation (Jeannerod, 2006), to predict that a one-off MI training session might also have a positive impact on the motor function of the hemiplegic upper limb. This conclusion is discussed in relation to the specific clinical problems which have guided this thesis below.

### **1.3 Key Clinical Problem Motivating the Research**

As shown above, research indicates that the motor recovery of the hand following stroke is poor in the United Kingdom and a number of authors has suggested that mental training using MI might provide an effective treatment option. Some have also attempted to develop protocols for administration of MI based therapy. Often, these have involved programmes of intervention spread out at regular intervals over several weeks. Page et al. (2011), for example, recommended 60 minute practice sessions, three times per week, over ten weeks. However, given the health care contexts in which stroke patients are likely to be encountered, at least in the

United Kingdom, this proposition may be problematic. This is discussed below.

The majority of patients in England, Wales and Northern Ireland is admitted directly to an acute stroke unit (ASU) (Intercollegiate Stroke Working Party, 2010). Mean hospital stay is 19.5 days, with a median stay of nine days (Intercollegiate Stroke Working Party, 2010). Only a short portion of this time is likely to be on an ASU, as patients will generally be transferred to a rehabilitation unit, which may be on another site, once medically stable. It is also known that early therapy intervention is essential to making a good motor recovery (Edmans, 2010; Intercollegiate Stroke Working Party, 2010). Since early occupational therapy input is likely to take place on an ASU, it follows that therapists in these settings will have a comparatively short time period in which to treat patients with hemiplegia affecting the upper limb. Certainly, the lengthy MI protocols outlined by authors such as Page et al. (2011) would be unworkable in such a setting.

If MI is to be used for the treatment of upper limb hemiplegia in the acute stroke patient, then it may be the case that it needs to be delivered in short, possibly one-off sessions. Furthermore, national guidelines currently specify that patients should receive 45 minutes of therapy daily, if appropriate (Intercollegiate Stroke Working Party, 2010). The pressure on occupational therapists' time is great in the acute setting, and MI programmes may therefore also need to be developed in a straightforward and convenient format for delivery by therapy assistants, perhaps as part of group therapy activities on the ward, if such national targets are to be met. This thesis therefore seeks to test whether such short, one-off MI sessions, delivered via a recorded script, can play an effective role in the recovery of the hemiplegic hand.

#### **1.4 Aims, Research Questions and Experimental Hypotheses of the Three Experiments Presented in the Thesis**

Three experiments are presented in the thesis. The aims, along with the research questions and hypotheses for each experiment are outlined below.

##### **1.4.1 Aim of experiment one.**

The aim of experiment one was to identify whether or not a one-off session of MI embedded in a kinaesthetic sensory modality (KMI) affects the performance of the “lumbrical grip” in the hemiplegic hand of adults with stroke. The lumbrical grip is

characterised by the flexion of the metacarpophalangeal joints with the simultaneous flexion of the interphalangeal joints, a movement pattern known to be underpinned by the activation of hand lumbrical muscles (Palastanga, Field & Soames, 2006).

#### **1.4.2 Experiment one research question.**

Does a one-off session of KMI make a difference to the production of a lumbrical grip in the hemiplegic hand of adults following stroke?

#### **1.4.3 Experiment one hypotheses.**

1. The ability to extend the index finger in the hemiplegic hand, as measured by the magnitude of the proximal interphalangeal (PIP) joint angle, while also flexing the MCP joint of the same finger, would be greater in a group which had undergone treatment with KMI.

2. The path of the index finger PIP joint, while grasping an object with the hemiplegic hand, would be smoother in individuals using KMI.

3. The angular velocity of the PIP joint in the hemiplegic hand would be greater in individuals using KMI.

#### **1.4.4 Aim of experiment two.**

The aim of experiment two was to identify if a one-off session of KMI had different effects on the formation of the lumbrical grip in the hemiplegic hand, when compared with MI based in a visual sensory modality (VMI).

#### **1.4.5 Experiment two research question.**

Does a one-off training session using KMI make any difference to the formation of a lumbrical grip in the hemiplegic hand, when compared with VMI?

#### **1.4.6 Experiment two hypotheses.**

1. The use of MI would be associated with improved coordination between the MCP, PIP and distal interphalangeal (DIP) joints, in line with the constraints of the lumbrical grip. Furthermore, the improvement would be greater in the group using KMI, when compared with the group using VMI.

3. There would be greater extension in the PIP and DIP joints and greater flexion in the MCP joints in the groups using MI when performing the lumbrical grip. The magnitude of these measurements would be greater in the group using KMI than that using VMI.

4. The movement time of the hand performing the lumbrical grip would be shorter for the groups using MI, and would be shorter still in the group using KMI.

5. The paths of the MCP, PIP and DIP would be smoother in the MI groups, and still smoother in the KMI group. This was measured using the variability in the angular paths and velocities of the joints and discontinuities in their acceleration profiles.

#### **1.4.7 Aim of experiment three.**

The aim of experiment three was to find out if there was increased peripheral muscular activity in the hemiplegic hand of people with hemiplegia when using KMI and VMI.

#### **1.4.8 Experiment three research question.**

Is there a raised EMG signal during one-off motor imagery practice of thumb abduction in people with hemiplegia affecting the hand following stroke?

#### **1.4.9 Experiment three hypotheses.**

1. Individuals with hemiplegia following stroke would be more likely to show a raised EMG signal than healthy individuals while using MI to imagine movement in the abductor pollicis brevis muscle.

2. KMI would produce this result, while VMI would not.

#### **1.4.10 Coherence between the three experiments.**

A number of coherent themes run through the experiments presented in this thesis. First, all experiments focus on the effects of motor imagery training on specified intrinsic hand muscles. For all three, an effort was made to identify a motor task which was largely underpinned by the activation of these muscles. The focus of the first two experiments is the hand lumbrical muscle and of the third the abductor pollicis brevis.

A second concern was the attempt to identify effects of MI on these intrinsic hand muscles when affected by hemiplegia. As a consequence all of the experiments involved groups of individuals with stroke. In addition, a concern with the pathophysiology of stroke and the way it impacts on motor processes runs through each individual experiment and the thesis as a whole.

All three experiments aimed to produce high resolution measurements in order



to capture subtle changes in patterns of movement and activation in intrinsic hand muscles. All, therefore, employed electronic methods of data capture, followed by digitization and statistical analysis. Experiment three also made use of electrogoniometry to control for superfluous thumb movements. This represents a move away from the use of clinical scales as outcome measures and the control of superfluous movement through human observation which has been a feature of earlier research.

Despite the different kinds of data captured in the three experiments, all three were linked by a concern with variability in patterns of activation. This is because several authors have pointed to increased variability in movement patterns as a key problem following stroke (Cirstea & Levin, 2000; Cirstea, Mitnitski, & Feldman, 2003; Van Vliet & Sheridan, 2007; Nowak et al., 2007). Accordingly, experiments one and two both captured data on the standard deviations (SDs) in joint angular path and discontinuities in the acceleration profiles of the joints. Experiment three focused in part on the SDs in the EMG signal captured from the muscles of interest.

Each of the three experiments places a strong emphasis on the importance of defining the sensory modality of the imagery employed. Indeed, in experiments two and three a comparison of the effects of the different sensory modalities of motor imagery on the intrinsic hand muscles was a central focus.

The three experiments presented in the thesis are linked conceptually by the motor simulation hypothesis. This maintains that MI involves the internal representation of a motor act which activates motor planning mechanisms, and is “highly specific to the action that is represented” (Jeannerod, 2006, p. 131). This idea is what lies behind the assumption in all of the studies that mental training with MI may produce measurable effects on the subsequent execution of an imagined movement in a specific muscle. In addition, the primary hypothesis of experiment three, that motor imagery would not produce peripheral activation in healthy individuals but might in persons with stroke, flowed directly from Jeannerod’s (2006) formulation of the motor simulation position.

The motor simulation hypothesis also proposes that the motor image is an ad hoc representation of a motor act, based on the activation of rules of motor control,

which decays rapidly once activated. This idea was especially attractive for this thesis, where an overarching aim was to test the effects of short, one-off “bursts” of MI. In addition, the hypothesis predicts that similar brain regions are activated by imagery as would be activated during the actual performance of a movement. If this is the case, then motor imagery embedded in different sensory modalities may activate different neural regions and might also produce varied effects on subsequent execution. This underlies the importance of carefully defining the sensory content of the motor imagery in all three experiments. The motor simulation hypothesis, therefore, also supported the careful definition of MI modality in all three experiments.

## Chapter Two

### The Effect of a One-Off Session of Kinaesthetic MI (KMI) Training on a Grip Using the ‘Lumbrical Action’ in Adult Hemiplegia

#### 2.1 Experiment One Research Question

What is the effect of a one-off session of KMI focused on the activation of the hand lumbrical muscles on the kinematics of a grip using the “lumbrical action” in adult hemiplegia?

##### 2.1.1 Experimental Hypotheses

The hypotheses to be tested in the experiment reported in chapter two were:

1. The extension of the index finger will be greater in a group using KMI than in a group using relaxation alone.
2. Overall MT will be significantly shorter for a group using KMI, compared with the relaxation group.
3. There will be a stronger negative correlation between the MCP and PIP joints in the hemiplegic hand of a group using KMI, compared with the individuals using only relaxation.
4. The group using KMI will show a significantly later time to peak PIP angular velocity than the relaxation group.
5. The KMI group will show evidence of a smoother trajectory in the angular acceleration profile of the PIP joint. This will be characterised by significantly fewer movement units, with a movement unit defined as one period of acceleration and one period of deceleration.

#### 2.2 Literature Review

##### 2.2.1 Aim and objectives of literature search for chapter two.

The aim of the literature search for chapter two was to identify evidence for the effectiveness of motor imagery in the rehabilitation of the hemiplegic hand. The objective was to locate randomized controlled trials (RCTs) involving the application of MI to the treatment of the hemiplegic hand or upper limb.

##### 2.2.2 Inclusion and exclusion criteria.

The literature search focused on RCTs appearing in peer reviewed journals. The participants needed to be adult humans with stroke. One study, that of Bovend'Eerd et al. (2010), also included individuals with other CNS pathologies. All studies compared mental practice using MI with a mainstream physiotherapy or occupational therapy intervention. Generally, studies which included additional novel interventions were excluded. However one study, that of Page et al. (2009), which combined MI with constraint induced movement therapy (CIMT), was included. This was because the study followed on from a series of RCTs led by the same author and shared many design features with the earlier studies. All the studies included a component which looked at the effect of MI training on the recovery of function in the upper limb or hand affected by hemiplegia. Only studies published in English were considered.

There was no upper age limit on the participants in the studies, nor were there any exclusion criteria based on gender. Furthermore, there were no limiting criteria based upon time since stroke. Nor were there any exclusions based upon the type of outcome measures used in the studies. Studies which applied MI to other aspects of stroke rehabilitation, such as the treatment of visual-spatial neglect, were not considered. Once studies had been identified which appeared to meet the criteria outlined, the titles and abstracts were surveyed in more detail to determine their suitability.

### **2.2.3 Databases and internet sites used.**

The databases searched were the same as those identified for chapter one, described above. In addition, the Electronic Thesis Online Service (EThOS) was searched (all years – October 2011).

### **2.2.4 Search strategies, search terms and number of hits.**

For MEDLINE, CINAHL and PSYCinfo a combination of free text and medical subject headings (MeSH) was employed. MeSH descriptors are terms used to index biomedical journal articles by the United States National Library of Medicine. For Web of Science free text only was used.

Free text terms included: motor image\*; mental image\*; mental pract\*; mental practi\$e; upper limb; hand; arm. The dollar sign in this list is a wildcard. The free text

terms were 'mapped' onto a thesaurus of MeSH terms and terms which were thought to be appropriate for the search were selected. The MeSH terms included: psychomotor performance; upper extremity; stroke; hemiplegia; paresis. The relationship between the search terms was specified using Boolean operators. The search strategies and search terms for Web of Science and MEDLINE are displayed in tables 2:1 and 2:2. These tables also show the number of hits for each search.

Table 2:1

*Search strategy for Web of Science (Science Citation Index Expanded) from 2001 to January 2012*

Search terms	Search limits				Hits
	Web of Science Categories	Search type	Document type		
("motor image*" OR "mental image*" OR "mental practi\$e") AND ("upper limb" OR "upper extremity" OR hand OR arm) AND ("stroke" OR "cerebrovascular accident")	Rehabilitation Neurosciences Engineering Biomedical	Title	Article		3
("motor image*" OR "mental image*" OR "mental practi\$e") AND ("upper limb" OR "upper extremity" OR hand OR arm) AND ("stroke" OR "cerebrovascular accident")	Rehabilitation Neurosciences Clinical neurology Behavioural sciences	Topic	Article		86
("motor image*" OR "mental image*" OR "mental practi\$e") AND ("upper limb" OR "upper extremity" OR hand OR arm) AND ("stroke" OR "cerebrovascular accident" OR hemiplegia OR hemiparesis)	Rehabilitation Neurosciences Clinical neurology Behavioral Sciences Psychology Experimental	Topic	Article		104

Table 2:2

*Search strategy for MEDLINE from 2001 to January 2012*

Search number	Search terms	Hits
1	PSYCHOMOTOR PERFORMANCE/	41056
2	("motor image*" OR "mental image*" OR "mental pract*").ti,ab	1153
3	UPPER EXTREMITY/	3098
4	("upper limb" OR hand OR arm).ti,ab	90673
5	STROKE/	22450
6	HEMIPLEGIA/	9728
7	PARESIS/	4195
8	1 OR 2	24837
9	3 OR 4	91507
10	5 OR 6 OR 7	27429
11	8 AND 9 AND 10	308

/ indicates MeSH terms

### **2.2.5 Search limits.**

The search limits for Web of Science included title and topic searches for articles in rehabilitation neuroscience, clinical neurology, behavioural science and experimental psychology, and biomedical engineering journals. In MEDLINE, CINAHL and PSYCinfo the searches were limited to title and abstract. These limits were applied to all searches, including those outlined for chapters three and four below.

### **2.2.6 Review of reference lists.**

The reference lists of all complete articles accessed were checked and any relevant articles obtained. In addition, the reference lists of relevant monographs and edited collections were checked and appropriate references accessed.

### **2.2.7 Grey literature.**

As stated above, EThOS was searched (all years – October 2011), however no appropriate theses were identified. In addition, reports produced by government and charities were identified through free text internet searches using Google Search.

### **2.2.8 Tool employed for critical appraisal.**

The methodological quality of the RCTs was assessed using the criteria

provided by the Physiotherapy Evidence Database (PEDro) (Moseley, Herbert, Sherrington & Marr, 2002). This has been noted to provide a wide-ranging assessment of studies' methodological rigour (Barclay-Goddard et al., 2011) and has also been found to demonstrate validity (de Morton, 2009). The scale grades the methodological quality of studies according to 11 criteria, with one mark for each criterion met. The key studies identified for the literature review had a mean PEDro score of nine, indicating moderate to high quality (Moseley et al., 2002). The degree to which each of the studies met the PEDro criteria is presented in table 2:3.

Table 2:3

*Key studies referenced, showing PEDro criteria*

Study	PEDro Criteria										
	1	2	3	4	5	6	7	8	9	10	11
Bovend'Eerd et al. (2010)	✓	✓	✓	✓	*	*	*	✓	✓	✓	✓
Ietswaart et al. (2011)	✓	✓	✓	✓	×	×	*	×	✓	✓	✓
Liu et al. (2004)	✓	✓	✓	✓	×	×	✓	✓	✓	✓	✓
Page et al. (2001)	✓	✓	✓	✓	×	✓	✓	✓	✓	×	✓
Page et al. (2005)	✓	✓	✓	✓	✓	×	✓	✓	✓	✓	✓
Page et al. (2007)	✓	✓	✓	✓	×	✓	✓	✓	✓	✓	✓
Page et al. (2009)	✓	✓	✓	✓	×	✓	✓	✓	✓	✓	✓
Riccio et al. (2010)	✓	✓	✓	✓	×	×	✓	✓	✓	✓	✓

✓ Meets the criteria × Does not meet criteria \*Addressed within study design, but control broke down at some point.

*Note.* Key to PEDro criteria: 1 = inclusion criteria stated; 2 = random allocation to group; 3 = concealed allocation to group; 4 = groups similar at baseline; 5 = blinding of subjects; 6 = blinding of administering therapists; 7 = blinding of outcome assessors; 8 = > 85% subjects assessed on key outcome measure; 9 = all subjects completed treatment, or intention to treat analysis conducted; 10 = between group statistical analysis reported; 11 = point measures and variability measures provided for key outcome.

## 2.3 Literature Review and General Introduction

### 2.3.1 Randomised control trials of MI in stroke.

As noted in the introduction to chapter one, the possibility that MI might be used as a viable treatment strategy for the hemiplegic hand has provoked discussion amongst researchers and clinicians. One problem, however, which has been highlighted by several authors (for example, Barclay-Goddard et al., 2011; Ietswaart et

al., 2011), is the paucity of RCTs of MI in the treatment of the hemiplegic upper limb. As shown above, a number of studies was identified which did meet the majority of the PEDro criteria. These are outlined and discussed critically below.

Page, Levine, Sistow, and Johnston (2001) conducted research using thirteen stroke patients with upper limb hemiplegia. The researchers assessed patients at baseline using the Action Research Arm Test (ARAT) and the Fugl-Meyer (FM) assessment. ARAT is based on the assessment of grasping, pinching and whole arm movements, which it grades on a four point scale ranging from inability to perform the task to normal performance. FM grades upper limb activities at three levels, also ranging from inability to normal. A group which used physical practice plus mental training of a series of activities of daily living (ADLs) involving the most affected upper limb showed mean improvements on the FM and on the ARAT when compared with a group which used physical practice only. These results were not, however, compared for significant difference by the authors. In this sense, the study does not meet all of the requirements for an RCT. However, this study did use a methodology which has been employed in later RCTs by this group of researchers. These are discussed below.

Page, Levine, and Leonard (2005) conducted an experiment with 11 stroke patients who were more than one year post-event. All participants received training on activities of daily living (ADLs). Six, in addition, mentally rehearsed these ADLs, while the rest underwent only a relaxation task as a supplement to physical practice. The ADLs included reaching for and grasping a cup, turning a page and using a pen. Training took place twice a week over six weeks. Outcomes were assessed using the ARAT. It was found that the mean change on the ARAT was significantly higher for the mental practice group than for the physical practice alone group. This finding seemed to support MI as a useful adjunct to conventional therapy.

In a later study, Page, Levine, and Leonard (2007) once more employed the FM and ARAT to assess thirty stroke patients. Fifteen of the patients were randomized to a group using physical practice of upper limb skills in addition to guided relaxation. The other fifteen were randomized to a group which practised the tasks as well as mental imagery training of these activities. The training tasks were functional upper



limb tasks similar to those previously employed by Page et al. (2005) and training also took place twice a week over six weeks, as had been done in the earlier study. In this study, the mental practice plus physical practice group showed highly significant improvements on both the ARAT and FM, lending further support to the conclusions of Page et al. (2005).

A further study by Page, Levine, and Khoury (2009) randomly assigned five patients to a group using modified constraint induced movement therapy (CIMT) and five to a group using CIMT alongside MI. They received three sessions of practice per week over ten weeks. Once again, the training tasks were a series of ADLs using the entire upper limb. Similarly, the key outcome measures were also the ARAT and FM assessments. It was found that the group using CIMT alongside MI showed significantly greater changes on the ARAT and FM when compared with the group which used CIMT only.

A further RCT which presented positive results for MI was carried out by Liu, Chan, Lee, and Hui-Chan (2004). They compared performance of a series of everyday living tasks for a group of 46 people with stroke, randomised to an MI training group and a conventional therapy group. One unusual feature of this study was the way in which MI training was employed. The MI group first used MI to identify deficits in their performance of the prescribed ADL tasks. The tasks which specifically addressed upper limb function included folding laundry, washing dishes and making tea. Following this, they used MI to imagine an optimal performance of the respective tasks and then watched a video of themselves performing the task. They then imagined the optimal performance again and watched the video once more to compare their actual performance of the task and to identify what their problems with executing the task were. This procedure was repeated until it was felt that the patient had learned the task. The control group, meanwhile, did not use MI but had their actions corrected by a therapist.

Liu et al. (2004) assessed the patients' improvement on the performance of the ADL tasks with an ad hoc seven point Likert scale. It is not clear from the authors' description whether this was completed by the researcher or an administrating therapist. The scores on the scale ranged from total dependency in task performance,

to total independence. In addition, the FM was also used as a measure of upper limb performance. Liu et al. (2004) were able to identify significant improvements for the MI group on the Likert scale, but not on the results for the FM.

Another study was carried out by Bovend'Eerd, Dawes, Sackley, Izadi, and Wade (2009). They conducted a study on a group of 30 neurological patients. This was a heterogeneous group and had diagnoses including acquired brain injury and multiple sclerosis as well as stroke. The main outcome measure used here was goal attainment scoring (GAS). This involves the identification of specific goals for the individual patient. Post-treatment performance is then compared with baseline and they are scored on a scale which rates them up to two gradations better than target goal achievement, or two gradations below. The authors found that there were no significant differences over time, that is from baseline, post-treatment and follow up. In addition, there seems to have been no difference between the group which used MI and the group receiving conventional therapy.

Riccio, Iolascon, Barillali, Gimigliano, and Gimigliano (2010) conducted a study on 36 stroke patients suffering from upper limb hemiplegia. Half of the sample was randomised to a group using conventional therapy and MI training for three weeks, followed by three weeks of conventional therapy only. The other half was randomised to a group receiving three weeks of conventional therapy only, followed by three weeks of conventional therapy in addition to MI. The key outcome measure used for the upper limb was the Arm Functional Test (AFT), which measures upper limb task performance on 12 activities. These range from simple exercises, such as placing the elbow on a table, to fine motor tasks such as picking up straws. These 12 tasks also formed the basis for the MI treatment. At three weeks it was found that the group which had employed MI up until that point, alongside conventional treatment, improved significantly on the AFT both for quality of execution and speed. However, at the end of another three weeks, when the other group had also received MI training along with conventional treatment, the difference between the group scores was slight (Riccio, 2010).

Ietswaart et al. (2011) conducted a relatively large scale study of 121 people with upper limb hemiplegia, all of whom were less than six months post-stroke. There

were three groups in this study. The first used KMI to practise basic upper limb movements such as closing and opening of the hand, reaching and grasping everyday objects, and activities of daily living, such as ironing. Ietswaart et al. (2011) also included a condition designed to control for the effects that heightened attention might have had on the participants. The authors intended attention to include both the attention being paid to the participants by the administering therapist and the recruitment of cortical regions via the simple act of mentally attending to the MI task. To control for this, patients were asked to imagine a variety of static visual images and scenes and also different sensory and olfactory images, none of which were related to the tasks trained in the KMI condition. In addition, a control group received standard therapy on their upper limb. The main outcome measure in this study was the ARAT, and secondary measures included functional independence scales such as the Barthel scale and a timed test of manual dexterity.

Ietswaart et al. (2011) did not find any differences between the groups on any of the outcome measures. Indeed, the probability value for the differences between the groups on the key measure used, the ARAT, was very high ( $p = .77$ ). The authors conclude that MI training on its own may therefore not be an effective intervention in the rehabilitation of the hemiplegic upper limb. Certainly, the size and carefully controlled design of this study may pose a challenge to the notion that KMI is an effective intervention for hemiplegic patients in the months following stroke.

### **2.3.2 Limitations of the use of clinical scales as outcome measures.**

One limitation of the studies described above may be the use of the ARAT, FM and AFT as outcome measures. As shown, these are clinical scales with relatively low resolution. In so far as the researcher is interested in showing only that MI may be a useful clinical approach, this may be justifiable. However, if one is interested in exploring the effects of MI on specific muscle groups or joint configurations in the hemiplegic upper limb, such low resolution tests may have limited value. This is discussed in more detail in the next paragraph.

It is known that people with post-stroke hemiplegia may compensate for limitations at a distal joint with compensatory movements at more proximal joints (Cirstea & Levin, 2000). Thus, it is entirely possible that the functional upper limb

tasks used in the studies outlined above may have been achieved via the deployment of such compensatory strategies, which the ARAT, FM and AFT would not be sensitive to. Of course, this also has implications for clinical practice, as therapists are often concerned with the rehabilitation of highly fractionated movement (Gjelsvik, 2008; Porter & Lemon, 1993; Raghavan, 2007).

The use of such clinical outcome measures in MI research may also make it difficult to speculate about the mechanisms by which MI might work. For example, it may simply be the case that greater mental focus on the goal of the prescribed task led to improved performance. This may indicate that attention can play a part in the recovery of the hemiplegic upper limb. MI, however, has generally been conceived of as a cognitive process which promotes synaptic Hebbian learning and which can facilitate activity in motor regions which are specific to the performance of the task in question (Ietswaart, 2011). If this hypothesis were to be meaningfully tested, then higher resolution measurements may be called for.

It might be argued that the limitations of clinical scales are somewhat magnified in Liu et al.'s (2004) study. Here, the main outcome measure used, a specially created Likert scale, seems particularly unreliable and open to subjective interpretation. It is interesting to note, for example, that, while significant differences were found between the groups on the Likert scale, the higher resolution FM did not identify any significant differences between the groups (Liu et al., 2004). Similar criticisms might be made regarding the use of the GAS by Bovend'Eerd et al. (2009). While it is a useful technique for formulating individualized clinical treatment programmes, it may not have the appropriate rigour for an experiment on the specific effects of MI.

### **2.3.3 Limitations of the measurement of ADL performance as an outcome measure.**

All of the studies listed above used every day ADLs in their MI training protocols. Liu et al. (2004), for example, included activities such as using a method of transport and visiting a park. The use of such ADLs, however, would make it difficult to isolate specific muscle groups or joint movements, being focused as they mainly are on functional goals, with the motor patterns which should be used to achieve those

goals left undefined. As shown above, such goals may be achieved by individuals with hemiplegia using compensatory movements (Cirstea & Levin, 2000). The use of ADLs as outcome measures may therefore be unhelpful if the researcher is concerned to identify effects of MI on pre-specified muscle groups.

### **2.3.4 The degrees of freedom problem in MI research.**

It has been a staple idea in motor control for many years that a functional goal can be accomplished via a myriad of articular and muscular permutations (Bernstein, 1967; Llinas, 2003). Latash (2008), for example, argued that there are an infinite number of solutions to the problem of how to perform a targeted reaching movement. This has given rise to an important area of debate and research known as the “degrees of freedom” problem (Bernstein, 1967, p. 126). This centres on the attempt to understand the precise parameters used by the motor system to control such redundancy and has shaped motor research up until the present time (O’Brien & Bracewell, 2010; Stuart, 2005). It may follow that, if the researcher is interested in finding out if MI training has a specific effect at the level of isolated muscle groups or joints, it would therefore be necessary to constrain the degrees of freedom of a training task in such a way that the variables of interest can be measured. Some implications for this in relation to MI research in the rehabilitation of the hemiplegic hand are considered in relation to the present thesis in the following section.

### **2.3.5 Dissociation of arm and hand.**

The point of the present research was to look at the effects of MI in the promotion of improved hand function in hemiplegia. In trying to isolate and control specific components of hand function, it was therefore first useful to ask to what extent the hand could be viewed separately from the rest of the upper limb for the purpose of movement analysis. This is explored below.

First, in relation to their descending neural control, the hand and the rest of the upper limb are, to an extent, dissociable. Lesion studies in macaques have shown that damage at the level of the corticospinal tract leads to problems producing individuated finger movements (Lawrence & Kuypers, 1968; Schwartzman, 1978). Lesions starting in the subcortical nuclei, however, can leave finger movements intact but cause impairments to more proximal upper limb muscles (Lawrence & Kuypers, 1968a).

A study of a child with impaired cortical function reported by Jeannerod (1988) suggested that the same pattern of descending neural control is present in humans. This author noted that the child had poor grip formation, while the more proximal upper limb components of reaching remained intact. Similarly, Lang and Schieber (2004) found that patients with lesions only affecting the corticospinal tract had highly specific problems in the production of individuated finger movements. It seems, then, that the mechanisms of descending neural control of the hand and arm are fairly distinct.

Evidence also suggests that the hand and the rest of the upper limb are linked to dissociable activation at the cortical level. Binkofski et al. (1998) presented fMRI evidence showing consistent differential activation of the anterior border of the posterior parietal lobe during grasping activities, when compared with pointing or resting conditions. The authors suggest that these findings indicate a special role for this region of the parietal area in grasping movements, and also maintain that this area in humans may be taken as a homologue of the monkey anterior intraparietal (AIP) sulcus, which has been found to play a specific role in grasping (Binkofski et al. 1998; Castiello, 2005). Similar findings were made by Frey, Vinton, Norlund, and Grafton (2005) in an fMRI study of healthy individuals. They found that human AIP was activated during grasping activities using opposition of thumb and index finger, while it remained quiet during a pointing task, which focused more on the activation of the proximal upper limb (Frey et al., 2005). Furthermore, studies of apraxia suggest that lesions of the left inferior parietal lobes affect complex interactions between hand and object, while damage to the superior parietal lobe is more likely to impair movements as the whole upper limb moves towards the object, again pointing to distinct patterns of cortical activation which can be linked to proximal and distal upper limb movement components (Jeannerod, 2006).

In addition to the factors described above, the fingers also have a much denser concentration of mechanoreceptor fibres when compared with the rest of the upper limb (Gardner, Martin, & Jessell, 2000). As a consequence, the hand has a vastly exaggerated representation on the sensorimotor homunculus in relation to its size (Gardner, Martin, & Jessell, 2000). One functional implication of this is that the hand

seems to have a special role to play in postural stability. Jeka (1987), for example, found that the sensory feedback provided via very light finger tip touch was enough to provide substantial reduction in postural sway.

In responding to the challenge of the degrees of freedom problem it seemed appropriate for a study of the use of MI in upper limb hemiplegia to try to control the movements of the limb as far as possible. At the same time, the evidence presented above suggested that it would be both sensible and justifiable to try to separate out the hand from the rest of the upper limb for experimental purposes. This is discussed further below.

### **2.3.6 The need to analyse hand and arm movements separately.**

Researchers looking at the effects of MI on hemiplegia have tended to treat the arm and hand as a single unit. This is the case with the RCTs described above. However, even studies which supposedly focus on the hand, for example that of Dijkerman, Ietswaart, Johnston, and MacWalter (2004), sometimes use activities that would also involve movements at the wrist and elbow. In the case of Dijkerman et al.'s (2004) study, this involved a task moving tokens with the hand. Similarly, the study protocol of Ietswaart et al. (2006), upon which the study by Ietswaart et al., (2011) was based, refers only to the use of non-specific ADLs using the upper limb as MI training tasks. Kontaxis, Cutti, Johnson, and Veeger (2009) have argued that movements selected in such an experiment should present a challenge to the particular joints in which the researcher has an interest. Arguably, this was not achieved in the studies described. The way in which this problem was addressed in the present research is discussed below.

The hand and the arm have fairly distinct musculature. The lumbrical muscles, for example, are part of the intrinsic group of hand muscles which have their origin in the hand itself. This muscle arises from the tendons of flexor digitorum profundus (FDP), an extrinsic hand muscle which has its origin in the forearm (Palastanga, Field, & Soames, 2006). The lumbricals insert into the dorsal digital expansion at the lateral aspect of the proximal phalanx (Palastanga et al., 2006). The lumbrical group is unique insofar as it crosses both a flexor and an extensor tendon (Palastanga et al., 2006). This means that a single contraction leads to both flexion of the

metacarpophalangeal (MCP) joint and extension of the interphalangeal joint in the linked finger, a movement described by Palastanga et al. (2006) as the “lumbrical action” (p.101). It was decided to look at people with post-stroke hemiplegia grasping an object with the unusual constraint of using a specific type of grasp posture based on the lumbrical action, as described above (Palastanga et al., 2006). This involved grasping an object with the proximal interphalangeal (PIP) joint of the hemiplegic index finger extended and the metacarpophalangeal (MCP) joint of that finger flexed. This grip would be underpinned by the activation of the lumbrical muscles, and therefore is referred to as the ‘lumbrical grip’ in this experiment. The patients’ ability to produce this coordinated combination of flexion and extension could then be taken as an indication of the level of activation in this muscle group when using MI training.

### **2.3.7 The use of optoelectronic motion capture.**

Since this highly specific hand grip posture was being used it was also necessary to identify a way of accurately measuring the movements of individual joints, in order, for example, to determine the extent of any improved performance after using MI training. Clearly, the clinical outcome measures used in the studies outlined above would not have provided the degree of resolution needed to measure any change in individual finger joints. A method had to be identified, therefore, which would facilitate such measurement. This is discussed below.

Arguably, the optoelectronic capture of action and subsequent analysis of the kinematic profiles of the movements is one of the most precise methods available in the field of human motor research (Schmidt & Lee, 2005). Kinematics involves describing movement without reference to the forces which produce the movement (Zatsiorsky, 1998). Digitization of movement and kinematic analysis provide a much more detailed quantitative analysis of hand movements than clinical outcome measures. However, this technique has seldom been used in MI research. Some researchers, such as Allami, Paulignan, Brovelli, and Boussaoud (2008) have, however, demonstrated its use in a study of hand movements in healthy individuals. Furthermore, Hewett, Ford, Levine, and Page (2007) used the technique when comparing the arm movements of people with stroke in an experiment on MI. These authors maintained that their study was the first to use this method to analyse the



effects of MI in upper limb hemiplegia.

Hewett et al. (2007) identified two key advantages for the method of optoelectronic motion capture. First, because it depends on electronic measurement of movement, the degree of subjective human error in measurement is minimised. This helps overcome the problem of reliability raised by the use of clinical outcome measures. Second, kinematics allows the measurement of a wider range of movement characteristics and more detailed measurement of those characteristics than that provided by clinical scales. In the case of Hewett et al. (2007), these measures included the velocity of the upper limb and the exact magnitude of extension and flexion at the joints of interest.

It is worth pointing out, in addition, that the use of kinematics answers the concerns raised by other authors regarding the use of clinical scales in research on motor function following stroke (Malouin et al., 2004; Raghavan, 2007). Nudo (1999), in particular, has explicitly called for the adoption of kinematic analysis in place of clinical outcome measures when analysing the movements of stroke patients. Furthermore, evidence suggests that the motor problems encountered in stroke may involve altered inter-joint coordination (Cirstea & Levin, 2000; Levin, 1996). Since the research presented here was to be concerned with the interrelated movement of the MCP and PIP joints it was felt that this technique was well suited to providing the detailed and multifaceted data needed to analyse these movements and assess any differences when using MI training.

### **2.3.8 The use of mainstream therapy alongside MI.**

A further point raised by research on MI as a feasible motor training approach is that it might work effectively only in conjunction with more conventional physical training. It will be recalled, for instance, that the positive results for MI shown by Page et al. (2005) and Page et al. (2007), were achieved when MI was deployed alongside mainstream therapy. Page et al. (2009), meanwhile, found success using MI along with the more novel of approach of CIMT. Although Ietswaart et al. (2011, p. 1378) failed to identify any benefits of MI, their patients also received what the authors describe as “standard” rehabilitation therapy in addition to MI training. Similarly, Hemmen and Seelen (2007) used MI with feedback triggered by EMG in their

experiment on the rehabilitation of the hemiplegic upper limb, while their patient group also received therapy as normal. A recent extensive review of the literature also identified that motor imagery may be more successful when used alongside physical practice (Schuster et al., 2011).

The inclusion of conventional mainstream physical therapy in some studies may, however, be problematic. This is because this aspect of the research sometimes seems to have been quite vaguely defined. Page et al. (2005), Page et al. (2007) and Page et al. (2009), for example, describe the goals of the physical treatment sessions, but not the specific therapeutic interventions used to achieve them. Similarly, Ietswaart et al. (2011) do not provide a detailed account of the standard therapy provided. The same is true of the study by Riccio et al. (2010). However, as has been pointed out above, there is a wide variety of rehabilitative strategies used by therapists (Langhorne et al., 2009; Woldag & Hummelsheim, 2002). Evidence also suggests that in the clinical arena therapists take a highly pragmatic approach to the treatment of the hemiplegic upper limb (Kuipers et al., 2006). Therefore, what takes place during conventional therapy should not be taken for granted by the researcher when designing an experiment.

It might be argued that the ‘therapy as normal’ aspect could have been poorly controlled in some studies and might work as a confounding variable exerting an unidentified influence over the results. If the researcher is attempting to conduct a valid test of the effects of MI training in upper limb hemiplegia, it may be necessary therefore to define the physical treatment provided with greater precision. This challenge was met in the present research by giving all patients a series of physical treatment sessions based on an identical treatment protocol, which is outlined in appendix 2:6. This protocol was designed by the author. The techniques used were based upon his own clinical training and practice and were in accord with standard treatment in the UK. The theoretical basis of the treatment programme, as well as a specific description of what was done, is given below.

#### **2.4 Theoretical reasoning behind treatment protocol.**

Authors have pointed to the importance of promoting afferent feedback from the hemiplegic hand during rehabilitation. Such information may allow the motor

control system to calibrate the force required to perform a task (Champion, Barber & Lynch-Ellerington, 2009). It is known, in addition, that afferent feedback can provoke activation in corticospinal areas (Rosenkrantz & Rothwell, 2004). Since the corticospinal tract is known to be crucial in controlling individuated finger movements (Lawrence & Kuypers, 1968; Schwartzman, 1978), then sensory stimulation also has a role in the activation of individual finger muscles. Overall, the summed impact of repeated sensory stimulation is thought to facilitate muscle activity (Champion, Barber & Lynch-Ellerington, 2009; Sherrington, 1947). In addition, it may promote the integration of the divergent efferent inputs to the intrinsic hand muscles (Champion, Barber & Lynch-Ellerington, 2009). For these reasons it is felt to be a useful prelude to active use of the hand during therapy (Champion, Barber & Lynch-Ellerington, 2009).

#### **2.4.1 Procedure of physical treatment protocol.**

In the physical therapy protocol of the present study sensory stimulation was first given via a chopstick with angular sides that was rotated from side to side by the researcher and moved over the surfaces of the fingertips, webspaces and sides of the hemiplegic hand. These locations, particularly the finger tips and web spaces, are dense with mechanoreceptors and the low frequency vibration which the revolving chopstick would have produced is known to preferentially stimulate the Merkel discs (Gardner, Martin & Jessell, 2000). Vibration is also known to activate the 1a sensors which attach to the intrafusal muscle fibres and are especially numerous in the intrinsic hand muscles (Champion, Barber & Lynch-Ellerington, 2009; Gjelsvik, 2008). Vibratory feedback also plays a role in cortical plasticity in hand motor areas (Jenkins, Merzenich, Ochs, Allard & Guic-Robles, 1990).

Following sensory stimulation, therapy aimed at promoting active movement in the hand intrinsic muscles commenced. The thumb is known to play an important role in stabilising the hand while grasping (Champion, Barber & Lynch-Ellerington, 2009; Wing and Fraser, 1993). Each session of active therapy therefore began with the researcher guiding the hemiplegic thumb into abduction and opposition. Evidence shows that the cortical plasticity produced by afferent feedback from intrinsic hand muscles is modulated by attention (Rosenkrantz & Rothwell, 2004). The individual

was therefore encouraged throughout to attempt active movement of the thumb, while the researcher continued to physically guide and support the action. The focus then turned to activation of the hand lumbricals, as recommended by Champion, Barber and Lynch-Ellerington (2009). The researcher once again physically guided the movement while explaining the target movement pattern to the participant. Here again, active participation was encouraged. Furthermore, the researcher used his finger tips to give light downward pressure into the palm, providing resistance to the lumbrical action and promoting strengthening of the muscles. Similar techniques were used for the index and little finger of the hemiplegic hand. The researcher guided the fingers into abduction and adduction, encouraging active movement throughout. Finally, the hemiplegic upper limb was facilitated by the researcher into a forward reach. The hand was guided into pronation onto a table placed in front of the seated individual, with the medial border making contact first with the table. The aim here was to facilitate a “contactual hand orientating response” (CHOR) (Champion, Barber & Lynch-Ellerington, 2009, p. 173). This is a friction based contact with a supporting surface and is considered important as a preliminary to functional activation of the hand (Champion, Barber & Lynch-Ellerington, 2009).

The aim of this physical treatment programme was to promote a more active intrinsic musculature in the hemiplegic hand. The specific objectives were to overcome the problem of “learned non-use” (Carr & Shepherd, 1998, p. 14) and to maintain the arches and postural stability of the hand (Champion, Barber & Lynch-Ellerington, 2009). Some limitations of the physical treatment protocol are outlined in the discussion section of this chapter.

## **2.5 The sensory content of MI training used in experiment one.**

A further limitation of some studies concerns the definition of the MI tasks used. MI can be based in different sensory modalities. Generally, researchers have employed KMI and VMI (Dickstein & Deutsch, 2007; Ietswaart et al., 2006; Mulder, 2007; Sharma et al., 2006). The emphasis in VMI is on the observed appearance of the movement, and has therefore been described as “third person” MI; KMI, meanwhile has been labelled “first person” MI (Mulder, 2007, p.1268). Mulder (2007) maintained that only first person KMI was effective in rehabilitation tasks, as

did Sharma et al, (2006). It is notable that Ietswaart et al. (2011) explicitly outlined a KMI strategy in their study. Page et al. (2005) and Page et al. (2007), however, did not state which modality was used in their experiments. Page et al. (2009, p. 552), meanwhile, referred to the use of “polysensory cues” in their MI protocol, but provided no further detail. Similarly, Liu et al. (2004) did not provide this information, although their protocol would seem to imply a VMI approach. Similarly, Riccio et al. (2010) did not give any information regarding the sensory content of the imagery used. Other studies which have not defined the sensory modality of the MI tasks used were those of Butler and Page (2006), Cincotta et al. (1999), Crosbie, McDonough, Gilmore, and Wiggam (2008), and Hemmen and Seelen (2007).

The failure to detail the sensory modality may be a limiting factor in the studies described above. This is because some evidence does suggest an advantage for the kinaesthetic modality when imaging movement. Stinear (2006), for example, used TMS to examine the levels of cortical excitability associated with KMI and VMI of a thumb tapping task. It was found that only KMI led to raised cortical excitation in the region corresponding to abductor pollicis brevis (APB). If, as is suggested by Jeannerod’s (2006) hypothesis of motor simulation, the proposed efficacy of MI training is linked to the fact that imagery recruits cortical regions which would actually be involved in motor execution, then this could potentially be an important factor to take into account in any MI experiment. As a consequence, the MI task used in the present research was clearly defined. KMI was used, and the script is shown in appendix 2:7

## **2.6 Kinematic Variables Analysed in Experiment One.**

As stated above, hand movements in this study were captured optoelectronically and the kinematics then used as the dependent variables for inferential statistical analysis. The kinematic variables measured are outlined below.

### **2.6.1 Mean PIP angle magnitude.**

There is some evidence that mental imagery training may improve the ability to extend a joint in the hemiplegic upper limb. Hewett et al. (2006), for example, found increased elbow extension in people with hemiplegia following such treatment, although the differences were non-significant. It has been shown above that the

extension of the PIP joint is partially underpinned by the action of the lumbrical muscles. It was therefore predicted that KMI aimed at the activation of the lumbrical muscles would be associated with significantly increased ability to extend the PIP joint. In addition, it was hypothesized that this would occur during the phase of the grasp in which the hand was actually holding the object, rather than approaching or leaving it, as this was actually the target task presented to the patients in the study.

It was felt, in addition, that measuring the mean angle rather than the maximum angle, as was done by Hewett et al. (2007), made sense. This is because the mean provides a clearer picture of the angle magnitude over the whole measurement period, rather than an isolated peak value which might not be representative of the entire movement.

### **2.6.2 Mean MT.**

Evidence suggests that one feature of motor problems following neurological injury is slowed movement. Jeannerod (1986), for example, found longer movement durations during a reaching task in a person with a head injury following a traumatic brain injury. Nowak et al. (2007), meanwhile, found significantly slower movement times for the hemiplegic hand in people with stroke when compared with healthy controls. Van Vliet and Sheridan (2007) also found that stroke patients had significantly longer MT in the hemiplegic upper limb than healthy individuals. These authors also found that a more challenging grasping task led to a slower movement time for their participants (Van Vliet & Sheridan, 2007).

Conversely, successful treatment strategies have been identified by their ability to decrease MT in tasks involving the hemiplegic upper limb. Treatment using repetitive transcranial magnetic stimulation (rTMS) has, for example, been linked to a significantly lower movement time for the hemiplegic hand following treatment (Nowak et al., 2007). In addition, Koesler, Dafotakis, Ameli, Fink, and Nowak (2007) found that treatment with electrical stimulation led to a higher frequency of index finger tapping movements in the hemiplegic hand. On this basis it was therefore predicted that treatment with KMI would lead to a shorter MT for the hand.

### **2.6.3 Mean velocity of the PIP joint.**

Nowak et al. (2007) found a significantly lower peak wrist velocity in reaching

movements made by people with upper limb hemiplegia when compared with healthy controls. These authors also found a significantly lower velocity for index finger tapping movements in the hemiplegic hand when compared with a healthy control group. In addition, Van Vliet and Sheridan (2007) noted significantly higher peak velocity in the arm movements of healthy individuals, when compared with those with upper limb hemiplegia following stroke.

As for MT, higher velocity has been seen as a sign of improved motor function. Wagner, Rhodes, and Patten (2008), for example, have pointed out that a higher peak velocity indicates a speedier and more efficient movement. Furthermore, Koesler et al. (2008) linked improved motor function following treatment of the hemiplegic upper limb to higher wrist velocity in reaching movements. Hewett et al. (2006) also found increased linear velocity in reaching movements in the hemiplegic upper limb following mental training. Perhaps most interestingly for the current study, improved motor function in hemiplegia has also been linked to higher peak velocity of index finger movements following rTMS treatment by Nowak et al. (2008). It was felt, however, that for the present study a measure of mean velocity might be more representative of the entire movement. It was therefore predicted that the mean angular velocity of the PIP would be significantly higher for the group using KMI.

#### **2.6.4 Time to peak PIP velocity of the PIP joint.**

Van Vliet and Sheridan (2007) noted that peak velocity occurred significantly earlier in the hemiplegic upper limbs of people with stroke when comparing a reaching and grasping movement with those of healthy controls. They related this to a shift towards motor control based on sensory feedback, a point also made by Lin et al. (2007). This is in comparison with an impulse controlled movement which would not make reference to sensory feedback. This latter control strategy has been seen as being typical of highly practised and skilled movements (Woodworth, 1899). Such movements would presumably be guided more by a central motor program (Schmidt & Lee, 2005).

Successful treatment strategies in upper limb hemiplegia have accordingly been found to lead to a shift in the position of temporal landmarks in movement. Lin et al. (2007) speculate that this may indicate a switch towards a more efficient motor

control strategy. It was therefore hypothesized for the present research that a group using KMI would show a later time to peak velocity when reaching.

### **2.6.5 Number of movement units for the PIP joint during the approach phase.**

The multi-joint upper limb movements of healthy individuals are characterized by a straight and smooth trajectory with a normally distributed profile for velocity (Levin, 1999). It has also been proposed that reaching in a healthy individual should include a single phase of acceleration, and a single phase of deceleration (Lin et al., 2007). It is further argued that the acceleration phase can be seen as an index of the extent of the movement segment which is pre-planned and based on feed-forward control (Lin et al., 2007). Furthermore, it has been maintained that individuals with stroke are likely to rely more heavily on feedback control, which is likely to be characterized by increased numbers of movement units (MUs), with one MU defined as one unit of acceleration and one unit of deceleration (Lin et al., 2007).

Several authors have highlighted the high levels of variability in upper limb kinematics in hemiplegia (Cirstea & Levin, 2000; Cirstea, Mitnitski, & Feldman, 2003; Van Vliet & Sheridan, 2007; Nowak et al., 2007). Wagner et al (2008) point out that a smoother movement is characterized by fewer peaks in the velocity profile. Lin et al. (2007) also hypothesized that an improved upper limb trajectory following treatment for the hemiplegic upper limb would be characterized by fewer MUs. It was predicted in the present research that the acceleration profile of the PIP joint of a patient group using KMI would be characterized by fewer MUs during the phase of the movement in which the hand was approaching towards the object to be grasped.

### **2.6.6 Correlation between MCP and PIP joints during the holding phase.**

Previous researchers have noted that a key feature of movement in the hemiplegic upper limb is a reduction in inter-joint coordination (Levin, 1996). It has been found, for instance, that the correlation coefficients for elbow and shoulder movements are much lower for the hemiplegic upper limb than in the non-hemiplegic upper limb (Levin, 1996). It has also been stated that improved inter-joint coordination should be a priority goal for rehabilitation of the hemiplegic hand (Raghavan, 2007).



Wagner et al. (2008) have argued that closely coupled joint movements may be marked by a high negative correlation. This suggests that, although the kinematic profiles of the joint movements are strongly linked, one joint is decreasing in size as the other is increasing. Wagner et al.'s (2008) comments fit well with the analysis of the lumbrical action. This is because, in a smooth lumbrical action, the PIP should increase its extension, while the MCP joint should increase in flexion. It was therefore predicted that the KMI group would display a higher level of negative correlation between the MCP and PIP joints, and that this correlation would be statistically significant.

## **2.7 Ethical Issues and Informed Consent**

The research was conducted in Bogotá, D.C., the capital of Colombia, as part of a study agreement between the University of Rosario and Bangor University in the United Kingdom. This presented a number of potential ethical challenges. These are explored below.

### **2.7.1 Ethical challenges**

The researcher was from a wealthy Western country while the participants were from a poorer nation with a recent history of conflict and high levels of internal displacement resulting both from violence and economic pressures. In addition, the population served by the hospitals from which the participants were recruited tended to be from the poorer neighbourhoods of the city. Marshall (2007, p. 5) has pointed out that such a contrast between researcher and participant means that “some individuals and communities may be vulnerable to exploitation or coercion because of their poverty or social status”. Furthermore, Resolution 008430 of 1993 of the Colombian Ministry of Health, which established standards for research conducted in the country, highlights the special consideration needed for dependent groups (“*grupos subordinados*”), defined as “those whose informed consent could be influenced by an authority” (Ministerio de Salud, 1993, p. 9). Although the researcher was not involved in the delivery of the individuals’ health care, it could, therefore, still have been the case that disparity in perceived status may have created space for coercion and dependency. The measures taken to minimise this risk are discussed below.

First, voluntary informed consent, which is considered internationally and also in Colombian law as the foundation of ethical research practice (Marshall, 2007; Ministerio de Salud, 1993), was obtained via a document judged to be clear, comprehensive and understandable for a member of the non-scientific community following review by the University of Rosario ethical review committee (ERC). Specifically, the patient information sheet met a key requirement of Resolution 008430 and the World Health Organization (WHO) (Marshall, 2007) for working with dependent groups: that it should be make clear that the person can leave the study at any time, without giving a reason, and that this will not have a negative impact upon any ongoing treatment they are receiving.

Marshall (2007, p. 24) has also highlighted the danger of “therapeutic misconception”. This may arise when individuals from disadvantaged communities believe that they may benefit from treatment on account of the perceived higher status of the researcher. It was therefore made explicit in the patient information sheet that the person could expect no immediate benefits from taking part in the study. An English translation of the patient information sheet is shown in appendix 2:3.

The WHO has also averred that the “gate-keeping” role of the ERC means that the composition of this body in the host country may be a factor in reducing the risks posed to informed consent by dependency and coercion (Marshall, 2007). The University of Rosario ERC, whose members are listed in appendix 2:2, met the recommendations made by the WHO in this respect and included a medical doctor, a member of the public, a lawyer and a bioethicist. The ERC also met further standards laid down by the Panamerican Health Organisation and enshrined in Colombian law by resolution 008430, which specify that the committee should be balanced with regard to age and gender. One aspect of the Colombian ERC which differed from an ERC in the UK was that it included an individual who was performing obligatory national service. This, however, is not felt to have posed any threat to the role of the ERC in ensuring that the principles of voluntary informed consent were upheld.

Marshall (2007) has also pointed out that language can be a further complicating factor in research of this kind. In this case, the researcher was conducting the work in Spanish, which was his second language. With regards to the process of

informed consent, the technique of back translation, recommended by Marshall (2007), was used. The informed consent document was first translated from English to Spanish and then back again to English as a means of checking validity. One issue that arose here, which at first sight may seem to present a risk for coercion by inflating the status of the researcher, concerns the use of the honorific “doctor”. This was inserted into the Spanish text by the local supervisor and approved by the ERC. This issue is discussed further in the following paragraph.

This title is conventionally used in Colombian Spanish as a term of respect for any professional person. For example, the Colombian occupational therapist that assisted the researcher had the title “doctor” on her office door in the hospital; it was generally understood, however, that she was neither a medical doctor, nor possessed a PhD. It will be noted that the letter of approval from the ERC (appendix 2:2) also used the term when addressing the researcher. This did not, however, represent any dissimulation on the part of the researcher or misunderstanding on the part of the ERC as to his academic status.

Another difference from United Kingdom convention is the use of the term “cerebrovascular accident” (CVA). This is now generally eschewed in the UK favour of the word “stroke” (Hankey, 2007). The ERC felt, however, that CVA was the more appropriate term in the Colombian context.

In order to facilitate the process of informed consent, individuals were given plenty of time to read the information sheet prior to deciding whether to indicate their consent by signing the form. During this time, all questions were answered verbally by the researcher. No pre-specified period in which the individual could reflect before giving their informed consent was required by the ERC. However, when individuals requested this, more time was immediately granted and a follow up telephone call was agreed within one week at a convenient time for the individual. It was made clear verbally that they were entirely free to decline to take part at this point, and that no further contact would be made. In retrospect, however, a pre-specified “cooling off period” for the participant before they made any decision may have been beneficial in further minimising any risk of coercion.

A further recommendation by the WHO is that research agreements between

wealthier and resource-poor countries should be conducted in the spirit of “collaborative partnership” between the researcher’s home institution and the host body and a key objective should be the development of research capacity in the host country (Marshall, 2007, p. 13). This was certainly aimed at in the present case. Concretely, the researcher’s UK supervisor visited Colombia in 2009 and delivered a series of lectures at both the university and hospital sites on a voluntary basis. The researcher himself also undertook teaching on the occupational therapy programme of the University of Rosario and on the engineering programme at Central University, also voluntarily. In addition, joint publication was undertaken and the findings of the study were published in a co-authored paper by the University of Rosario. The research was also registered with the Administrative Department of Science, Technology and Innovation of the Republic of Colombia, known as *Colciencias*. This organ is involved in the co-ordination of research within Colombia, ensuring that it complies with national government strategy for the development of science and technology. In addition, the project was listed on the website of Méderi Hospital Universitario Major, the main partner hospital in the research. It should be noted that registration of the study in this way is also in line with the recommendations of the Declaration of Helsinki (World Medical Association, 2008).

### **2.7.2 Legislation guiding ethical approval.**

The ERC decision to approve the research was guided by the principles of the World Medical Organisation Declaration of Helsinki (World Medical Organisation, 2008), as is also required by the UK Medical Research Council (1998). The ERC made additional reference to the guidelines of the International Conference of Harmonization of Good Clinical Practice (European Medicines Agency, 2002). The decision was also guided by two pieces of Colombian legislation: Resolution 008430, which has been discussed above, and Resolution 002378 of 2008 of the Ministry of Social Protection. These resolutions are based upon Law 10 of 1990 and Law 100 of 1993, which established the current framework for the delivery of health services in Colombia. The articles of these legal resolutions are in line with the research guidelines established for the United Kingdom by the Department of Health (2005) and the Medical Research Council (1998) and those recognised by the researcher’s

professional body, the College of Occupational Therapists. Specifically, they require that the research should be scientifically sound and that the dignity, security and well being of participants are secured by the researcher. Anonymity of any participants must be ensured. Informed consent via a signed declaration must be obtained and any risks explained clearly. In addition, the ERC is required to review and approve the curriculum vitae and qualifications of the researcher. The researcher must also be familiar with Colombian Law and the Helsinki declaration. The ERC decision to grant approval to the research is shown in the letter included in appendix 2:1, followed by an English translation in appendix 2:2.

One difference between the Colombian and UK requirements was that separate ethical approval was not required from the two hospitals involved in the research. This is because the hospitals were actually owned by the University of Rosario, therefore ethical committee approval also covered these institutions.

### **2.7.3 Requirements of the researcher's home university.**

Bangor University requires that students registered at the university should familiarize themselves with the legal requirements and ethical practices of the host country while conducting research abroad. In addition, it is stipulated that any research should be approved by an ERC, although further approval from Bangor University is not required. As has been shown, all of these requirements were met by the researcher.

### **2.7.4 Indemnity arrangements.**

The indemnity arrangements were outlined in the contract agreed between the two universities. The full text of this arrangement, signed by the relevant parties, can be read in appendix 2:4. The details of the liability arrangements for death and personal accident applicable to both institutions can be read in section eight.

All study participants were enrolled in the obligatory national health insurance scheme, which ensures emergency health care. No special arrangements were made for compensation to individuals recruited to the study in the event of harm. This is in line with the policy of the University of Rosario and was discussed by the researcher with his supervisor at the university and approved by the ERC. This was in accord with the contractual agreement between the two universities. In addition, this is in line with the standard advice given to research participants by Bangor University. The

telephone details of the president of the ethics committee were provided in the informed consent documentation, and the individual invited to contact him with any questions about the study.

#### **2.7.5 Supervision of the research.**

As part of the contractual agreement a supervisor deemed appropriate by Bangor University was appointed by the University of Rosario. The researcher was admitted as a visiting student to the research group “Health, Work and Cognition”, based at the Faculty of Health and Human Development at the University of Rosario. The researcher was also required to appear before an annual supervisory committee at Bangor University. In addition, ongoing supervision was provided throughout by the researcher’s supervisor at Bangor University.

### **2.8 Method**

#### **2.8.1 Recruitment sites.**

Patients were recruited from two hospitals in Bogotá, D.C., Colombia. One was a large general hospital, Hospital Universitario Major; the other, a smaller twinned hospital, Barrios Unidos, which was more orientated towards subacute care and rehabilitation. In 2011 the hospitals were ranked at 42 by the business magazine *América Economía* in a list of the best hospitals in Latin America. The rankings were decided, among other things, according to levels of patient dignity and safety, research development and efficiency (*América Economía*, 2011). Both hospitals were owned by the University of Rosario.

#### **2.8.2 Recruitment process.**

The researcher visited both hospitals on a daily basis during the recruitment period, which took place from March to December 2009. Neither hospital had a dedicated stroke unit. It was therefore necessary to identify potential candidates from the population of general patients who had been referred to the occupational therapy and physiotherapy departments. In Hospital Universitario Major, this was done by daily inspection of the admissions list to the physiotherapy and occupational therapy services. The primary diagnosis was listed next to the patient’s name, and so it was possible to identify if he or she had had a stroke. In addition, occupational therapists and physiotherapists at the hospital, who had previously been made aware of the

inclusion criteria of the study, would inform the researcher by telephone if they knew of a new stroke patient who they felt would be appropriate. In Barrios Unidos hospital there was no central electronic referral list for therapies. The researcher therefore visited each ward daily and asked the ward manager if there were any new patients with a medically confirmed primary diagnosis of stroke. The researcher also visited the therapy departments in both hospitals to check if there were any individuals with stroke who were visiting for outpatient appointments.

Once a potential candidate had been identified using one of the methods described above, that individual was approached in person by the researcher. The researcher had to initially make his own clinical judgement as to the individual's suitability for the study. Some inpatients, for example, appeared drowsy upon approach. If they failed to focus their attention upon the auditory stimulus of their name being spoken or the tactile stimulus of having their shoulder gently rubbed, then it was decided that they may not have capacity to give informed consent and the meeting was terminated by the researcher. If they were able to initially focus their attention, but not sustain this, as evidenced by maintaining appropriate eye contact, then the interview was also stopped.

In cases where the researcher felt it was appropriate to proceed, the study was explained to the patient. If they were interested following this initial explanation, they were supplied with the patient information sheet. At this point the individual either read the sheet immediately, or, if they requested more time, the researcher would arrange to visit them again on the hospital ward within one week. Once the person had stated that they would like to take part in the study, the upper limb components of the Motor Assessment Scale were used to test their baseline motor function. This testing took place in the hospital setting. The use of such testing to screen participants prior to the signing of the informed consent documentation is in line with the procedure used by Page et al. (2001; 2005; 2007). It was felt that it was a good idea to have a clear idea of the person's suitability, rather than having them give informed consent and then telling them that they would not, in fact, be appropriate. Once screening had taken place, and informed consent had been given, then the physical treatment protocol of the study was commenced.

### **2.8.3 Participant characteristics.**

The inclusion criteria were: (a) over 18 years of age; (b) less than one year post stroke; (c) hemiplegia affecting the hand resulting in a loss of dexterity and/or power as a result of the stroke; (d) a score of one or more on the hand activities component of the Motor Assessment Scale; (e) an ability to understand all instructions given to them while being tested using the upper limb components of the Motor Assessment Scale; (f) score of four or less on the Modified Ashworth Scale; (g) an ability to mobilize and transfer independently without an aid; (h) normal or corrected to normal vision; (i) not partaking in any other clinical studies. The patients' scores on the baseline tests are shown in table 2:4. All patients who met the inclusion criteria agreed to take part in the study.

### **2.8.4 Payments.**

No payments were made for taking part in the study. Any transport costs were met by the researcher.

### **2.8.5 Skills and experience of researcher.**

All testing, administration and data collection was carried out by a band seven occupational therapist with over six years experience in neurological rehabilitation and specialist training in stroke rehabilitation. The researcher had passed the three introductory course modules on the treatment of adult hemiplegia organised by the British Bobath Tutors Association (BBTA) in June 2005. The researcher had also passed the basic course entitled "Assessment and Treatment of Adults with Neurological Conditions – The Bobath Concept", also organised by BBTA, in June 2007. The researcher also had professional experience delivering guided relaxation treatment to patient groups. In addition, the researcher had a Master of Science degree in Applied Neuropsychology from the University of Bangor, awarded in September 2007. Furthermore, the researcher held a level one certificate in Spanish from the University of Liverpool and was a fluent Spanish speaker.

### **2.8.6 Note on the sampling process.**

The theoretical population for this research was all living adults with stroke who fitted the inclusion criteria. This was clearly an impractically large and difficult to access population. However, even if the population was defined as all adults with



stroke admitted to the two hospitals, the researcher still did not have access to their details. Even if he had it would nonetheless have been immensely difficult to randomly identify patients from such a list, without any information as to their suitability for the study, contact them and arrange to meet and assess them. This was essentially, then, a non-probability convenience sample. It will be noted that, even with this approach, it still took ten months to recruit ten patients who fitted the inclusion criteria. This type of sampling is common in motor control studies using a neurological population, which generally have relatively small sample sizes (see, for instance, Roy et al., 2011; Page et al., 2001; Page et al., 2007). Some limitations of this approach are outlined in the discussion section of this chapter.

Table 2:4

*Patient Scores on Baseline Tests*

Group	Patient	MAS Upper arm function	MAS hand movements	MAS advanced hand	Ashworth score
KMI	1	6	5	2	2
	2	4	5	1	2
	3	4	2	1	2
	4	6	5	2	2
	5	4	3	1	2
Relaxation	6	5	5	3	2
	7	5	6	2	1
	8	4	1	1	2
	9	6	5	1	2
	10	4	5	0	3

**2.8.7 Sample size and characteristics.**

To the best of the researcher's knowledge, no previous studies of this sort had been undertaken. Data were unavailable, therefore, upon which to calculate power and sample size. However, the intended and actual sample size of the study was ten. A group of ten patients between the ages of 57 and 92 ( $M_{\text{age}} = 74.4$ ,  $SD_{\text{age}} = 10.31$ ) was recruited. Five were male. All completed the study. The participants' demographic

data are shown in table 2:5.

### **2.8.8 Safety and comfort considerations.**

Participants were informed that they could leave the study at any time without giving a reason. The researcher, whose qualifications and training are described above, monitored for any discomfort or distress at all times. No adverse events were observed by or reported to the researcher during the study.

Table 2:5

#### *Participant Characteristics*

	Patient	Age	Gender	Time since stroke	Affected side	Hand dominance
Non-KMI group	1	57	M	2 months	L	R
	2	74	F	3 months	L	R
	3	92	F	2 months	L	R
	4	82	F	6 months	L	R
	5	76	F	2 months	R	R
KMI group	6	81	F	15 days	R	R
	7	71	M	4 months	R	R
	8	63	M	6 months	L	R
	9	67	M	3 months	L	R
	10	81	M	2 months	L	R

### **2.8.9 Pilot study.**

A pilot feasibility study was conducted using one person with hemiplegia (male, 67 years), recruited via the hospitals as described above. The aim was to identify any problems in the research design. The objectives were: (1) to ensure that the camera could “see” all markers when placed on the hemiplegic hand; (2) to test that the method of delivering the MI instructions via a pre-recorded tape was appropriate and could be understood by the person; (3) to identify any hitherto unforeseen problems with the motion capture system; (4) to check that the researcher’s instructions were easily understood by the person. Following this pilot, it was found that camera settings had to be modified so that the capture of the hand movements was made in one continuous sequence, rather than each grasp being captured as a separate

sequence, which proved to be impractical with the motion capture system being used

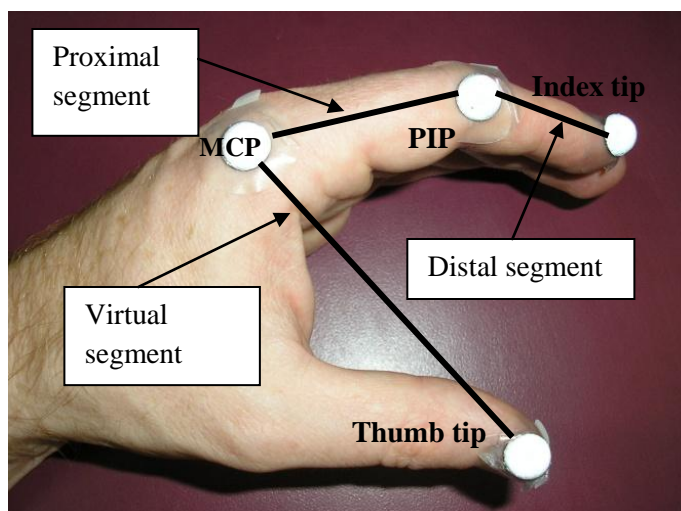
#### **2.8.10 Measures used.**

The Motor Assessment Scale (MAS) was used as a screening test of hand function. This scale has been shown to demonstrate both reliability (Carr, Shepherd, Nordholm, & Lynne, 1985) and validity (Carr & Shepherd, 1998). Assessment is based on the performance of functional tasks on which the patient is scored on an ascending scale from zero to six. The examiner may use separate segments of the MAS without compromising its reliability or validity (Carr & Shepherd, 1998). In this study only the three upper limb components of the assessment were used.

An assessment of upper limb muscle tone was carried using the Modified Ashworth Scale. This scale has been shown to demonstrate inter-rater reliability when used to test the upper limb (Bohannon & Smith, 1987). The Modified Ashworth Scale rates tone on an ascending scale, with zero indicating no increase in muscular tone and five indicating rigidity.

#### **2.8.11 Joint kinematics and biomechanical model of hand.**

Infrared reflective markers were placed on the index finger tip, the proximal interphalangeal (PIP) and the metacarpophalangeal (MCP) joints of the index finger of the hemiplegic hand. In addition, a marker was placed on the MCP joint of the thumb and the thumb tip. Data gathered from these latter two markers were not used in the final analysis. The location of the markers is shown in figure 2:1.



*Figure 2:1.* Location of reflective markers on hand. The figure also shows the biomechanical hand model.

Joint angles were defined as the attitude of two bony segments in relation to each other. The proximal phalangeal segment was defined as the distance between the MCP marker and PIP marker. The distal phalangeal segment was defined as the distance between the PIP marker and the marker on the index finger tip. In addition, there was a virtual segment which was defined as the distance between the MCP marker and the thumb tip. These segments are shown in figure 2:1.

For joint kinematics analysis the angles were defined as follows: (a) the PIP joint angle was defined as the vertex of the proximal phalangeal segment and the distal phalangeal segment; (c) the MCP joint angle was defined as the vertex of the proximal interphalangeal segment and the virtual segment.

### **2.8.12 Research design.**

A single-factor independent groups design was used.

### **2.8.13 Apparatus.**

The patients were required to grasp a black compact disc (CD) case which was 15.5 cm deep, 16.5cm high and 5cm wide. Infrared reflective markers were fixed to the participants' hands in the locations shown in figure 2:1. This was done with clear, double-sided adhesive tape manufactured specifically for this purpose. Movement was captured using a six-camera optoelectronic system, capturing at 50Hz (BTS

Bioengineering, Milan). The cameras and experimental set up are shown in figure 2:2.

The video and commentary explaining the experimental task to the patients were presented on a laptop computer. The MI and relaxation tasks were delivered via a tape recording. All recorded scripts were read by a female native speaker of Colombian Spanish with specialist training in media and communication. Electronic labelling of the reflective markers was accomplished using Smart Tracker software (BTS Bioengineering, Milan).

The definition of joint angles, along with the measurement of joint path, velocity and acceleration was completed using Smart Analyzer (BTS Bioengineering, Milan). These data were then exported as text files and stored on spreadsheets. Data reduction was accomplished using Matlab (Mathworks Inc., Natick) version 7.4. The syntax used is given in appendix 2:5.



Rear view



Side view

*Figure 2:2. Views of experimental set-up*

#### **2.8.14 Procedure.**

Each patient received three sessions of conventional therapist-facilitated physical treatment on their hemiplegic hand. The facilitation was aimed at improved activation of the intrinsic hand muscles. Each session lasted 15-20 minutes. All conventional physical treatment was based on an identical treatment protocol that was designed by the researcher, whose clinical experience and training are described above. The details of the protocol are described in appendix 2.6. The three treatment sessions took place in the hospital setting. When these treatment sessions were completed the patients then attended the movement analysis laboratory. Once there, they were treated according to the procedures outlined below. Limitations regarding the timing of the physical treatment are outlined in the discussion section of this chapter.

#### **2.8.15 Experimental procedure.**

Participants were seen separately. Upon arrival in the laboratory, the door was closed and they were asked to sit comfortably on a chair in front of a table. All participants then watched a video that displayed the formation of a grip using the lumbrical action. The hand in the video formed the lumbrical grip without grasping anything five times. The video then showed the hand grasping the black CD case using the lumbrical grip, also repeated five times. An accompanying recorded commentary explained that the viewer would be asked to perform this task themselves. If the person had left sided hemiplegia, then the video showed a left hand forming the grip and if the person had right sided hemiplegia then the video showed a right hand forming the grip.

A coin was then tossed by the researcher to randomly allocate patients to group. This is regarded as an appropriate randomisation technique as part of the PEDro checklist (Moseley, Herbert, Sherrington & Maher, 2002). The randomisation process broke down for the last person tested. This issue is addressed in the discussion section of this chapter.

The experimental group listened to recorded instructions beginning with a guided relaxation task. This was based on the principles of “passive muscular relaxation” as described by Payne and Donaghy (2010, p. 71). The guided relaxation

task was repeated six times, with each repetition lasting on average 20 s. This was then followed by recorded instructions on KMI, focusing on the hand lumbrical action. The KMI task was repeated six times, with each repetition lasting on average 33 s. In all, this recording lasted for 7 min 15 s.

The control group listened to a recorded guided relaxation task only, which made no mention of the lumbrical muscles. This began with a guided breathing exercise. This was repeated three times, with each repetition lasting on average 24 s. This was then followed by a guided muscular relaxation task, which was repeated five times. Each repetition lasted on average 29 s. This recording lasted for 4 min 39 s. The details of these recorded scripts are provided in appendix 2:7.

The file was then positioned on the same side as the participant's hemiplegic hand. It was placed on its end with the spine facing the participant in their frontal plane. The participants then grasped the file 14 times with their hemiplegic hand. They were reminded verbally to try to use the grasp pattern shown in the video.

#### **2.8.16 Data analysis and reduction.**

In the BTS Smart Capture System (BTS Bioengineering, Milan), the three dimensional marker coordinates are first interpolated automatically using cubic splines, which are discussed in more detail below. They are then low-pass filtered automatically using a filter with a 10Hz frequency cut-off point (Menegoni et al, 2008). These data are stored by the system in text definition files (TDF).

Following capture, the TDF files were opened using Smart Tracker software (BTS Bioengineering, Italy: Milan). The Smart Tracker software allowed each marker to be labelled following the hand biomechanical model illustrated in figure 2:1. Smart Analyzer software (BTS Bioengineering, Italy: Milan) was then used to define the joint angles of interest, which are also shown in the biomechanical model illustrated in figure 2:1. Angular paths were then calculated for MCP and PIP using the same software. Following this, these data were differentiated twice using the software: the first differentiation gave the angular velocity profiles for the two joints; the second differentiation provided the angular acceleration profiles. These data were stored in TDF files. The TDF files were then exported as text files for storage in spreadsheets and offline processing.

There were three spreadsheets for each joint: one for joint path; one for velocity; one for acceleration. Since angular velocity and acceleration for each joint were derived from the initial angular path, the lengths of the columns for each individual movement were the same. The initiation and termination of each grasping movement was then identified visually from the computer monitor. Data lying outside of these initiation and termination points were deleted from the spreadsheet. The issue of visual identification of these points is addressed further in the discussion.

Each of the separate 14 grasps performed by the patients had slightly different time durations. To make these movements statistically comparable it was therefore necessary first to normalise the time. The process of time normalisation was accomplished using Matlab software (The Mathworks Inc., Natick: USA). The syntax used for this is included in appendix 2:5. Each grasp was normalised to two seconds.

The data were then interpolated. This interpolation was accomplished using splines. Splining involves the identification of third order polynomials which allow the estimation of the curve connecting two consecutive points of the data. Splining had several advantages for this research. First, the interpolation of an array of data using cubic splines is felt to provide a curve whose shape approximates closely to that of the original data (Robertson & Caldwell, 2004). Second, splining is thought to provide a good interpolation method for data captured at low sampling rates (Robertson & Caldwell, 2004). Since these data were captured at a relatively low rate of 50Hz, this was considered to be an appropriate technique. Third, splining is not based on the assumption that the data show linearity, as is assumed in linear interpolation (Robertson & Caldwell, 2004). Since it was predicted that movements in the distal joints of the hemiplegic hand were likely to show high levels of non-linearity, it was felt that splining might therefore be a useful approach to data interpolation. The Matlab (The Mathworks Inc., Natick: USA) syntax used to accomplish the interpolation with cubic splines is also included in appendix 2:5.

Following time normalisation and spline interpolation, ten ensemble-averaged joint paths during grasping for the PIP and MCP joints for each patient could be constructed. This means that there were ten columns of data for each of the joint paths that all had an equal number of data points. Since there were 101 data points in each



column, each point represented one per cent of the ensemble-averaged movement (Robertson & Caldwell, 2004).

The use of ensemble averaging was especially useful in this research. This was because the concern was to capture the kinematic characteristics of the movement of the joint throughout the grasp. Without ensemble averaging, isolated mean values would have had to be calculated from the raw data for each individual grasp. Arguably, this would not have provided a clear picture of the entire movement pattern and would have made it very difficult to identify, for example, the typical time points at which the grasp phase changed from the transport (approaching) phase to the manipulation (holding) phase for each patient. In addition, it would have prevented the graphical display of averaged movements for each group. This is because, while the raw data for each individual grasp could be represented by a single curve, the data from more than one grasp could not, as each grasp was of a different duration. Ensemble averaging allowed such graphical representation to be accomplished. Figure 2:5, for example, shows the curves of ensemble-averaged curves for PIP path, angular velocity and acceleration for both groups. Overall, it has been suggested that the technique of time normalisation, interpolation with splines and the calculation of ensemble-averaged data allows the “kinematic patterns” of movement to emerge more clearly than when working with mean values calculated from non-ensemble-averaged data (Robertson & Caldwell, 2004, p. 26).

As shown above, ten ensemble-averaged grasps for each patient were calculated. Where possible, these were calculated from data for the central grasps, so that the first two and last two grasps were removed from the analysis. This was to control for initial learning and fatigue effects towards the end of the movements. However, artefactual issues occasionally meant that this was not possible, for example if one of the markers was temporarily obscured, and grasps were sometimes selected from nearer the start or end of the sequence of repeated movements.

Neither normative data nor guidelines were identified as to the appropriate number of repeated movements to include in an analysis of angular rotatory movements in hemiplegic finger joints. In addition, Kontaxis et al. (2009) have commented that no definite protocols exist as to the optimal number of repeated

movements needed to ensure that a representative ensemble-averaged profile can be constructed for an upper limb movement (Kontaxis et al., 2009). These authors state that this decision is dependent on factors which include the task to be performed and the disability level of the actor (Kontaxis et al., 2009). They do, however, suggest a minimum of three repetitions. Given the high levels of variability encountered in hemiplegic upper limb movements (Cirstea et al., 2003; Levin, 1996), it was felt appropriate to include ten repetitions in the analysis.

#### **2.8.17 The identification of phases of the hand movement.**

The hand movement involved three phases: (a) an approach phase, as the hand was brought towards the CD case; (b) a holding phase, as the hand grasped the CD case; (c) a leaving phase, as the hand released the case. The initiation of the movement had already been identified visually from the representation of the infrared markers on the computer monitor, opened using Smart Tracker (BTS Biosystems, Milan: Italy). It was only necessary, therefore, to identify the point at which the ensemble-averaged PIP and MCP joints appeared to grasp and to leave the CD case, in order to identify the data which represented each distinct phase. The method used to accomplish this is described below.

First, the ensemble-averaged joint paths for each patient were inspected visually. As grasping occurred, the magnitude of the joint angle changes was expected to reduce because the joints would be partially stabilised by grasping the CD case. Once such a point had been identified visually from the curve for joint path, it was then verified using the ensemble-averaged velocity curve for that joint. Since the joints were nearly stabilised as the hand grasped the object, the velocity at that point should have been at or near zero. If the values on the ensemble-averaged velocity curve which corresponded to the point identified on the joint path for the start of the holding phase were indeed at, or near, zero, this was taken as verification that visual inspection had identified the phase change correctly.

The same approach was taken to identify the leaving phase. Here, the start of the phase was again first identified visually from the curve of the ensemble-averaged joint path. It was predicted that the velocity value for the joint just before that point in the joint path curve should be at or near zero. It should then have shown a sudden

change in slope as the hand released the case. If such a point could be identified on the velocity curve near the equivalent point identified from the curve representing that joint's path, then this was taken as verification that the end of the holding phase and the start of the leaving phase had been rightly identified. A graphical illustration of this process is provided in figure 2:3. Some of the theoretical implications of this process of phase change identification are addressed further in the discussion.

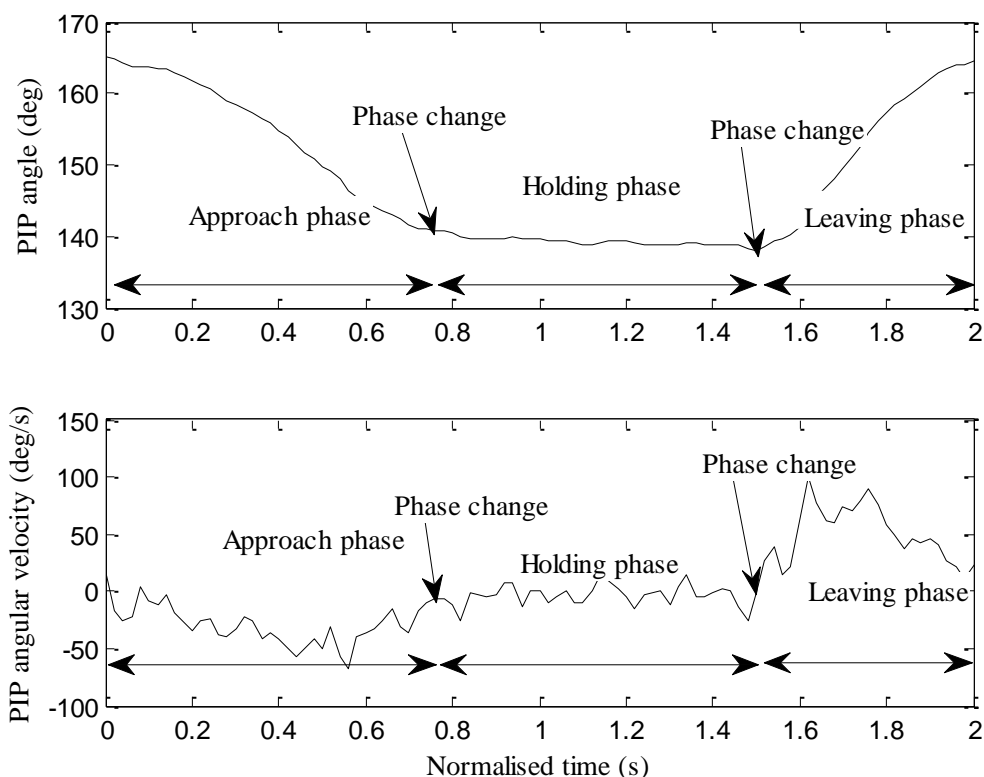


Figure 2:3. PIP angular path and velocity for participant one, showing phase changes.

## 2.9 Results

### 2.9.1 Participant flow through the study.

Participant flow through the study is shown in figure 2:4.

### 2.9.2 Statistical analysis.

Dependent variables were compared for significant difference using an independent samples *t* test with *p* set at  $< .05$ . Where there was unequal variance in the data, testing was done using a *t* test with degrees of freedom adjusted to account for

this. The issue of unequal variance is discussed further in the discussion section of this chapter. Spearman's product moment correlation coefficient ( $r$ ) was also used. Throughout the results section, 'experimental group' refers to the group which use KMI, while 'control group' refers to the group which used relaxation only.

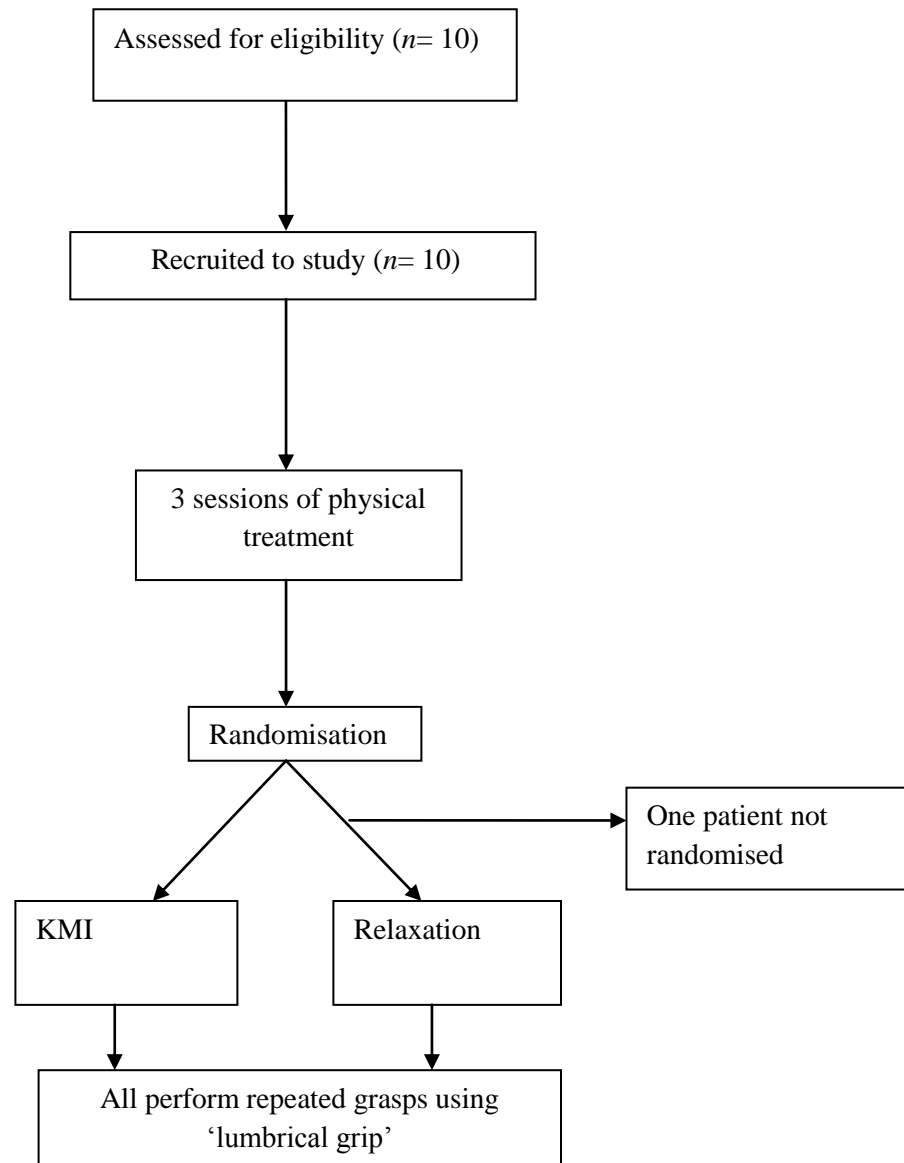
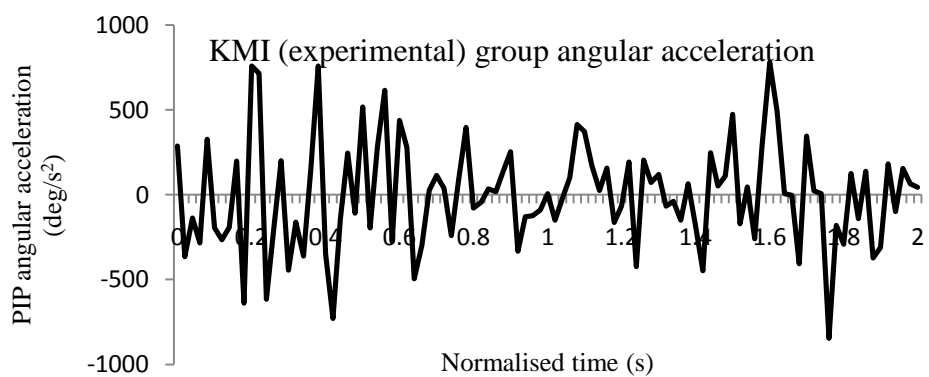
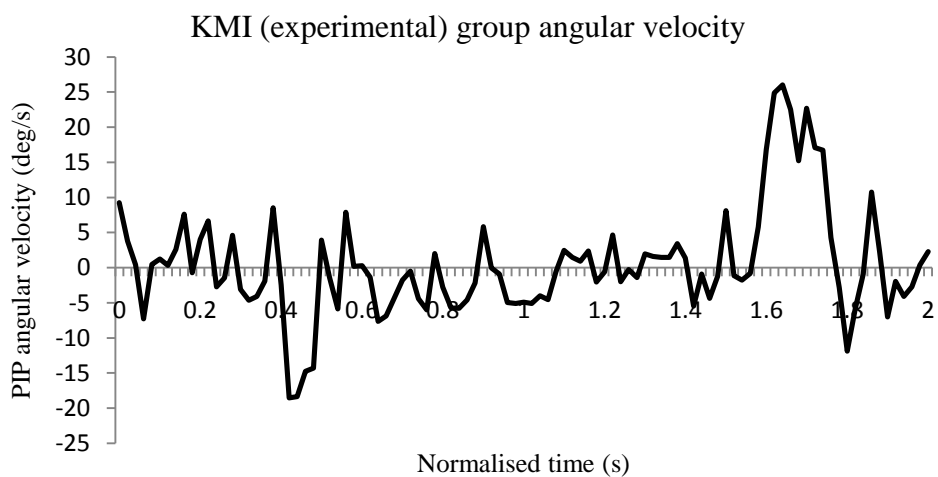
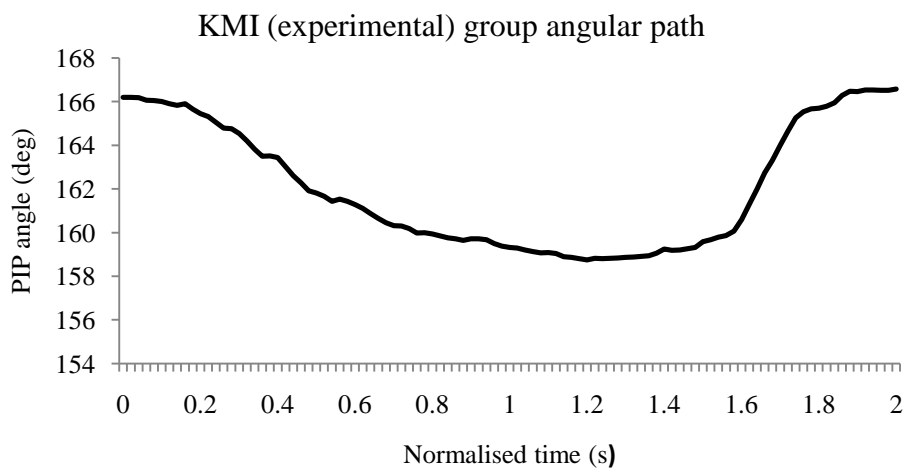


Figure 2:4. Participant flow through study.



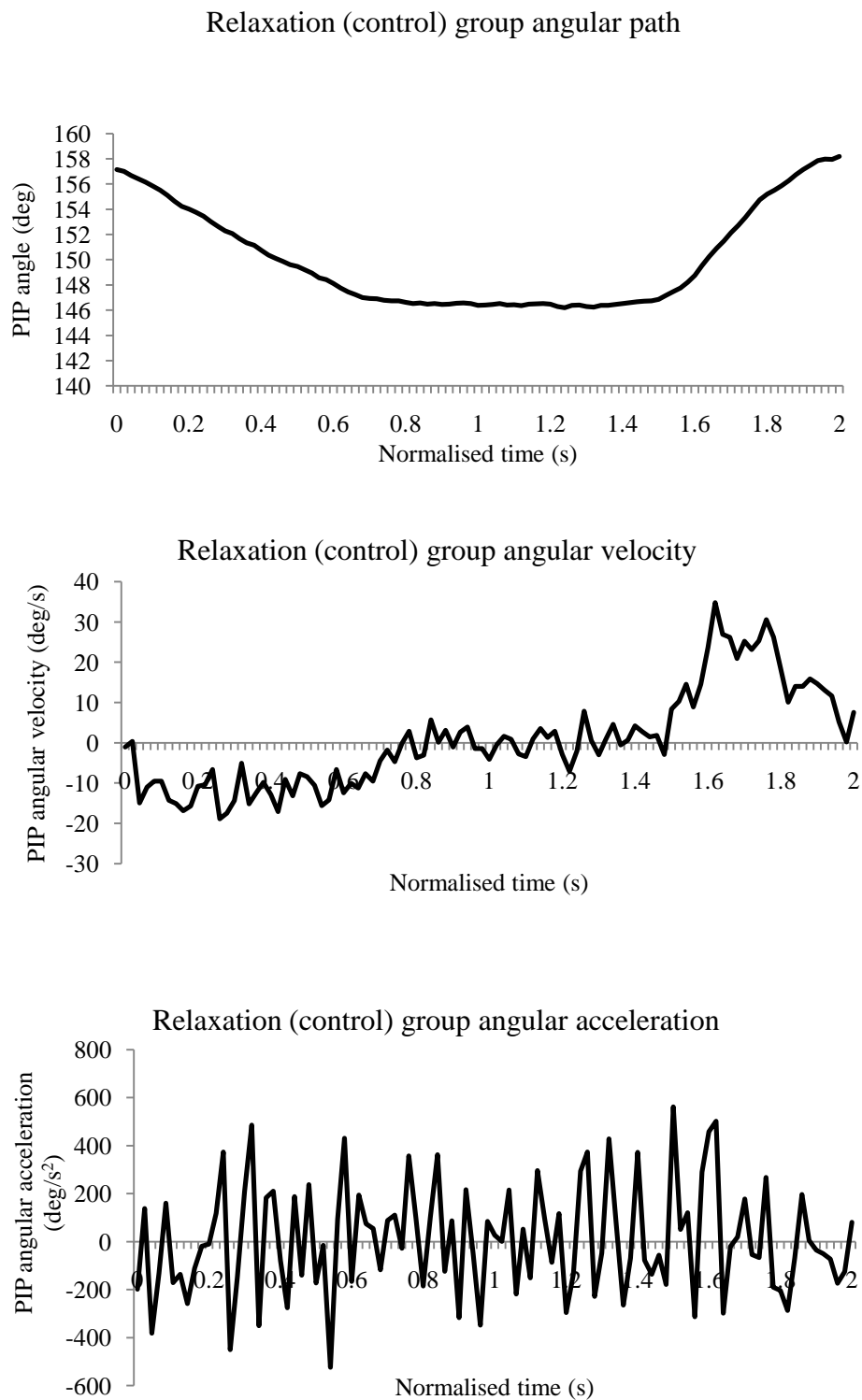


Figure 2:5. Ensemble-averaged PIP angular path, velocity and acceleration for KMI and relaxation groups.

### **2.9.3 Mean PIP angle.**

The mean (SD) PIP angle for the control group approach phase was 152° (4.48°), the mean PIP angle for the experimental group approach phase was 163.88° (10.46°). Although the magnitude of the experimental group was larger, as predicted, these differences were found to be non-significant,  $t(5) = -2.33$ ,  $p = .067$ .

The mean (SD) PIP angle for the control group holding phase was 150.1° (6°) and for the experimental group 159.45° (9.42°). Again, these differences were not found to be significant,  $t(7) = -1.87$ ,  $p = .103$ .

### **2.9.4 Mean MT.**

MT for all three phases of the grasp movement was calculated from the raw MTs for each grasp for each patient. The mean (SD) MT for the control group was 3.54 (1.01) s, while the mean MT for the experimental group was 2.12 (0.51) s. These times were significantly different  $t(6) = 2.793$ ,  $p = .031$ . The mean MTs for both groups are shown in figure 2:6.

### **2.9.5 Mean MT during the approach phase.**

It was of interest to find out if the movement time was different during the approach phase of the grasp only. It was found, however, that there was no significant difference between the groups for this comparison,  $t(4) = .191$ ,  $p = .858$ .

### **2.9.6 Mean PIP angular velocity.**

Mean (SD) PIP angular velocity for the control group during the approach phase was -11.75 (12.726) deg/s and for the experimental group, -4.89 (13.798) deg/s. The mean velocities for each group were not significantly different,  $t(8) = -0.817$ ,  $p = .437$ .

Mean (SD) PIP angular velocity for the control group during the holding phase was -0.424 (2.281) deg/s and for the experimental group 1.398 (5.471) deg/s. The difference between these values was not significant,  $t(5) = -0.687$ ,  $p = .522$ .

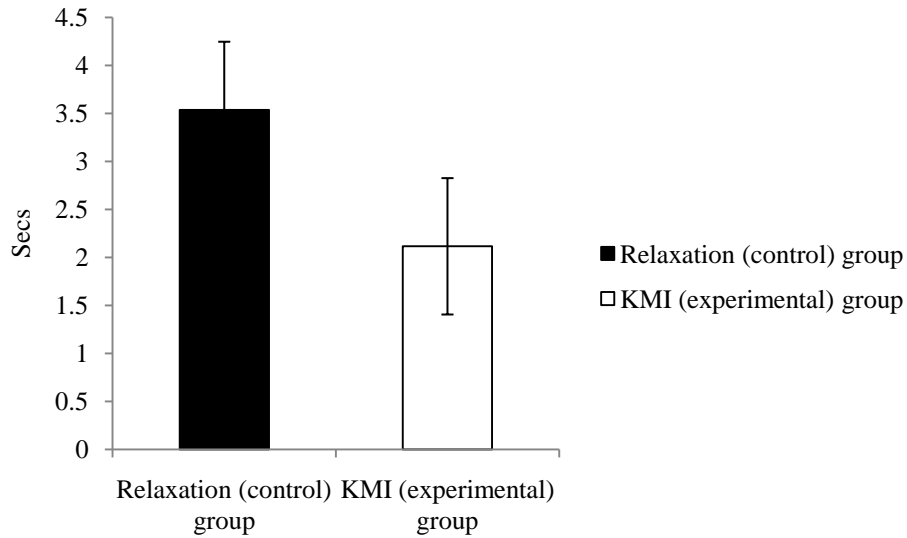


Figure 2:6. Mean MT for whole grasping movement.

### 2.9.7 Time to maximum PIP velocity.

The mean (SD) time to maximum angular velocity for the control group PIP was 1.24(0.438) deg/s and for the experimental group PIP, 1.36 (0.462) deg/s. These values were not significantly different,  $t(8) = -0.283$ ,  $p = .785$ .

### 2.9.8 Number of MUs during the approach phase.

The mean (SD) number of MUs during the approach phase for the control group was 7.8 (1.64) and for the experimental group, 6.6 (1.81). These values were not significantly different,  $t(8) = 1.095$ ,  $p = .305$ .

### 2.9.9 Correlation between MCP and PIP joints during the holding phase.

Spearman's product moment correlation coefficient ( $r$ ) was calculated for the mean MCP and PIP joint angles during the holding phase of the movement. These values are shown for both groups in table 2:3. The correlation coefficient for the control group was found to be non-significant,  $r(5) = .672$ ,  $p = .214$ . Neither was the correlation coefficient for the experimental group statistically significant,  $r(5) = .231$ ,  $p = .708$ .



Table 2:6

*Pearson's Product Moment Correlation Coefficient (r) for MCP and PIP Mean Joint Path Magnitude for the relaxation and KMI Groups During the Holding Phase*

	MCP relaxation	MCP KMI
PIP relaxation	0.672	PIP KMI 0.231

## 2.10 Discussion

### 2.10.1 Mean magnitude of PIP angle.

The mean PIP joint angle during the approach phase of the movement was larger for the experimental group, although this just missed statistical significance ( $p = .067$ ). This provides support for the hypothesis that KMI treatment based on the promotion of the lumbrical action, in addition to conventional physical treatment, leads to greater extension of the PIP joint than conventional physical treatment alone.

This may be a useful finding from a clinical point of view because the ability to extend the fingers following stroke has been seen as an indicator of the likelihood of the recovery of the rest of the hemiplegic upper limb. Beebe and Lang (2009), for example, found that higher active range of movement in the middle finger one month post-stroke was significantly correlated with the quality of upper limb movement two months later. Smania et al. (2007), meanwhile, found that active finger extension at fourteen days post-event was significantly correlated with upper limb recovery 180 days after the stroke.

If the finding of higher PIP extension in the KMI group can be explained in relation to the use of MI training, this would suggest that KMI had had a highly specific effect that led to the activation of the lumbrical muscle. This may make sense in the light of other studies which have shown that MI might work by facilitating raised activation levels in targeted intrinsic hand and foot muscles. Yue and Cole (1992), for example, found that individuals who mentally trained abduction of their little finger displayed a 22% increase in force of abduction over four weeks training.

Mulder, Zijlstra, Zijlstra, and Hochstenbach (2004) found that a group of individuals who had a limited ability to abduct their big toe improved significantly in their ability to execute this action following two sessions of mental training. Cicinelli et al. (2006), meanwhile, used TMS to measure the size of the cortical map during KMI of the abductor digiti minimi (ADM) in the affected hemisphere of stroke patients. These authors found that, while at rest, there were significantly fewer excitable areas in the ADM region in the affected hemisphere when compared with the unaffected hemisphere. However, during MI the difference between the two hemispheres was nearly eliminated. In addition, there was no significant increase in cortical volume or area during MI for a control muscle, the extensor digitorum, suggesting that the effect of MI had been specific for the target muscle (Cicinelli et al., 2006). Furthermore, Porro et al. (1996) used fMRI to scan the brains of healthy individuals performing and using KMI of a thumb opposition activity. The authors found significantly higher activation in M1 during KMI than during resting. These studies therefore seem to show that MI may have highly specific effects on the dexterity, force and cortical activity related to an intrinsic hand muscle or an activity which may involve the intrinsic muscles as a group. This may help explain the finding in the current study that patients using KMI of the lumbrical action were able to produce more extension in the PIP joint of their hemiplegic index finger.

It may be useful here to refer to theoretical work on MI. Jeannerod (2006) has linked MI to the motor planning, pre-execution stage of movement. This author argues that MI activates brain regions which overlap those which would be involved in actual movement execution. In the present study, therefore, it may be the case that KMI improved the ability to form a motor plan for production of the lumbrical action, thus producing a higher degree of extension in the hemiplegic PIP joint for the KMI group. One might speculate, for example, that KMI promoted the establishment of a motor program for lumbrical activation, perhaps through a process of Hebbian synaptic learning (Kandel, 2000; Kandel, Jessell, & Sanes, 2000; Johansson, 2000), such as has been thought to underpin the learning which some studies have linked to the MI process (Ietswaart, 2011).

### **2.10.2 Correlation between MCP and PIP joints.**

It was predicted that KMI, in line with Jeannerod's (2006) hypothesis and other motor theoretic positions (for example, Schmidt, 1975), might work to improve the overall pattern of joint configurations which would be expected on successful execution of the lumbrical action. Such a pattern would be distinguished by flexion of the MCP combined with extension of the PIP. This would also make sense physiologically, as the lumbrical muscle produces flexion of the MCP and extension of the PIP in a single contraction. One benefit of the present study in this respect was that measurements were made of the correlation of these joint paths, allowing the extent to which this pattern of joint configuration was produced to be assessed.

Specifically, it had been predicted that a stronger lumbrical action would lead to a greater negative correlation between the two joints in the KMI group. However, the correlation between the MCP and PIP joint paths was actually lower during the holding phase of the grasp for the KMI group, as compared with the relaxation group. The correlation was, in addition, non-significant. Furthermore, it did not meet the demands made in the initial hypothesis, that the correlation should be negative. Thus, it may be difficult to draw firm conclusions based on the partial confirmation of the hypothesis regarding the magnitude of PIP extension during the approach phase, without having simultaneous confirmation of the prediction that KMI would be linked to stronger negative correlation between the joints.

Another sense in which the initial hypothesis has only partially been supported by this finding of higher extension in the PIP joint in the KMI group, is that this occurred during the approach phase of the movement. The target action for the participants, as presented in the video, was the action of grasping the CD case, not the approach to it. Therefore, if KMI had been involved in promoting a motor program based on the performance of the specified task, one would have expected the higher PIP extension shown by the KMI group to occur during the holding phase of the movement rather than the approach phase. The mean PIP angle for the KMI group during the holding phase of the movement was indeed higher than that for the relaxation group, however, the difference between the two groups was non-significant and the confidence level was lower than that for the approach phase.

### **2.10.3 Mean MT.**

Mean MT for the entire movement was found to be significantly faster for the KMI group than the relaxation group. This may indicate that KMI had a facilitatory effect on the timing of the task performance. That MI training may have such an effect seems to be confirmed by studies such as that of Hewett et al. (2007), who noted an increased reaching velocity in upper limb hemiplegia following MI treatment. Other studies have found an increase in the velocity of finger movement sequences following MI training (Avanzino, 2009). These findings, therefore, may point to a useful role for KMI in improving this aspect of motor performance in the hemiplegic upper limb. There are, however, a number of implications from this finding for the predictions made in the current research which may need further examination. These are discussed below.

The prediction was that KMI would have a highly specific effect on the activation of the hand lumbrical muscles. However, the finding regarding increased MT for the KMI group seems to partially contradict this. This is because the overall MT includes all three phases of approaching, holding and leaving the object. In this sense, activation of more proximal upper limb muscles is likely to have been heavily involved. This may, therefore, suggest that, while KMI did have a facilitatory effect, this effect was a very general one. This could mean, for instance, that the very act of focusing more attention on the hand during KMI may have led to the increased movement time. In turn this suggests that KMI may represent part of much more generalized process of attention, rather than a mechanism which provides a specific stimulus to a specified muscle.

The finding regarding MT was tested further by comparing the movement times during the approach phase of the movement only. If there was a difference here which favoured KMI, then this may have provided evidence that KMI had an effect at the planning stage of the movement. However, there was no significant difference identified between the groups. This lends further support to a generalised effect of KMI on the gross MT.

A second issue raised by this faster MT is that MT per se was not specified as part of the KMI treatment task. Rather, the task emphasized the overall patterning of

the grasp posture, as opposed to the speed of the movement. Indeed, the movements were self-paced by the patients. This was a deliberate choice in the experiment design. As pointed out above, it was felt that a timing imperative would overload the patients, given the already difficult nature of the grasping task they were being asked to undertake. In addition, it was hoped that self-paced movements would allow a more natural picture of the reaching and grasping of these patients to emerge, in spite of the grasp posture constraint included in KMI. The fact that movement was still significantly faster in the KMI group may provide further evidence, therefore, that this particular difference may have been more a by-product of general attention to the hemiplegic limb, rather than a specific facilitation from KMI. This position certainly finds some support from MI literature. Jackson et al. (2001), for example, put forward three possible explanations for the efficacy of MI training. First, they suggested that the mental training might have a motor priming effect, leading to very specific activation of targeted muscles. They labelled this position the “psychoneuromuscular” theory (Jackson et al., 2001, p. 1137). Second, MI may work by facilitating the rehearsal of the cognitive aspects of a movement, thus improving its symbolic representation in the cortex and improving learning. Third, it may have a much more general affect based on improving the person’s motivation and cognitive focus on the goals of movement. The results outlined above related to the MT for the MI group may support this third position.

It is interesting to note that, despite the reduced MT for the movement as a whole, the mean angular velocities for the PIP joint during the approach phase and the holding phase were not significantly different for either group. Indeed what stands out is the very low confidence level of the differences between the two values. This may suggest that any effect noted on the global temporal characteristics of the movement, noted above, was not effective at the level of the angular kinematics of the PIP joint. Once again, this may point to a more general effect of KMI, rather than a highly effector specific one.

#### **2.10.4 Time to peak velocity.**

In the introduction it was explained that improved motor performance in reaching and grasping tasks has been associated with a shift in the location of temporal

kinematic landmarks during the action. As shown above, writers have related changes in the timing of the peak velocity to the planning level of movement (Lin et al., 2007; Jeannerod, 1986; Van Vliet & Sheridan, 2007). A later time to peak velocity for the PIP joint was predicted for the KMI group in the present study. However, no significant differences were found between the two groups. If the views of other authors are correct, this may suggest that KMI did have not any influence at the planning level of the movement, as might have been predicted on the basis of Jeannerod's (2006) motor simulation hypothesis. This would also lend support to the argument being developed in this discussion, that the effects of KMI noted, for example, in a significantly faster MT, were more a by-product of greater attention being paid to the hand.

#### **2.10.5 Number of MUs.**

A further kinematic variable measured, the number of movement units (MUs) during the approach phase, was also intended as a measure of motor planning processes during the reach. The origin of the idea that this variable might be related to motor planning comes from the work of Crossman and Goodeve (1963), who found that the movement of the upper limb towards a target was not made up of one single phase of acceleration and deceleration, but actually of multiple corrections marked by successive velocity peaks. These ideas have been developed by Flash and Hogan (1985), Nagasaki (1989) and Lin et al. (2007). Specifically, Lin et al. (2007) predicted that more efficient motor control in adult hemiplegia would be characterised by fewer acceleration peaks, and therefore fewer MUs. They argued that this would indicate a move towards more feed-forward based control of the limb path. In the present study, no significant difference was found between the MUs for the KMI and relaxation group. Indeed, the relaxation group had slightly fewer MUs. These results are discussed further below.

The lack of significant differences for the number of MUs could be taken as further support for the notion that KMI did not have an effect at the level of motor planning. This is because the number of MUs during the transport phase has been seen by writers, as shown above, as indicative of the motor control strategy being used to guide the movement. Fewer MUs would presumably indicate a move towards a higher

level of impulse control, as described by Woodworth (1899), and greater reliance on the initial movement parameters specified by the motor program. Conversely, a higher number of MUs would indicate greater use of online sensory feedback to guide the movement. If these views are correct, it may be the case that KMI as used in this study did not lead to a more efficient motor program based control of the movement.

It may also be important to bear in mind in relation to this discussion that the studies of Crossman and Goodeve (1963), Flash and Hogan (1985), Nagasaki (1989) and Lin et al. (2007) all looked at the movement of the upper limb as a whole towards a target. They were not studies based on the measurement of angular kinematics. In Lin et al.'s (2007) experiment the MUs were measured from a single wrist marker. The present study took a very different approach, in that the angular motion of a distal joint was measured as it moved towards a target posture. It is not clear, therefore, whether the theoretical assumptions made by the aforementioned authors would apply to the present research. This remains an open question, as, to the best of the author's knowledge, the angular kinematics of distal joints in hemiplegia have not previously been measured.

## **2.11 Methodological Issues Raised by the Experiment**

### **2.11.1 The issue of massed versus spaced MI practice.**

Motor control researchers have debated the most advantageous balance of practice and rest periods with regards to motor learning over many years (Schmidt & Lee, 2005). Generally, it has been argued that longer rest periods between practice sessions promote more effective learning, suggesting an advantage for distributed rather than massed practice (Schmidt & Lee, 2005). Donovan and Radosovich (1999) carried out a meta-analysis of motor control studies and were able to show that, although this assumption was broadly correct, it did not necessarily apply in all cases. Tasks with a high cognitive and motor skill demand, for example, did not seem to show such a marked learning benefit with longer rest periods. Meanwhile, for simple tasks, the advantages of spaced practice increased as the timing of rest periods decreased. This was a somewhat counterintuitive finding, as shortening rest periods would seem to bring the task more in line with massed practice (Donovan & Radosovich, 1999). Donovan and Radosovich (1999) also found that there was no

clear advantage for either training schedule with relation to the retention of skill following training.

The question of the optimal duration and intensity of physical training in neurological rehabilitation has been a neglected area (Dobkin, 2004). Animal models have, however, provided some data. It has been shown, for example, that massed physical training in rats following an induced stroke may actually lead to an increase in the size of the lesion (Dobkin, 2004). On the other hand, massed treadmill training in other quadrupeds with stroke has shown a beneficial effect on gait (Dobkin, 2004). Dobkin (2004) stressed, however, that such studies may have only limited applicability to the rehabilitation of humans with stroke, although findings regarding the optimal timing of physical training in neurologically intact individuals, as described above, may be of relevance.

Little work has been carried out on the benefits of different timing schedules in relation to motor imagery as used in stroke rehabilitation (Schuster et al. 2011). Schuster et al. (2011) have attempted to address this through the use of the “PETTLEP” model as a framework within which to analyse research on MI in neurological rehabilitation. PETTLEP is a systematic approach to the construction of MI training programmes, originally developed within sport psychology (Holmes, 2001). The title is an acronym drawn from a proposed list of components which MI should include: physical, environmental, task, time, learning, emotion and perspective. The specific question of massed versus distributed timing was not addressed by Holmes (2001). However, using PETTLEP, Schuster et al. (2011) were able to show that MI studies which showed positive outcomes employed MI practice sessions which lasted around 17 min each, spread out over 34 separate sessions. In addition, they were able to recommend that there should be a maximum of two MI sessions every minute (Schuster et al., 2011).

Schuster et al. (2011) have highlighted the heterogeneity of MI research protocols, making meta-analysis a challenge. Nonetheless, analysis of some existing work may allow some further tentative conclusions to be drawn regarding massed and spaced practice. Some relevant studies are discussed below.

Allami et al (2008) conducted research on the optimal balance of MI and



physical practice in motor learning with 25 healthy individuals. The task was to pick up an object using a precision grip, upon which was balanced a marble, from different orientations and place it in a slot. Two hundred and forty trials over 45 min were needed to learn the task, which, while not explicitly stated, seems to imply a massed approach to physical practice. Four experimental conditions were used. In the first, participants practised 240 physical trials of the task. The second deployed first person MI of the task for the first 25% of 240 trials, followed by physical practice, the third used MI for 50% of trials prior to physical practice and the fourth used MI for the first 75% of the trials, again completing the remainder with physical practice. For the first executed trial, the groups using 50% and 75% MI showed significantly shorter MT when compared with the physical practice alone group or the 25% MI group. This initial result would seem to suggest an advantage for massed MI. However, in the final executed movement there was no difference in MT between the 50% MI group, the 75% MI group and the physical practice only group.

Allami et al. (2008) also analysed their data by breaking the training task down into two parts based on difficulty. The more simple first movement was reaching and grasping the object. The second movement was inserting it into the slot, and was considered to be more complex. For the last trial of movement one there were no MT differences, suggesting that the amount of MI training within the 45 min period did not have any influence on the simpler task. However, for movement two, the 50% and 75% MI groups showed a significantly faster MT at the final trial than the other groups. These findings seem to suggest that massing more MI within a set period of time is only beneficial for more complex movements. This may be in agreement with the finding of Donovan and Radosovich (1999) that more demanding tasks benefited less from distributing lengthy rest periods throughout training, although the tasks considered by those authors were arguably at a far higher level of complexity than that analysed by Allami et al. (2008).

Papaxanthis et al. (2002) studied the effects of spacing MI following movement execution on the matching of mental and executed MT. Sixteen healthy participants were asked to imagine and execute a walking task and a writing task. One group performed ten physical trials, followed immediately by 10 MI trials. They

performed ten more MI trials after 25m, 50m and 75m rest periods. Thus the time elapsed since the initial physical task performance increased with each trial. However, the authors did not identify any differences between the real and imagined MT for any of the conditions. This suggested that the distribution of the mental practice sessions made no difference to the accuracy of the imagined time (Papaxanthis et al., 2002). This is in contradiction to the findings outlined above from Schmidt and Lee (2005) and Donovan and Radosovich (1999) that spaced practice seems to benefit performance.

Courtine et al. (2004) conducted a study in which 20 healthy participants were required to imagine or execute a walking task along a horizontal or upward or downward sloping surface. The first group performed massed MI prior to movement execution, while the second used MI distributed around trials of actual walking. The individuals were required to estimate their mental walking times, and these were matched against their actual walking times. There were no differences between these times, indicating that the spacing of the MI and movement execution had had no effect (Courtine et al., 2004). There was, however, greater variability in the estimates of imagined movement time. In addition, this variability was lower for the distributed MI condition. This study did not examine the effects of distributing MI and rest periods. However, it draws attention to the importance of considering the order of physical and mental practice.

Malouin et al. (2004b) used massed training delivered in seven blocks over 25-30 min. Twelve persons with stroke and six healthy individuals were required to perform one physical sitting to standing transfer, followed by five MI trials of this task; this constituted one block. It was found that the individuals with stroke were able to significantly improve their ability to load their more affected leg, thus improving their balance. Moreover, these improvements were still evident when retesting was done 24 hours later. Malouin et al. (2004b) also note that no change in the timing of the tasks was observed, suggesting that a key gain may have been an improvement in the quality of movement control, rather than the speed of execution. Overall, these authors suggest that the results of this and an earlier study (Malouin et al., 2004), indicate that the spacing of physical practice and MI should be in the ratio 1:5

(Malouin et al., 2004b). The results also seem to suggest that massed practice, or at least practice with comparatively short rest periods in between, may be effective for people with lower limb hemiplegia when using MI and physical practice together.

### **2.11.2 Limitations of current study regarding spacing of training sessions.**

In the current study the average length of MI was 32 s, with 6 sessions. There was an average break of 4.3 s between each session and the MI was therefore very close to massed practice. The MI was preceded by three physical practice sessions, as described above. Thus the ratio of physical to mental practice was near to that recommended by Malouin et al. (2004b). It is felt, however, that a limitation of the research is that the precise spacing of the physical trials, both in relation to each other, and in relation to MI, was not adequately controlled. For example, it was inconvenient for some individuals to attend the hospital for physical practice sessions on consecutive days, so that the spacing between the sessions could not be standardized for all participants. In addition, the relative timing of the physical and mental practice sessions was not the same for all. For example, some individuals completed all of the physical practice sessions in the days prior to visiting the laboratory to perform MI, while some had the final physical practice on the same day, a couple of hours before attending the laboratory. Given the emerging evidence that the distribution of rest, physical practice and MI may exert an effect on aspects of motor imagery and motor performance, it is felt that it would be important to control these aspects more carefully in future work.

### **2.11.3 Limitations regarding the timing of physical therapy interventions.**

A further limitation concerns the timing of the physical therapy intervention. The precise timing of each session was not recorded, although the individual sessions lasted for 15-20 min. A more accurate record of these times would have been desirable and may have contributed to the rigour of the study. Furthermore, the space between the sessions was not standardised. For example, in some cases two sessions were delivered on the same day, with a break of around 15-20 min in between. Again, more accurate recording of the exact timing of the breaks between treatment would have added to the study's precision.

As shown above, another issue regarding the timing of the physical treatment

was that some participants received their final physical treatment on the same day as motion capture was undertaken. There was a break of around two hours prior to attendance at the laboratory and motion capture. These individuals may then have benefited from more recent physical treatment prior to performing the experimental grasping task, which may have affected the results. Standardisation of the timing of physical treatment should therefore be aimed at in future work.

#### **2.11.4 Frequency of motion capture.**

As pointed out above, the motion capture frequency was 50 Hz, which was comparatively low. However, other studies of upper limb hemiplegia using kinematics have used comparable frequencies. Lin et al. (2004), for example, captured at 60 Hz, while Iwamuro, Cruz, Connelly, Fischer, and Kamper (2008) used a rate of 30 Hz, and in neither case was the capture rate highlighted as problematic. Of course, a faster capture rate would always be desirable and allow greater accuracy of measurement, however, a rate of 50 Hz may be considered more than adequate for capturing upper limb movements in a disabled population.

#### **2.11.5 Measuring the start and end points of angular finger movements.**

In addition to the more theoretical questions discussed above, the present research has also presented a number of methodological issues. One of these concerns the timing of the movements. As explained above, it was decided to allow the participants to move at their own pace, with the aim of capturing a picture of a more naturally timed movement. An alternative may have been to provide an external signal to move. However, as Rosenbaum, Meulenbroek, Vaughan, and Jansen (2001) have pointed out, normally occurring movements are not generally timed externally. An alternative may have been to link the hand to a switch which would flip once the hand moved. However, as will be seen below, this would have been impractical when looking at the individual joint movements in a hemiplegic hand. This is because different patients initiated the reach towards the CD case with different joints; sometimes the PIP moved first, sometimes the wrist, at other times the thumb, and so on. The flip of a single switch would not have captured this and would have given a misleading picture of the MT. Similarly, as this was not a simple timed movement towards a target, there was no single movement endpoint that could be measured and

compared for each participant. Furthermore, it was felt that to provide the participants with further constraints than those already given may have simply overloaded them while performing what was an already challenging task.

The present research therefore posed the problem of demarcating a start and end point for each movement when viewing the captured data. The determination of these kinematic landmarks has been identified as a perennial problem in human movement studies (Latash, 2008). This is because human movement is characterized by high levels of variability. Even actions such as transferring from sitting to standing are now known to be marked by relatively high levels of intra-individual variation over repeated measures in a healthy population (Chau, Young, & Redekop, 2005). Such variability is even more marked in movements of the upper limb (Bernstein, 1967). Authors have proposed a variety of solutions for the measurement problems human movement variability presents (Chau et al., 2005; Kerr, Durward, & Kerr, 2004; Latash, 2008). However, it should be emphasized as part of this discussion that this measurement problem becomes even more complex when analyzing upper limb movements in hemiplegia, characterized as they are by even higher levels of discontinuity than those found in a healthy population (Cirstea et al., 2003; Freitas & Scholz, 2009; Lin et al., 2007; Jeannerod, 1985; Kleissen, Buurke, Harlaar, & Zilvold, 1998; Thiess et al., 2009).

The present study posed three specific challenges. First, the captured movement was a multi-joint reaching and grasping action which combined elements of rotatory angular motion and translatory motion into an overall picture of general motion (Zatsiorsky, 1998). Second, despite meeting the inclusion criteria for the study, all patients inevitably presented with differing patterns of upper limb impairment. Third, the measurements had been made of the most distal joints in the human body, which would be likely to show higher levels of variability in their trajectories than more proximal joints. The very high levels of variability in these movements can be seen in figure 2:5 which shows ensemble-averaged joint path and angular velocity and acceleration curves for the PIP joint over the whole movement for both groups. It will be noted that the velocity and acceleration curves are marked by multiple peaks.

These challenges were immediately apparent upon visual inspection of the

joint marker images on the computer monitor. In some individuals, for example, the wrist moved first, which was indicated by a translational forward movement of the whole hand with no simultaneous rotatory movement at the joints of interest. In others the thumb and index opened together before the reach was initiated, while in others the thumb began the movement. The utilization of a simple start signal would not have solved this problem. In addition, the solution of Lin et al. (2007), in which the transport phase was measured from a single wrist marker, would not have captured the complexity of this multi-joint movement.

It was decided to identify the initiation point of the movement visually. Specifically, this was achieved by visually inspecting, frame by frame, the images of the markers during the entire capture period, and identifying the point at which the hand began the movement towards the CD case. Similarly, the point at which the hand returned to a resting position after leaving the object was also identified visually. As stated above, the variability in the movement meant that a single kinematic landmark which identified the movement onset and offset in each patient could not be determined. While the author is confident that these visually identified movement sequences did provide an accurate estimation of the movement duration, it does not obviate the need to identify a more reliable method of identifying these points. This is necessary if the question of possible subjective bias is to be clearly addressed and is discussed further in chapter three.

#### **2.11.6 Limitations of sampling process.**

The key problem with convenience sampling, as used in this study, is that the sample may not be representative of the whole population. The essence of random sampling is that each unit of the population should have an equal chance of inclusion in the sample (Boniface, 1995). However, in the convenience sample used here, the entire population was not sampled. This created an immediate bias towards individuals who were still in the early recovery stages following stroke, as they were still inpatients or visiting rehabilitation units following recent discharge from an acute or subacute setting. In addition, the individuals recruited from Hospital Universitario Major and Barrios Unidos hospitals would all have had some level of work-based health insurance or been paying towards a contributory scheme. This is not

representative of the whole Colombian population, many of whom are reliant purely upon the obligatory state health plan. Overall, these factors might limit the generalizability of the research findings to the wider population of adult stroke patients.

#### **2.11.7 Limitations of physical treatment protocol.**

It has been suggested that that some MI studies may have risked extraneous variables influencing their results as a result of a failure to define and hence control ongoing therapy interventions with sufficient clarity. The provision of a detailed treatment programme, which was experienced by all participants, was therefore an attempt to address this problem. In one sense, however, this contradicts the clinical recommendation that therapy should not be stereotyped and should be tailored to the needs of the individual patient (Raine et al., 2009). Therefore, the scientific demand for standardisation and control is slightly out of kilter with the clinical aim of a personalised treatment programme.

#### **2.11.8 Randomisation.**

A further methodological issue concerns the randomisation process. This was achieved by tossing a coin prior to allocation to group. The key problem here is that the researcher was both administering the treatment and conducting the data gathering and measurement. A way had to be found, therefore, to 'blind' the researcher as to group allocation, while he was providing the conventional physical treatment to each patient. Tossing a coin once all physical treatment had been completed provided a useful solution, as it meant that the researcher only knew which group the patient had been allocated to once all treatment sessions were finished. However, it might be countered that this does not provide the most reliable method of allocation, and may provide scope for subjective bias. Once again, the author is confident that bias did not enter into this process, however it is the case that a more reliable method of allocation should be found.

A further point in relation to randomisation concerns the breakdown of the blinding process for the final patient allocated. This breakdown occurred because it was clear by the end of the study that there was only one place left to be filled to make up the intended sample size of ten. The researcher therefore knew while administering

the physical treatment which group this final person would go into. In mitigation, it should be pointed out that the physical treatment given was based on exactly the same protocol as for the other participants, and which is outlined in appendix 2:6. Again, the author feels that this protocol was followed in exactly the same way for this last allocated patient.

In a study such as this, in which the researcher was administering the treatment, conducting the motion capture and analysing the data, it is difficult to see how true blinding can be achieved. Barclay-Goddard et al. (2011) in their Cochrane Collaboration Review explicitly highlight the difficulty of ensuring blinding in MI protocols based on a clinical population, and go far as to state that full blinding may be impossible in such a study. It is also important to bear in mind that one recent large scale and carefully controlled study of MI on people with stroke has also reported a breakdown in the blinding process, leading to only 86% of the patients being assessed by a blinded assessor (Ietswaart et al., 2011). The authors of this latter study dealt with the problem through an honest statement about the gap in the control of the study variables, and it was not felt to present a major challenge to the study's validity. While it is hoped that the same can be said of the present study, it remains the fact that this lack of blinding towards the end of the study presents a problem which must be addressed in future work.

#### **2.11.9 Experiment design.**

A further limitation of this study is the single factor independent groups design. This is not necessarily an inappropriate design for all behavioural research (Boniface, 1995). Moseley et al. (2002), for example, point out that it is acceptable for a study to have one intervention and one control, which in the case of the present research was KMI as the intervention and relaxation as the control. However, an essential precondition is that there should be homogeneity of variance among the participants. In other words, the researcher should be confident that like is being compared with like in the first place, in order to be able to state with some degree of certainty that any differences between the groups were a result of the experimental manipulation. During the course of this study it became clear that such an assumption could not be made. Again, referring to figure 2:5, it can be seen that there were very



high levels of variability in the movements captured. Indeed, to control for this, the independent samples *t* test used as an inferential statistical test had to be adjusted for some of the dependent variables measured. However, it is still imperative that future work should attempt to anticipate this problem at the stage of the research design. One way to address the issue would be by including an element of repeated measures in any design. This would mean that patients could act, at least in part, as their own control group, as pre- and post-test measurements could be compared for significant difference. These issues will be addressed further in chapter three.

## **2.12 Conclusion**

A number of kinematic measures were used as dependent variables in the study. It was found that the mean magnitude of the PIP joint for the KMI group was larger during the approach phase of the movement, and that this difference approached significance. However, the mean magnitude of the PIP was not significantly larger during the holding phase. It was felt therefore that the finding of larger mean PIP angle during approach was unlikely to provide evidence for a specific effect of KMI. This was because the lumbrical grip hand posture which was the target of KMI was actually meant to occur during the holding phase of the grasp. In addition, the correlation between MCP and PIP was not significant during the holding phase. This was taken as further evidence that the greater PIP magnitude during the approach phase probably not a product of training using KMI.

In addition, two variables had been proposed as representative of underlying motor planning processes. These were the time to peak velocity and the number of MUs during approach. No significant difference was found for these variables between the groups. It was therefore argued that KMI was unlikely to have influenced the performance of the movement at the planning stage. It was, however, noted that one of these variables, the number of MUs, may be problematic as a measure of motor planning processes in a hand movement such as that analysed in the present research.

Mean MT was significantly faster for the movement as a whole for the KMI group. This was taken as evidence that KMI may work as part of a general process of heightened attention directed towards the limb, rather than at the level of activation of specific muscles and joints. This finding was underlined by the fact that MT during the

approach period of the reach appeared to be unaffected by the use of KMI.

## Appendix 2:1

Original letter confirming ethical approval (see appendix 2:2 for English translation).



**COMITÉ DE ÉTICA EN INVESTIGACIÓN  
ESCUELA CIENCIAS DE LA SALUD  
UNIVERSIDAD DEL ROSARIO**

## MIEMBROS

**ALBERTO VELEZ VAN MEERBEKE**  
MAGISTER EN PSICOLOGÍA

**OSWALDO QUINDIAN ESPINOSA**  
LICENCIADO EN PSICOLOGÍA

**ÁNGELA DELFÍN PEREZ LOPEZ**  
LICENCIADA EN PSICOLOGÍA

**JAIRO RODRIGUEZ BILLESIMBERG**  
LICENCIADO EN PSICOLOGÍA

**CARLOS ENRIQUE TRILLES PEREZ**  
LICENCIADO

**CATALINA VIZCARRA BANCOS**  
LICENCIADA EN PSICOLOGÍA

**ISABEL PRIZZI DE LOS**  
LICENCIADA EN PSICOLOGÍA

**RICARDO ALVARADO SANCHEZ**  
LICENCIADO EN PSICOLOGÍA

**MARCELA RUIZ DURAN VARGAS**  
LICENCIADA EN PSICOLOGÍA

**FABIAN GONZALEZ ACOSTA**  
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**YOLLY SAMPERE KUTCHIBACCI**  
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**LUISA MARCELA TORRES PEREZ**  
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**VIVIANA PALACIO ESPINOSA**  
LICENCIADA

**DAVID PALAL**  
LICENCIADO EN PSICOLOGÍA

**PABLO ENRIQUE MORALES MARTIN**  
LICENCIADO EN PSICOLOGÍA

CEI-AMH002-000015

Bogotá, Febrero 03 de 2009

Doctor

**JONATHON PAUL O'BRIEN****Investigador Principal****Protocolo: ¿ES EL USO DE IMAGEN MOTORA EFECTIVA EN LA REHABILITACIÓN DE LA MANO HEMIPLÉJICA?.**

Ciudad

Respetado Doctor O'Brien:

Acusa recibo de las correcciones sugeridas por los miembros del Comité de Ética en Investigación en reunión del día miércoles 21 de enero de 2009, mediante actas No.133, luego de su revisión se aprueba el protocolo y el Informe de Consentimiento para el Paciente.

El comité de Investigación se rige por los lineamientos jurídicos y éticos del país a través de las resoluciones 008430 de 1993 y 002378 de 2008 del Ministerio de Protección Social. Igualmente se siguen las normas contempladas en la declaración de Helsinki (Seúl, Corea 2008) y de la Conferencia Mundial de Harmonización para las Buenas Prácticas Clínicas.

Cordialmente,

**ALBERTO VELEZ VAN MEERBEKE**

Presidente

c.c. Archivo

mez  
21-01-09  
27

**Appendix 2:2****English translation of approval letter from the University of Rosario ethical review committee.**

Ethics committee

School of Health Sciences

University of Rosario

**Members**

Alberto Veléz van Meerbeke,  
Neuropaediatrician, President.

Ovidio Oundjian Besnard,  
Lawyer –Member of the  
community.

Ángela-Maria Ruiz Sternberg,  
Gynecologist-Obstetrician,  
Epidemiologist.

Jaime-Enrique Ruiz Sternberg,  
Gynecologist-Obstetrician,  
Epidemiologist.

Carlos-Enrique Trillos Peña,  
Epidemiologist.

Catalina Latorre Santos,  
Epidemiologist, Master in health  
administration.

Isabel Pérez Olmos, Psychiatrist,  
Epidemiologist.

Ricardo Alvarado Sánchez,  
Master in public health,  
Secretary of committee.

Martha Rocio Torres Narvaez,  
Physiotherapist.

Patricia Granada Acosta,  
Paediatrician.

Belen Samper Kutschbach,  
Nutricionist, Epidemiologist,  
Bioethicist.

Luisa Marina Matheus, PhD,  
Biochemist and Molecular  
Biologist.

Ximena Palacios Espinosa,  
Psychologist.

David Palau, Obligatory medical  
service.

Pablo Emilio Moreno Matín,  
Social Worker and Master in  
family studies.

Bogotá, February 3<sup>rd</sup>, 2009

Doctor\* Jonathon Paul O'Brien

Principal Researcher

Protocol: Is the Use of Motor Imagery Effective in the  
treatment of the hemiplegic hand?

Dear Doctor\* O'Brien

Receipt is acknowledged of the corrections suggested by  
the Research Ethics Committee, which met on  
Wednesday 21 January 2009, in meeting minutes number  
133. Following its revision the protocol and the informed  
consent for the patient have been approved.

The committee governs according to the legal and ethical  
guidelines of the country by means of resolution 00843  
of 1993 and 002378 of the Ministry of Social Protection.  
At the same time they have followed the rules considered  
in the Declaration of Helsinki (Seoul, Korea, 2008) and  
the International harmonization conference for Good  
Clinical Practice.

Cordially,

Alberto Veléz van Meerbeke

\*Please see the note on the use of this term above.

## **Appendix 2:3**

### **English translation of patient information sheet and consent form.**

#### **Informed consent form for the patient**

**Title of the study: Is the use of motor imagery effective in the rehabilitation of the hemiplegic hand?**

**What is the purpose of the study?**

To analyze the effectiveness of a new technique in the rehabilitation of hand which is weakened following a stroke. The technique is similar to that used by sportspeople during the daily training and is called 'motor imagery'. It consists of imagining an action before performing it.

**How many people will take part?**

Six patients.

**Where will it take place?**

In the movement laboratories of the University of Rosario (Quinta de Mutis site) and Central University.

**Why this study?**

After a stroke, people normally experience problems moving one of their hands, and this can lead to disability and interfere with daily activities. Motor imagery is a new technique used in the rehabilitation of the hand and has shown to be successful in recent studies. The purpose of the study is to investigate the effectiveness of this technique in the rehabilitation of the hand after a stroke.

**What will my participation consist of?**

A therapist will guide you through a series of exercises for your hand and also carry out a test of vision and memory. Following this, you will be allotted to one of two groups. While one group will imagine movements, the other will follow a routine of mental relaxation. Another activity involved is the performance of some simple movements (grasping objects) with the hand affected by the stroke. These movements will be measured by cameras which can detect movement via adhesive sensors stuck to your hand.

**What are the procedures?**

Before taking part in the study you will be examined individually by an occupational therapist specialized in the rehabilitation of the upper extremities following stroke in order to determine if you can take part in the study. This will involve a test of the sensation in your hand and a short test of memory and vision. After this the therapist will ask you to perform a series of simple exercises, such as moving your fingers and knuckles, which will be monitored and assisted by the occupational therapist.

You will be seen individually. First you will have some plastic reflectors and sensors stuck with tape on your hand. These will allow the camera to record the movements and take measurements of your hand. At the same time a light nylon girdle will be used to support your trunk and to avoid excessive movement during the experiment. Also a light nylon band will be used to secure the wrist of the weak hand on the table, also to minimize excessive movements. None of these restrictions should cause any discomfort.

You will observe a video and listen to a recording which will guide you in the performance of the exercise. After this, you will be asked to grasp an object ten times with your weak hand and then move the thumb of that hand ten times. The camera will record these movements.

#### **What are my obligations?**

You will be asked not to discuss the details of the experiment with other participants during the study because having detailed knowledge of the procedures could influence the performance and affect the results. At the same time, you will be asked to inform the researchers immediately if you feel any pain, discomfort or inconvenience as a result of your participation in the study.

#### **What does the experiment consist of?**

You will be asked to watch a short video and then listen to a recording explaining the technique of motor imagery. After this you will be asked to perform a series of short movements with your weak hand

#### **What are the risks?**

This is a non-invasive study in which you will be asked to perform a simple everyday task (grasping an object). The risks are no greater than those met in everyday clinical practice for the rehabilitation of the hand weakened by a stroke. The principal researcher is an experienced occupational therapist, who is specialized in the treatment of the upper extremities, and who will monitor any discomfort reported by yourself when moving your hand. If the researcher considers that there is any risk which may cause you harm during the experiment, it will be stopped immediately. The study is

defined as “Research with minimal risk”, in accordance with Resolution 008430 of 1993.

The costs of transport to the movement laboratory will be covered and you will receive assistance from the main researcher and an assistant, both of whom are health professionals and specialists in rehabilitation.

**What are the benefits?**

We cannot promise any immediate benefits from taking part in the study. However, the results could help in the development of new methods in the treatment of patients with stroke.

**Does the experimental procedure have any risks?**

The experimental procedures (motor imagery practice) used in the study will not cost anything to you or your family and there are no known risks. However, the evidence suggests that these procedures can improve mobility.

**Is there any indemnity contemplated in relation to the risks?**

Not applicable

**Are there any agreements regarding insurance cover?**

Not applicable

**Am I obliged to participate? Can I leave at any point?**

Your participation in this study is entirely voluntary. You can leave the study at any point you wish without giving a reason. Your leaving the study will not have any impact on any other treatment which you are receiving.

**Will others have access to the results of the study?**

Although the ethics committee and the authorities charged with monitoring the study will have access to your clinical history during the study, your confidentiality will be guaranteed at all times. This is to ensure that the appropriate standards will be maintained by the researcher, as have been agreed by the ethics committee. You will be asked at the same time to sign an informed consent form to allow this.

**Will the documents which identify me be confidential?**

Your identity confidentiality will be secured throughout the study and the details related to your case will not be published. If the results of the study are published,



your confidentiality will be protected and your identity as a patient will not be revealed.

**What happens if I or my representative has relevant information which would affect me remaining in the study?**

If at any moment new information emerges which could affect your ability to take part in this study, you must inform us immediately.

**Under what circumstances will the study be stopped?**

Your participation in the study will be stopped if you no longer meet the requirements to participate or if any way the study has a negative effect on your well-being.

**How long will the study last?**

Your participation in the study will last a total of four days, three of these with the therapist in the hospital and one in which you will go to the movement analysis laboratory.

**Who is responsible for the study?**

If you require any further information regarding your consent and your rights please call doctors Jonathon O'Brien, telephone number: 2889947, Juan Alberto Castillo Martínez, telephone number 3474560, ext. 330-406, or Doctor Alberto Vélez Van Meerbeke, President of the Research Ethics Committee, University of Rosario, telephone number 3474570, ext. 395.

**Space for your name. Signature and number of your identity documents as a subject of the research.**

Name.....

Address.....

Telephone number.....

Identity card number.....

Signature.....

**Space for the name, signature, address and identity documents of two witnesses**

Name.....

Address.....

Telephone number.....

Identity card number.....

Signature.....

Name.....

Address.....

Telephone number.....

Identity card number.....

Signature.....

**Agreements between Bangor and Rosario Universities.**

**Ysgol Seicoleg  
Prifysgol Bangor**

Adeilad Brigantia, Ffordd Penrallt  
Bangor, Gwynedd LL57 2AS

Ffôn: (01248) 382211 - Ffacs: (01248) 382599  
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**DRAFT – SUBJECT TO CONTRACT**

**(1) BANGOR UNIVERSITY**

and

**(2) UNIVERSITY COLEGIO MAYOR DE NUESTRA SEÑORA DEL  
ROSARIO**

---

**AGREEMENT FOR THE  
PLACEMENT OF A PhD STUDENT  
TO CONDUCT A PROGRAMME OF STUDY  
AND RESEARCH AT ANOTHER INSTITUTION**

---

Relating to study undertaken by Jonathon Paul O'Brien 500092943  
at Bangor University

**Oliver Turnbull BSc PhD C. Psychol**  
Pennach yr Ysgol • Head of School

**DRAFT – SUBJECT TO CONTRACT**

**THIS AGREEMENT** is made on the 5th day of June 2008

**BETWEEN:-**

- (1) **BANGOR UNIVERSITY** College Road, Bangor, Gwynedd, LL57 2UX; and,
- (2) **UNIVERSITY Colegio Mayor de Nuestra Señora del Rosario**, Bogotá, D.C., Colombia, Carrera 6a No. 14-16.

**BACKGROUND**

The University's regulations for the Degree of Doctor of Philosophy (PhD) permit students who are pursuing full-time research to undertake part of their studies at another institution in the UK or overseas. Where such studies amount to more than a total of 6 months away from the University, the regulations require that the studies be conducted as defined by a formal agreement between the University and the collaborating institution.

**NOW IT IS HEREBY AGREED** as follows:-

**1. Interpretation**

1.1 In this Agreement, the following expressions shall have the following meanings unless the context otherwise requires:

<b>"Background Intellectual Property"</b>	any Intellectual Property Rights controlled or owned by any of the Parties prior to the Commencement Date or any Intellectual Property Rights generated by either of the Parties independently of this Agreement and controlled or owned by that Party or any Intellectual Property Rights to which the Party has the necessary rights for the purpose of this Agreement;
<b>"Commencement Date"</b>	1 <sup>st</sup> of July 2008
<b>"Confidential Information"</b>	Background Intellectual Property and other information in whatever form (being written, oral, visual, or electronic) relating to any of the Parties and its business, including, but not limited to material, whether technical, commercial, financial, or information relating to the Intellectual Property Rights or otherwise.
<b>"Fees"</b>	the sums due under this Agreement, further details of which are set out in clause 5.
<b>"FOIA"</b>	the Freedom of Information Act 2000 (as amended from time to time);

## DRAFT – SUBJECT TO CONTRACT

<b>“Information”</b>	as defined under section 84 of the FOIA;
<b>“Intellectual Property Rights”</b>	all intellectual and industrial property rights, including without limitation, patent(s), rights in know-how, trade marks, registered designs, models, unregistered design rights, unregistered trade marks and copyright (whether in drawings, plans, specifications, designs and computer software or otherwise), database rights, topography rights, any rights in any invention, discovery or process and applications for and rights to apply for any of the foregoing, in each case in the United Kingdom and all other countries in the world;
<b>“Local Supervisor(s)”</b>	staff of the Institution who is approved by the University, in accordance with its Regulations, as being qualified to supervise the Student during the Programme;
<b>“Parties” or individually a “Party”</b>	the Institution and/or the University;
<b>“Programme”</b>	the programme of study and research pursued by the student at the Institution, as approved by the University and subject to the conditions set out in Schedule 1, and forming part of the student’s work towards the award of a PhD at the University;
<b>“Programme IPR”</b>	the Intellectual Property Rights created specifically for the Programme, which will be required by any party who is to provide the Programme;
<b>“Registration”</b>	the registration of the Student at the University for a defined programme of study leading, subject to the Regulations, to the award of a PhD;
<b>“Regulations”</b>	the regulations of the University in force from time to time, including any codes of practice or policies;
<b>“School”</b>	the academic school at the University in which the Student is registered for a PhD;
<b>“Student”</b>	the student pursuing the Programme at the Institution and registered at the University for a PhD;
<b>“Term”</b>	the period starting on the Commencement Date and ending on September 31 <sup>st</sup> 2010 or such other period(s) as agreed in writing between the Parties from time to time;
<b>“Supervisor”</b>	the person appointed by the University, in

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## “Supervisory Committee”

accordance with its regulations, to supervise the work of the student;

a committee convened by the University in accordance with the Regulations to monitor the progress of the Student.

- 1.2 Headings are for convenience only and shall not affect the interpretation of this Agreement.
- 1.3 References to Clauses, sub-clauses and Schedules are to the clauses and sub-clauses of and schedules to this Agreement.
- 1.4 The Schedules form a part of this Agreement and any reference to this Agreement includes the Schedules.

**2. Duration**

- 2.1. This Agreement shall remain in full force and effect for the Term unless otherwise terminated earlier in accordance with the provisions of this Agreement.

**3. Obligations**

- 3.1. The objective of this Agreement is to ensure that the Programme is provided to the standards reasonably expected by the University and by the Student.
- 3.2. The Programme approved for the purposes of this Agreement shall be subject to the University’s quality assurance procedures and shall be governed by the Regulations (as amended from time to time).
- 3.3. Both Parties shall comply with the Data Protection Act 1998 (as amended from time to time in relation to any matters arising under this Agreement).
- 3.4. The University shall comply with its obligations as set out in Part A of Schedule 2.
- 3.5. The Institution shall comply with its obligations as set out in Part B of Schedule 2.

**4. Procedural Matters**Transfer of students

- 4.1. With the consent of the Student, and by written mutual agreement between the University and the Institution, the Student may be transferred to an alternative programme and/or alternative provider. Such a programme may be delivered by the University, the Institution and/or a third party.
- 4.2. A Student transferred to an alternative programme under the provisions of clause 4.1 shall waive any rights to pursue any part of the Programme under this Agreement.

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- 4.3. Upon transfer to an alternative programme, the Student will remain registered at the University until such time as the Student discontinues his/her Registration or the end of the Registration period, whichever is sooner.
- 4.4. Wherever possible, and subject to the requirements of this Agreement, both the University and the Institution shall accommodate any reasonable request from the Student to transfer to an alternative programme.
- 4.5. Wherever possible both the University and the Institution shall allow the Student to exit from the Programme and shall provide such advice as may be appropriate to assist the Student to decide whether to continue or discontinue his/her Registration.
- 4.6. Where the transfer to an alternative Programme impacts negatively on the Student's academic progress towards the completion of a PhD, the University shall endeavour to assist the Student by organising an alternative programme, equivalent research opportunities at the University, or by allowing the student to amend the topic/subject area for the PhD.

Appeals, complaints and academic misconduct

- 4.7. An appeal by the Student against the decision of the Examination Board shall be decided by the appeals procedure of the University.
- 4.8. The complaints procedure of the Institution shall apply to all complaints by the Student in relation to the Programme other than those relating to the University's services for which complaints should be made according to the University's Student Grievance Procedure.
- 4.9. If the complaint relates to the quality of the Programme and if having pursued the complaint within the Institution the Student feels that it has not been dealt with satisfactorily, the Student shall be entitled to pursue the complaint in accordance with the University's Student Grievance Procedure.
- 4.10. The Institution shall maintain a record of all complaints made by the Student relating to the Programme and of the outcome of each complaint.

Registration and Certificates

- 4.11. The Student is registered for a University award and may not, unless by prior agreement of the University, register concurrently for any other named awards.
- 4.12. Unless by prior agreement of the University, the Student may not submit the results of any of the work conducted during the Programme in order to gain credit as part of any other named award.
- 4.13. The Student will be awarded a PhD in accordance with the Regulations and in accordance with the University's practices for degree ceremonies and the issuing of certificates.
- 4.14. Unless by prior agreement of the University, the Student may not be awarded a qualification of the Institution, or any other institution, based simply on the fact that he/she has successfully completed a PhD under the Regulations.
- 4.15. With the agreement of the University, the Institution shall appoint a Local Supervisor(s) to supervise the work of the Student on the Programme. For the duration of the Agreement, the Local Supervisor is expected, unless defined

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otherwise by the agreement of the University, to provide support to the student as defined in the Regulations for the supervisor.

**5. Fees**

- 5.1. There are no fee implications for this particular programme of study.

**6. Intellectual Property Rights**

- 6.1. All Background Intellectual Property shall remain the property of the Party introducing the same.
- 6.2. Subject to clause 6.4, each Party shall own the Intellectual Property Rights generated by it under this Agreement and shall be responsible for securing ownership of such Intellectual Property Rights from its employees, students and other agents.
- 6.3. The prior written approval of the University must be sought and obtained in writing in each case before its corporate identity (name, logo, colours and typefaces) is used in any publicity material. The University reserves the right to withhold consent at its absolute discretion.

In the event of the transfer of the Programme to a third party, the Parties agree that such transfer shall include the assignment of the Programme IPR by the University and **UNIVERSITY COLEGIO MAYOR DE NUESTRA SEÑORA DEL ROSARIO**

**7. Confidentiality**

- 7.1. Both Parties shall during the continuance of this Agreement and for a period of three (3) years after its termination use all reasonable endeavours to ensure that any Confidential Information under this Agreement shall be treated with at least the same degree of care and discretion to avoid disclosure as the receiving Party uses with its own confidential information, which it does not wish to disclose and shall not disclose the Confidential Information to any third party without the other Party's prior consent.
- 7.2. The undertaking in Clause 7.1 above shall not apply to Confidential Information:-
- 7.2.1. which, at the time of disclosure, has already been published or is otherwise in the public domain other than through breach of the terms of this Agreement;
- 7.2.2. which, after disclosure to any Party, is subsequently published or comes into the public domain by means other than an action or omission on the part of any of such Party;
- 7.2.3. which a Party can demonstrate was known to him or subsequently independently developed by him and not acquired as a result of this Agreement;



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- 7.2.4. lawfully acquired from third parties who had a right to disclose it with no obligations of confidentiality to the other Party; or
- 7.2.5. is required to be disclosed by applicable law or court order or by any Party's regulatory body, which is empowered by Statute or Statutory Instrument, but only to the extent of such disclosure and the Receiving Party shall notify the Disclosing Party promptly of any such request.
- 7.3. Staff, students and any agents, consultants or sub-contractors engaged to work on the Programme(s) and any other disclosures strictly necessary for the purposes of carrying out the Programme(s), shall be subject to the principles of confidentiality no less than those as set out in this clause.

**8. Liability**

- 8.1. Except as set out in clause 8.2, no Party to this Agreement shall be liable to the other whatsoever (whether in contract, tort (including negligence), breach of statutory duty, restitution or otherwise) for any indirect or consequential loss (all of which terms include, without limitation, pure economic loss, loss of profits, loss of business, depletion of goodwill and like loss) howsoever caused arising out of or in connection with this Agreement.
- 8.2. Nothing in this Agreement limits or excludes any Party's liability for:-
- 8.2.1. death or personal injury; or,
- 8.2.2. any fraud or for any sort of liability that, by law, cannot be limited or excluded; or,
- 8.2.3. for any matter which it would be illegal for any Party to exclude or to attempt to exclude its liability.
- 8.3. Except as set out in clause 8.2, each Party's liability under this Agreement shall be limited to £200,000.

**9. Freedom of Information**

- 9.1. The Institution acknowledges that the University is subject to requirements under the FOIA and shall assist and cooperate with the University to enable it to comply with any information disclosure requirements.
- 9.2. Where the University receives a request to disclose any information that, under this Agreement, is the Institution's Confidential Information, it will notify the Institution and will consult with them. The Institution shall respond to the University within five (5) days after receiving the University's notice of the request. In the event that the Institution fails to respond within the requisite period, the University reserves the right to disclose any such information it deems appropriate.
- 9.3. The University shall be responsible for determining at its absolute discretion whether the Information is:-
- exempt from disclosure in accordance with the FOIA;
  - to be disclosed in response to a request for information under the FOIA and in no event shall the Institution respond directly to a

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request for information unless expressly authorised to do so by the University.

- 9.4. The Institution acknowledges that the University may be obliged under the FOIA to disclose information following consultation with the Institution and having taken its views into account.

**10. Termination**

- 10.1. Either Party may terminate this Agreement by notice in writing in the event that:-
- 10.1.1. the other Party is in material breach of this Agreement and has failed (where the breach is capable of remedy) to remedy the breach within twenty-eight (28) days of the receipt of a request in writing from the non-defaulting Party to remedy the breach;
  - 10.1.2. other Party fails to pay by the required date any sum due or payment has not been made within 28 days of written notice by the Party requesting it to do so;
  - 10.1.3. on the day after that on which there is no longer a Student pursuing the Programme.
  - 10.1.4. with immediate effect if the other Party shall become insolvent, enter into a composition with its creditors, be unable to pay its debts, have a trustee, receiver, administrative receiver, administrator or liquidator or similar officer appointed in respect of all or any part of its business or shall be wound up in each case under the laws of the jurisdiction applying to it;
  - 10.1.5. in other circumstances by the mutual agreement of both Parties.
- 10.2. Termination of this Agreement howsoever caused shall be without prejudice to the rights of the Parties accrued at the date of termination of the Agreement.

**11. Force Majeure**

- 11.1. Neither Party shall be liable to the other for any failure or delay or for the consequences of any failure or delay in performance of this Agreement if it is due to an act of force majeure.
- 11.2. An act of force majeure shall include any act preventing either Party from performing any or all of its obligations which arises from or is attributable to acts, events, omissions or accidents beyond the reasonable contemplation and control of the Party so prevented, act of God, war or national emergency, riot, civil commotion, terrorism, malicious damage, compliance with any law or governmental order, rule, regulation or direction, accident, fire, explosion, flood, storm or epidemic.

**12. Exclusivity**

- 12.1. The Institution may not franchise or assign the Programme to any institution, third party or other person in the United Kingdom or other parts of the world

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without express prior written agreement from the University, such consent to be at the University's absolute discretion.

**13. General**

- 13.1. This Agreement supersedes all prior agreements, arrangements and understandings between the Parties relating to the Programme(s) and constitutes the entire agreement between the Parties in relation to the Programme(s).
- 13.2. This Agreement does not create any rights enforceable by any person not party to it including, for the avoidance of doubt, any Student(s) and/or persons supervised in respect of the Programme(s) referred to in this Agreement.
- 13.3. In the performance of all services hereunder the Parties shall be deemed to be and shall be independent contractors. No employee, consultant or agent of either Party has any authority to bind the other Party.
- 13.4. The invalidity or unenforceability of any provision of this Agreement shall not prejudice or affect the validity or enforceability of any other provision of this Agreement.
- 13.5. This Agreement shall be construed in accordance with English and Welsh Law and the Parties agree to submit to the exclusive jurisdiction of the English and Welsh Courts.
- 13.6. Each of the Parties warrants it has the necessary power to enter into this Agreement.
- 13.7. Clauses 6, 7, 8 and 9 shall survive termination of this Agreement howsoever caused.
- 13.8. Any notice under this Agreement shall be in writing and be signed by or on behalf of the Party giving it. Any notice to be served on either of the Parties by the other shall be sent by prepaid first class post or by e-mail or by telex or by facsimile and shall be deemed to have been received by the addressee within 7 days of posting or 24 hours if sent by telex or facsimile to the correct telex number (with correct answerback) or correct facsimile number of the addressee.
- 13.9. Any agreement to change the terms of this Agreement in any way shall be valid only if the change is made in writing and approved by the mutual agreement of the authorised representatives of the Parties hereto.
- 13.10. Failure by any Party to exercise or enforce any rights conferred upon it by this Agreement shall not be deemed to be a waiver of any such rights or operate so as to prevent the exercise and enforcement at any subsequent time or times of that right or any other rights.
- 13.11. The Institution shall not be entitled to assign any or all of its obligations without the prior written consent of the University; such consent may be withheld at the University's absolute discretion.

**14. Dispute Resolution**

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- 14.1. Any question or difference which may arise concerning the construction, meaning or effect of this Agreement, or any matter arising out of or in connection with this Agreement shall in the first instance be referred to the Head of College of the University and **UNIVERSITY Colegio Mayor de Nuestra Señora del Rosario** for discussion and resolution as soon as reasonably possible and, in any event, within 21 days of such referral.
- 14.2. If the matter is not resolved at this meeting, the escalation will continue through to the Vice Chancellor of the University and the Rector of **UNIVERSITY Colegio Mayor de Nuestra Señora del Rosario** as soon as reasonably possible and, in any event, within a further 21 days.
- 14.3. If the matter remains unresolved to the satisfaction of either Party, the Parties will attempt to settle it by mediation in accordance with the CEDR Model Mediation Procedure. Unless otherwise agreed between the Parties, the mediator will be nominated by CEDR.
- 14.4. To initiate the mediation either Party must give notice in writing ('ADR notice') to the other Party to the dispute requesting mediation. A copy of the request should be sent to CEDR. The mediation will start not later than 20 days after the date of the ADR notice.
- 14.5. No Party may commence any court proceedings/arbitration in relation to any dispute arising out of this Agreement until it has attempted to settle the dispute by mediation and either the mediation has terminated or the other Party has failed to participate in the mediation, provided that the right to issue proceedings is not prejudiced by a delay.

**IN WITNESS** whereof this Agreement has been executed by the Parties as of the day and year first above written

## DRAFT – SUBJECT TO CONTRACT

**Schedule 2****Obligations****Part A – Obligations of the University**

The University will at all times during the continuance of this Agreement:-

- (i) Ensure that the Student pursuing the Programme during the Term is registered as a student of the University on a PhD.
- (ii) Ensure that the academic standards and the quality of the Programme are consistent with the standards expected to complete a PhD in the subject area as defined by University.
- (iii) Establish a Supervisory Committee for the Student in accordance with the Regulations and, where the Supervisory Committee deems that it is appropriate, to include the Local Supervisor as a permanent member of the Committee or to invite the Local Supervisor to selected meetings of the Committee.
- (iv) Ensure that a risk assessment has been completed for the Programme in accordance with the Regulations and any specific additional procedures and requirements in force within the School.
- (v) Keep a permanent record of the Student's results for the awards of the University.
- (vi) Be responsible for monitoring the Student's academic progress and responding to any concerns about such progress in accordance with the Regulations.
- (vii) Be responsible for pursuing, in accordance with its Regulations, any accusations against the Student regarding inappropriate conduct or unfair practice.
- (viii) Be solely responsible for the conferment of awards and the issuing of certificates.
- (ix) Provide any such information as might be reasonably requested by the Institution to permit the Institution to deliver the Programme.

**Part B – Obligations of the Institution**

The Institution will at all times during the continuance of this Agreement:-

- (i) Assign a Local Supervisor to co-ordinate the Programme and to supervise the Student.
- (ii) Ensure that the Local Supervisor liaises with the Supervisor as required to deliver the Programme.
- (iii) Ensure that the Local Supervisor, or another authorised member of the Institution's staff, attends meetings of the Supervisory Committee.

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- (iv) Through the Local Supervisor to observe all decisions and directions of the University with regard to the achievement and maintenance of academic quality in respect of the Programme.
- (v) Co-operate fully with the University in relation to any quality assurance processes, monitoring, assessments and reports undertaken by the University.
- (vi) Have administrative responsibility for the day to day operation of the Programme in accordance with the terms of this Agreement.
- (vii) Be responsible for the administration of the Student, subject to the University's procedures and regulations.
- (viii) Maintain the Student's records in relation to the Programme which shall, amongst other things, reflect the Student's results for the award of a PhD and provide the information as required (in both detail and format) by the University.
- (ix) Be responsible for the general health, safety and welfare of the Student on the Programme, and ensure adequate access to resources and services as defined in Schedule 1.
- (x) Report to the University any concerns about the Student's conduct, as might affect the Student's academic progress or that might lead to disciplinary action by the University against the Student.
- (xi) Provide any such information as might be reasonably requested by the University or by the Student to facilitate transfer to another programme.
- (xii) Subject to the Institution doing all such things as the University may reasonably require to enable it to ensure that the standards required by the University in relation to the Programme are met, the Institution shall be responsible for its own policies, planning and financial matters relating to programme provision not covered by this Agreement.

**Ysgol Seicoleg  
Prifysgol Bangor**

Adeilad Brigantia, Ffordd Penrallt  
Bangor, Gwynedd LL57 2AS

Ffôn: (01248) 382211 · Ffacs: (01248) 382599  
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**School of Psychology  
Bangor University**

Adeilad Brigantia, Penrallt Road  
Bangor, Gwynedd LL57 2AS

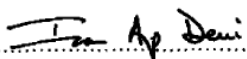
Tel: (01248) 382211 · Fax: (01248) 382599  
e-mail: psychology@bangor.ac.uk  
www.psychology.bangor.ac.uk

**DRAFT – SUBJECT TO CONTRACT**

**Authorised to sign for the University**

Name:- ...Dr Ioan Ap Dewi.....

Position:- .....Academic Registrar

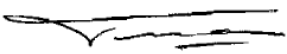
Signature:- 

Date:- .....19.08.08.....

**Authorised to sign for UNIVERSITY Colegio Mayor de Nuestra Señora del Rosario**

Name:- .....  
Hans-Peter Knudsen Quevedo .....

Position:- .....Rector.....

 Signature:- .....

Date:- .....

**Oliver Turnbull BSc PhD C. Psychol**  
Pennach yr Ysgol • Head of School

**MEMORANDUM OF AGREEMENT****BETWEEN****UNIVERSITY COLEGIO MAYOR DE NUESTRA SEÑORA DEL ROSARIO****AND****BANGOR UNIVERSITY**

The University of Rosario and Bangor University, wishing to establish cooperative relations, and especially to develop an academic and scientific interchange between the two institutions through mutual assistance in the areas of education, and research, agree as follows:

**PART I – SCOPE OF THE COOPERATION**


The areas of cooperation include, subject to mutual consent, any program offered at either institution as felt desirable and feasible on either side and that both sides feel contribute to the fostering and development of the cooperative relationship between the two universities.

The assistance to be provided by each of the contracting parties will be teaching, research, exchange of faculty and students, and staff development, etc., as deemed beneficial by the two institutions.

**PART II – GENERAL AREAS OF COOPERATION**

Assistance shall be carried out, subject to availability of funds and the approval of the University of Rosario and of Bangor University, through such activities or programs relating to, but not limited to:

1. Exchange of faculty members
2. Exchange of students
3. Joint research activities
4. Participation in seminars and academic meetings
5. Exchange of scientific materials and other information
6. Special short-term academic programs

 The terms of such mutual assistance and necessary budget for each program and activity that is implemented under the terms of this agreement shall be mutually discussed and agreed upon in writing by both parties prior to the initiation of the particular program of activity, and such programs and activity shall be negotiated on an annual basis. Each



institution shall designate a Liaison Officer to develop and coordinate specific activities or programs.

### **PART III- RENEWAL, TERMINATION AND AMENDMENT**

This agreement shall remain in force for a period of five (5) years from the date of the last signature, with the understanding that it may be terminated by either party giving notice to the other party in writing no later than the end of March in any year. The agreement may be extended by mutual consent of the two parties.

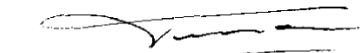
This agreement may be amended by an exchange of letters between the two institutions. Such amendments, once approved by both institutions, will become part of this Memorandum of Agreement.


The present agreement is signed in two (2) copies, each of them in English, all texts being equally valid.

IN WITNESS WHEREOF, the parties hereto have offered their signatures: **09 JUN. 2008**

For UNIVERSITY OF ROSARIO

For. BANGOR UNIVERSITY

  
Hans-Peter Knudsen Quevedo  
Rector

  
Dr I Ap Dewi  
Academic Registrar

**Appendix 2:5****Matlab syntax for time normalization and splines.**

1.  $X = [\text{'time from start to end of grasp'}]$
2.  $Y = [\text{'PIP position measurements over X'}]$
3.  $CS = \text{spline}(X, Y)$
4.  $XX = \text{linspace}[\text{'start time for grasp movement'}, \text{'end time for grasp movement'}, \text{'101'}]$
5.  $YY = \text{ppval}(CS, XX)$
6.  $YY = YY'$

**Appendix 2:6****Protocol for hemiplegic hand physical treatment.**

1. Sensory stimulation of the sides of the hand using the edge of a chop stick, starting with medial border (abductor digiti minimi), and including sides of fingers, finger tips and webspaces, and working back round to abductor digiti minimi. Repeated twice
2. Facilitation of thumb into opposition, repeated five times.
3. Facilitation of hand lumbrical action against resistance, repeated ten times.
4. Facilitation of lumbrical action in individual fingers, repeated ten times for each finger.
5. Facilitation of abduction in little finger, repeated ten times. The person was encouraged to actively move the finger during facilitation.
6. Facilitation of abduction in index finger, repeated ten times. The person was encouraged to actively move the finger during facilitation.
7. Facilitated placing of the hand on a table, with medial border making contact first each time. Repeated five times.

**Appendix 2:7****Script used for guided KMI training.*****Guided breathing script (repeated six times).***

“Focus on your breathing, breathe in and out normally, forget about any other sounds in the room or outside the room, just focus on your breathing. Put any worrying thoughts out of your mind. Close your eyes if you wish, but don’t let yourself fall asleep”

***KMI script (repeated six times).***

“Now relax your muscles. Relax the side of your body which has been affected by the stroke. Relax the hand which has been affected by the stroke. Now remember the video of the hand grip which you saw earlier. I want you to imagine your weak hand making that movement. Keep your hand quite still. Imagine the feeling of the knuckles bending and the fingers straightening at the same time, the knuckles bending upwards and the fingers staying quite straight. Keep your hand very still, just imagine the feeling of the movement.”

***Reorientation script.***

“Now become aware of the room again, if you have had your eyes closed open them and look around you. The session has now ended.”

**Script for guided imagery focusing on general sensory awareness.*****Guided breathing script (repeated three times).***

“Focus on your breathing, breathe in and out normally, forget about any other sounds in the room or outside the room, just focus on your breathing. Put any worrying thoughts out of your mind. Close your eyes if you wish, but don’t let yourself fall asleep.”

***Muscular relaxation script (repeated five times).***

“Now focus on the sensation in your legs and feet and then let your legs and feet relax. Next focus on the sensation in your hands and arms, and let your hands and arms relax. Now you are quite relaxed, focus on the sounds in the room around you. Now concentrate on your breathing for a while longer, just breathe normally.”

***Reorientation script.***

“Now become aware of the room again, if you have had your eyes closed open them and look around you. The session has now ended.”

## Chapter Three

### **Does a One-off Training Session Using First Person Kinaesthetic Motor Imagery or Third Person Visual Motor Imagery Make a Difference to the Kinematics of the Lumbrical Grasp in the Hemiplegic Hand of Stroke Patients?**

#### **3.1 Experiment Two Research Question**

Do kinaesthetic and visual motor imagery have different effects on the kinematics of lumbrical grip formation in the hand affected by hemiplegia?

##### **3.1.1 Experiment Two Hypotheses**

1. The use of MI will be linked to improved coordination between the MCP, PIP and DIP joints when grasping an object using the lumbrical grip.
2. The group using KMI will show a marked improvement in the coordination of these joints.
3. The use of MI will be associated with greater extension in the PIP and DIP joints and greater flexion in the MCP joints when grasping an object using the lumbrical grip.
4. The effects predicted in hypothesis three will be more marked in the group using KMI.
5. Overall MT will be faster in the groups using MI.
6. Overall MT will be fastest in the group using KMI.
7. The angular paths of the MCP, PIP and DIP joints will be smoother in the group using MI.
8. The angular paths will be smoothest in the group using KMI.

#### **3.2 Literature Review**

##### **3.2.1 Aim and objectives of literature search for chapter three.**

The aim of the literature search for chapter three was to present evidence that authors have proposed two distinct categories of motor imagery which are based upon sensory modality. The first objective was to identify evidence showing that two distinct modalities of imagery can be clearly demarcated. The second objective was to find if work had been done to compare the effects of these two imagery types on the

rehabilitation of the hemiplegic hand.

### **3.2.2 Inclusion and exclusion criteria.**

As for chapter one, this was a scoping study and the criteria were therefore quite broad. Studies were sought which compared the effects of kinaesthetic and visual motor imagery. There were no inclusion criteria based upon methodology and a wide variety of studies was referenced. These included studies of mental rotation, electroencephalography, neural imaging and human behaviour. Furthermore, there were no inclusion criteria based upon body part, so that studies focusing on gait and whole body movements were included. In addition, it became clear that very little work had been done in this area involving persons with stroke, as can be seen in table five. The inclusion criteria were therefore widened to include studies involving neurologically intact individuals.

### **3.2.3 Databases and internet sites used.**

The same databases were used as described above for chapter two. The time period of the searches was also the same.

### **3.2.4 Search strategies, search terms and number of hits.**

The search strategy used for PSCYinfo is shown in table 3:1. This is a re-run of a search originally carried out in April 2010 and repeated in October 2011 and January 2012. Here, the free text terms included: kinaesthetic; visual; stroke; upper limb; motor imag\*. MeSH terms included: cerebrovascular accidents; arm (anatomy); hand (anatomy). This search was adapted for MEDLINE and CINAHL. For Web of Science, search terms included: kin\$esthetic; visual; imag\*; upper limb; neurorehabilitation; stroke.

Table 3:1

*Search strategy for PSCYinfo for chapter three (1806 – January 2012)*

Search number	Search terms	Hits
1	CEREBROVASCULAR ACCIDENTS/	6601
2	Stroke.ti,ab	8463
3	"ARM (ANATOMY)"/ OR "HAND (ANATOMY)"/	2131
4	("kinesthetic" OR "kinaesthetic").ti,ab	683
5	visual.ti,ab	47155
6	(motor AND image*).ti,ab	1093
7	1 OR 2	9494
8	upper AND limb	844
9	3 OR 8	2833
10	7 AND 9	305
11	4 AND 5 AND 6	35
12	10 AND 11	1
13	7 AND 11	1

/ indicates MeSH term

### **3.2.5 Review of reference lists.**

As for the previous searches described above, reference lists of included articles were surveyed and any relevant citations were obtained.

### **3.2.6 Search limits.**

Search limits were the same as those outlined for chapter two above.

### **3.2.7 Critical appraisal tool.**

Only one RCT involving individuals with stroke that compared visual and kinaesthetic motor imagery was identified. The PEDro score for this study was six, indicating moderate quality (Moseley et al., 2002). The PEDro criteria are shown in table six below.



Table 3:2

*PEDro criteria for relevant RCT identified for chapter three*

Study	PEDro criteria										
	1	2	3	4	5	6	7	8	9	10	11
Kim et al. (2012)	✓	✓	✓	✓	×	×	×	×	×	✓	✓

✓Meets the criteria    ×Does not meet criteria

*Note.* Key to PEDro criteria: 1 = inclusion criteria stated; 2 = random allocation to group; 3 = concealed allocation to group; 4 = groups similar at baseline; 5 = blinding of subjects; 6 = blinding of administering therapists; 7 = blinding of outcome assessors; 8 = > 85% subjects assessed on key outcome measure; 9 = all subjects completed treatment, or intention to treat analysis conducted; 10 = between group statistical analysis reported; 11 = point measures and variability measures provided for key outcome.

### 3.3 Introduction

#### 3.3.1 Definitions of kinaesthetic and visual MI.

As has already been shown, authors argue that mental imagery is a cognitive process embedded in working memory structures (Dickstein & Deutsch, 2007; Kosslyn, Ganis, & Thompson, 2010; Malouin et al., 2004). Working memory underpins the manipulation and use of information during ongoing activity and may be based on visual-spatial, verbal or kinaesthetic modalities (Dickstein & Deutsch, 2007; Malouin et al., 2004). It has been maintained, therefore, that a mental image may also be constructed out of inputs from discrete sensory modalities (Kosslyn, Ganis, & Thompson, 2010). It was also shown in chapter one that MI has been described as a special case of mental imagery involving imaging of human movement (Jackson et al., 2001). Like mental imagery generally, MI can also engage different sensory modalities. Research on the role which MI might play in neurorehabilitation has focused on MI based on the modalities of vision (VMI) and kinaesthesia (KMI). The latter term was coined by Bastian (1887), referring to the sensations of movement produced by the skin, muscles, tendons and joints; later authors have updated the definition to include the sensory input from the muscle spindles, Golgi tendon organs

and the vestibular system (Schmidt & Lee, 2005).

### **3.3.2 Source of sensory information and perspective of the imager.**

Two closely linked issues are the questions of the source of the sensory information and the perspective taken by the imager. Regarding the first of these, Bastian (1887) commented that, while, the origin of visual information would be extrinsic to the organism, the source of kinaesthetic sensation could be either extrinsic or intrinsic. Contemporary writers have accordingly regarded VMI as ‘external’ imagery (Dickstein, Gazit-Grunwald, Dunskey, & Marcovitz, 2005; Malouin et al., 2004). However, some modern researchers have also suggested that ‘internal’ imagery might be based on vision as well as kinaesthesia (Dickstein & Deutsch, 2007; White & Hardy, 1995; Hardy & Callow, 1999). Other writers seem to regard kinaesthesia in a more restrictive way than Bastian (1887), arguing that MI based on this modality is a purely ‘internal’ form of imagery (Dickstein et al., 2005; Malouin et al., 2004). Callow and Hardy (2004) echo Bastian’s formulation, arguing that kinaesthesia can be employed in both internal and external imagery. Fourkas, Avenanti, Urgesi, & Aglioti, (2006) agree with this, with the proviso that the imagined action should be performed by the person themselves. There is, therefore, a lack of consensus about how to categorize KMI and VMI in relation to the origin of the imagined movement.

The question of the perspective taken by the imager is intertwined with that of the origin of the imagined sensation. As shown in chapter two, Mulder (2007 p. 1268) stated that it is possible to characterize KMI as internal imagery and also as “first person” imagery, in which one imagines the sensory experience of a movement produced by oneself. Alternatively, VMI may be thought of as a “third person” form of imagery in which the person visualizes themselves performing the movement “as from a distance” (Mulder, 2007, p.1268). Dickstein and Deutsch (2007) and Malouin et al. (2004) concur with this view, and regard KMI as involving a first person and VMI implying a third person perspectives. Voisin, Mercier, Jackson, Richards, & Malouin (2011), however, state that KMI may be first or third person, while VMI can only be third person. Dickstein and Deutsch (2007), along with Fourkas et al. (2006), do, however, extend the definition of third person imagery, stating that it may involve imagining another person moving through the external environment. For the purpose

of this introduction, KMI is regarded as first person/internal imagery and VMI as third person/external imagery.

### **3.3.3 The use of KMI and VMI in rehabilitation.**

As a consequence of the view that MI can be based on kinaesthesia or vision, controversy also exists among researchers as to whether VMI or KMI would be more effective in the promotion of motor recovery following stroke. Voisin et al. (2011), for example, maintain that KMI is more attuned to the activation of motor related brain mechanisms. One might conclude on this basis, therefore, that KMI would be more suited to motor rehabilitation. Indeed, authors such as Mulder (2007) and Jackson et al. (2001) have argued that KMI is likely to have a greater impact on the recovery process. In addition, Schuster et al. (2011) found that more successful motor imagery trials tended to employ KMI. Dickstein & Deutsch (2007), however, have demurred, arguing that the demarcation of MI by sensory modality is a misleading scholastic device. They maintain, rather, that imagery should be tailored to the needs of the individual and also take into account the training task and environment; this would be preferable, they suggest, to designing treatment protocols based on a presumption of the superiority of either VMI or KMI.

Some authors have explicitly called for more work to be done in this area, (Nilsen et al., 2010), and it could be argued that insufficient guidance as to the relative efficacy of KMI or VMI can be gleaned from reviews of the MI literature. One early and widely cited meta-analysis of research on the effects of mental training on human motor performance, for example, did not highlight any distinction between the two (Driskell, Copper, & Moran, 1994). More recently, reviews by Braun et al. (2006), Sharma et al. (2006) and Zimmerman-Schlatter et al. (2008), specifically related to MI in rehabilitation following stroke, did not directly address the issue. If KMI and VMI are indeed distinct entities which can be applied in different ways in stroke rehabilitation, then a clearer understanding of the mechanisms which underpin them and an assessment of their relative utility may be a priority for research.

The following review of the literature focuses on two issues concerning KMI and VMI. The first of these is the extent to which it is possible to distinguish empirically two distinct categories of imagery based on their sensory modality. Here,

evidence from neural imaging, electroencephalography, TMS and behavioural studies is critically assessed. The second focus, which follows on from this, is a discussion of one attempt which has been made to apply KMI and VMI in stroke rehabilitation, and how far these different MI modalities can be said to have differential effects on the recovery process.

### **3.3.4 The cerebral localization of VMI and KMI.**

Kosslyn et al. (2006) have argued that speculation about cognitive processes, such as the modality specific properties of mental imagery, should ultimately be constrained by empirical evidence regarding the brain mechanisms which might underpin them. As part of their own case that the mind employs visual images which actually picture some basic features of the outside world, these authors have accordingly highlighted the retinotopic organization of Brodmann Area 17 (BA 17), where locations in the visual world are directly mapped onto the cortex. In one study, for example, Kosslyn et al. (1999) had asked 8 participants to mentally visualize very simple images of one of four sets of black rectangles against a white background and were able to show evidence of activation in BA 17. The authors then attempted to falsify this finding by using TMS to temporarily disrupt BA 17 during perception and imaging of the same stimulus and also introduced a sham control condition in which TMS deliberately missed BA 17. They immediately asked the participants to make decisions about the stimuli based on their mental images of them. They found that the response times were significantly slower when BA 17 had been disrupted, compared with the sham condition (Kosslyn et al, 1999). Based on this evidence, it would seem that the mind relies, at least in part, on visual images which can be inspected thanks to being projected onto BA 17.

Further evidence for the retinotopic activation of BA 17 during visual imagery was provided in a study by Klein et al. (2004). Here, six healthy individuals watched a bow-tie shaped stimulus at either horizontal or vertical orientations. They were then required to mentally image the stimulus at these different rotations. BA17 was imaged using fMRI throughout viewing and imaging. The results for the viewing condition showed clear patterns of horizontal and vertical activation that correlated with the shifting rotation of the shape. In the imaging condition, significant levels of activation

in BA 17 were also noted in five out of six participants, although this activation did not follow a retinotopic pattern when compared with a baseline reading (Klein et al., 2004). When the vertical and horizontal imagery conditions were directly compared, however, the activation pattern was found to resemble the retinotopic pattern noted for the watching condition in four of the volunteers.

While the previous two studies found evidence of a localizable neural basis for visual imaging, Sirigu and Duhamel (2001) tested the hypothesis that visual imagery and the imagery of movement could be dissociated cortically on two patients. One had a lesion in the left parietal lobe, affecting the supramarginal and angular gyri, which evidence suggests are involved in movement planning and the representation of action (Jeannerod, 2006; Wurtz & Kandel, 2000). The other patient had bilateral lesions in the inferior temporal lobes, which form a part of the ventral visual pathway and is known to play a role in the identification of complex visually presented stimuli (Kandel & Wurtz, 2000).

Both patients in Sirigu and Duhamel's (2001) study were asked to perform two motor tasks. In the first, they mentally estimated the time needed to point to targets of different widths, with the distance to be moved remaining constant. It was found that the inferior temporal lobe patient performed this task normally, with a strong correlation between real and imagined movement time towards the target. The left parietal patient's mental estimates and real movement times, however, correlated poorly. Both patients' estimates were then fitted to a curve based on a function derived from Fitts's equation (Fitts, 1954; Fitts & Peterson, 1964). It was found that the inferior temporal patient's estimates were predicted by the equation, as would be found in normal movement, but that this was not true for the left parietal patient. In the second motor task the patients were required to estimate the maximum speed at which they could perform a sequence of finger movements. The left parietal patient underestimated the speed at which he could perform the task. The inferior temporal patient scored normally.

In a series of tests of visual imagery ability Sirigu and Duhamel (2001) asked the patients to describe visual properties of objects and animals. The parietal patient scored normally on this task, while the inferior temporal patient performed very

poorly, with results more than three standard deviations below normal. The inferior temporal patient was then required to match an abstract shape to the identical shape, which was rotated and placed among an array of other forms. The results for this test were similarly random. It should be pointed out that this patient was able to describe the semantic properties of shapes normally. For example, when presented with pictures of animals he was able to describe their normal habitat. The results indicated, according to Sirigu and Duhamel (2001), that the individual was unable to mentally retrieve information from a visual store. In addition, using a metaphor developed by Kosslyn et al. (2006), and also used within working memory theory (Baddeley, 2003), they suggested that the patient was incapable of working with information stored in a temporary “visual buffer” (Sirigu & Duhamel, 2001, p. 914).

Klein et al. (2004) and Kosslyn et al. (1999) do seem to show evidence both for the existence of visual mental imagery and also for its reliance, at least in part, on some level of retinotopic activity. Sirigu and Duhamel (2001), meanwhile, provide results indicating that patients with brain lesions in regions which underpin object recognition and motor planning have difficulty imaging tasks which would engage those faculties. This could suggest, therefore, that imaging of movement may also involve some level of activation of cerebral regions which are adapted to the control of executed movements, providing evidence for the existence of distinct modalities of mental imagery.

It might be argued, however, that the findings of these studies have limited generalizability. Sirigu and Duhamel (2001)’s study was based on only two brain-lesioned participants, while Klein et al.’s (2004) study was only of six individuals. Klein et al. (2004) point out, in addition, that not all of their participants showed the retinotopic pattern during imagery. These authors also point to a degree of inter-individual variability in the group that did display this pattern. The horizontal activation during imagery, for example, was significant in the right hemisphere for four individuals and in the left hemisphere for three, while the activation level in the horizontal meridian of BA 17 was actually stronger in one person when imaging the vertically presented stimulus. It may be problematic, therefore, to infer that the retinotopic patterns noted in BA 17 during imagery in these studies have a universal

cognitive function. Further, both the Kosslyn et al. (1999) and Klein et al. (2004) studies used simple monochrome stimuli, which were actually static in the case of Kosslyn et al. (1999). It was also the case that both studies were of healthy individuals. Overall, therefore, it may be difficult to draw conclusions about the presence and function of what would presumably be far more complex visual and kinaesthetic representations of motor activity in the brain from these pieces of research. In addition, two of the studies may tell us little about how such representation would work in a brain compromised by stroke.

### **3.3.5 fMRI studies of VMI and KMI.**

More recently, Guillot, Collet, Nguyen, and Malouin (2009) have tried to answer some of these outstanding questions by using fMRI to look at the peculiar neural consequences of imaging with KMI or VMI. Thirteen competent imagers were selected on the basis of an imagery questionnaire and also by their performance on an autonomic nervous system test of the timing of an imagined movement. The individuals were trained on a finger sequencing activity and then asked to perform VMI, KMI or physical execution of this task while their brain was scanned. A control condition was also used in which the subjects listened to two alternating tones. The investigators stipulated that VMI should be of the individual themselves performing the task; KMI was defined purely in relation to its sensory content, without reference to first or third person perspective.

When Guillot et al. (2009) compared KMI and VMI directly they found a distinct belt of activity for VMI in BA 17, 18 and 19, in keeping with earlier findings reviewed by Kosslyn et al. (2006). Exclusive activity was also noted for KMI in the putamen and caudate nucleus bilaterally and in lobules VI, VIIb and VII and Crus I of the cerebellum. The overall frontal activation for KMI was also stronger than for VMI, with marked activity in BA 44 and the anterior and posterior SMA. The location of VMI-linked activity in the frontal region tended to be more posterior and superior. In the posterior cerebrum, VMI was correlated with higher activity in the superior parietal lobe, extending to the precuneus. KMI, however, was associated with an increased signal in the lateral and anterior areas of the inferior parietal lobule; VMI was linked to more posterior activity in this structure.

Guillot et al. (2009, p. 2169) concluded that VMI could indeed be shown to activate primary and secondary visual perception areas and, in an echo of Jeannerod's (2006) work, they also suggested that KMI must involve a process of "motor simulation" which is correlated with activity in cortical and subcortical motor regions. It is certainly the case that Guillot et al. (2009) were able to show some clearly demarcated regions activated exclusively by VMI or KMI. In particular, the areas activated by VMI in the occipital cortex might be associated with the neural substrata concerned with vision. However, it could be argued that the overall patterns of activation were less distinct. Both imagery modalities, for example, were correlated with raised signalling in the lateral pre-motor cortex. In addition, although a direct comparison of KMI and VMI showed that certain motor regions were more active in KMI, this does not indicate an absence of activity in these regions during VMI. When compared with the control condition, for instance, VMI was linked to significantly higher activation in the right primary motor area as well as the posterior and anterior SMA. In addition, in the same comparison, VMI was correlated with activity in the anterior and posterior putamen and globus pallidus bilaterally and in cerebellar regions bilaterally, which are all regions involved with motor activity (Guillot et al., 2009). The authors also maintained that the inferior and superior aspects of the parietal lobules were only active during KMI. However, their results do seem to show that VMI was linked to inferior parietal lobule activation when compared with KMI, albeit in a more posterior location, and that VMI was linked to bilateral activity in the inferior parietal lobule (BA 40) when compared with the control (Guillot et al., 2009). The authors also state that KMI, when compared with VMI, led to exclusive activation in ventral pre-motor BA 44. However, when VMI was compared with the physical execution condition it was linked to activity in BA 45, which has classically been grouped with BA 44 as Broca's speech area (Dronkers, Pinker, & Damasio, 2000). This evidence suggests, therefore, that, while it may be possible to link VMI exclusively to activity in primary and secondary visual regions, the case that KMI activates motor regions to the exclusion of VMI may be harder to maintain.

A further reservation about the conclusions drawn by Guillot et al. (2009) concerns the theoretical background of the work. The authors initially made what they



describe as a “theory-driven” hypothesis that KMI resembled executed movement more closely than VMI (Guillot et al., 2009, p. 2169). On this basis they predicted that KMI would be more likely to activate motor areas. However, three of the four sources which they reference here would not necessarily support such a hypothesis. The papers by Hashimoto and Rothwell (1999) and Decety et al. (1991) cited by the authors, for example, did not control for the sensory modality of the imagery tasks used: the participants were simply asked to imagine the movement. In addition, the earlier paper by Guillot et al. (2007) which they refer to, arguably did not clearly distinguish KMI and VMI, as the participants were instructed to use a combination of both approaches simultaneously. This may show, therefore, the challenge posed when attempting to marshal compelling and consistent evidence in making the case that KMI and VMI are indeed distinct entities with dissociable effects.

### **3.3.6 KMI, VMI and the electroencephalogram.**

Another way to explore the contrasting cerebral effects of KMI and VMI is the recording of neural electrical activity via the electroencephalogram (EEG) signal. Neuper, Scherer, Reiner, and Pfurtscheller (2005), for example, explain that mental imagery of a hand movement has been found to desynchronize the central beta rhythm, which occurs at between 12-28 Hz, and the mu rhythm, which occurs at 8-12 Hz. According to the authors, these patterns would also be found in real movements as well as at the planning stage of movement control (Neuper et al., 2005). Here, the authors implicitly equate mental imagery and pre-movement planning mechanisms, in line with the views of Jeannerod (2006). This congruency of mental imagery and the EEG signal means that imagery can be utilized in brain-computer interfaces (BCI), as the EEG signal provides a medium through which the brain can control an exterior apparatus. One outstanding issue, however, is the high level of variability in some individuals’ EEG signal during mental imagery practice. Neuper et al. (2005) hypothesized that this might partially be blamed on experimenters failing to control for the sensory modality of imagery. They therefore sought to compare the differing EEG responses linked to clearly defined KMI and VMI tasks, with the aim of developing a MI protocol for BCI research.

The authors instructed 14 healthy individuals to watch a video of an animated

hand opening and closing and then squeeze a ball in their hand. The participants then performed KMI or VMI of the action: KMI focused on the feeling of the movement; VMI on forming a “mental video” of the animated hand (Neuper et al., 2005, p. 669). EEG readings were taken from 33 scalp sensors during all the conditions. The authors were concerned to discover how far the variables of sensor location and frequency band of the EEG readings could be used as predictors of the sensory modality of the MI task being performed.

Neuper et al. (2005) found that the readings taken from the left sensorimotor area, the contralateral area for all the tasks, produced a 67% accuracy rate for KMI and 56% accuracy for VMI and that this difference was found to be significant. They also demonstrated that KMI was linked to highly localizable neural activity which affected the EEG signal in a predictable way. However, the mean classification accuracy for VMI found for the occipital sensors was only 58%, just above the classification accuracy for these sensors for KMI. It is interesting to note that, notwithstanding the different methods used, this stands in marked contrast to the findings of Guillot et al. (2009), discussed above, who found a very strong correlation between VMI and augmented signaling in the occipital region. In addition, Neuper et al. (2005) point out that it was difficult to control for the participants' compliance with instructions. It cannot be absolutely certain therefore if all the individuals were using VMI or KMI as specified by the experimenters.

A further limiting factor in Neuper et al.'s (2005) study, not highlighted by the authors, concerns the second of their research questions. This focused on the most relevant frequency bands when identifying KMI or VMI. The results here were much closer than for sensor location: for both KMI and VMI the most relevant frequencies were around 10-12 Hz, with similar relevance scores at this level for the two modalities (Neuper et al., 2005). When frequency patterns of the signal were used, therefore, to try to identify the modality of imagery, the effects of KMI and VMI were harder to distinguish. Once again, it would seem that the results of an attempt to clearly link KMI and VMI to specific activation patterns are open to conflicting interpretations.

### 3.3.7 The effects of KMI and VMI on cortical excitability.

Stinear et al. (2006) used TMS to test the varying effects of KMI and VMI on corticospinal excitability. They hypothesized that only KMI of thumb abduction would facilitate raised excitability in the primary motor region (M1) representing abductor pollicis brevis (APB). The authors adopted a definition of MI in line with that of Jeannerod (2006), as a cognitive phenomenon which precludes motor neuronal outflow at the spinal level. On the basis of this definition they predicted that the F wave response for APB should remain unaffected by either modality. The F wave is an EMG signal which occurs following excitation of a motor nerve. It involves an antidromic action potential being sent back to the muscle from the spinal cord and can be taken as one measure of the excitability of the cord (Kamen & Gabriel, 2010).

Stinear et al. (2006) conducted two experiments. In the first, TMS was applied to the contralateral primary motor cortex (M1) of ten healthy participants. This was intended to produce MEPs in APB and a control muscle, abductor digiti minimi (ADM). The MEP is the muscular electrical activity produced following electrical or magnetic stimulation of the cortex (Kamen & Gabriel, 2010). In their second experiment another ten healthy individuals had the median nerve, which supplies APB, electrically stimulated with the aim of producing an F wave response in the muscle. In both experiments the participants were asked to visualize their thumb moving (VMI) or to imagine the sensation of their thumb moving (KMI). A resting condition and a control condition that involved imagining a neutral static visual image were also added. All tasks were timed by a metronome. In the first experiment four magnetic stimuli were delivered to M1 during each condition; two coincided with task performance as timed by the metronome, the other two were applied after task performance (Stinear et al., 2006). In the second experiment 24 bouts of electrical stimulation were delivered during each of the four conditions.

The investigators found a significantly higher MEP amplitude for APB during KMI when the task performance period was compared with the post performance period. The same comparison for VMI, however, was not significant. No significant differences were found for the F wave amplitude or persistence for any of the conditions. Stinear et al. (2006) interpret these results as showing that KMI modulates

excitability in the corticospinal system, while VMI does not. They suggest that KMI activates the somatosensory cortex, which has direct connections to M1, while the visual system has a more indirect relation to the internal representation of the body. It is these differences, they argue, which may explain the differential effects noted for VMI and KMI (Stinear et al., 2006). They suggest, in addition, that the absence of F wave facilitation during any of the conditions means that KMI must work at the supraspinal level, facilitating activity in M1 and the corticospinal conduit.

Some aspects of Stinear et al.'s (2006) analysis and presentation of data may, however, require further comment. First, in their comparison of KMI during task performance and following task performance a one-tailed *t* test was used (Stinear et al., 2006). This is more powerful than the two-tailed test which would normally be used. As a consequence it is easier to find a significant difference between test means and can make a type I error, the mistaken rejection of a null hypothesis, more likely (Vincent, 1998). Second, although Stinear et al. (2006) provide a figure showing levels of MEP under the four conditions which does show a higher MEP in APB for KMI, this is a reading from only one individual. Further, this figure is annotated as representing the 'central tendency' of the data (Stinear et al, 2006, p. 160). It is not, however, indicated which measure of central tendency was used. A median value, for example, would simply provide the middle value of the data, while the mean would take all the values into account: these could potentially give very different results (Vincent, 1998). In conclusion, therefore, while this paper may demonstrate increased corticospinal excitability for KMI, a less powerful *t* test and a more exhaustive presentation of data might make the extent of this increase easier to assess.

### **3.3.8 Behavioural effects of KMI and VMI.**

The preceding studies reviewed all focused on the neural consequences of mental motor imagery based in different modalities. Rodrigues et al. (2010), however, have attempted to distinguish the behavioural effects of KMI and VMI, looking at their respective influence on postural control as measured by centre of pressure (COP) displacement. COP is the point at which the ground reaction force, that is the equal and opposite force exerted from the ground to the body, acts (van Deursen & Everett, 2005). In a stable upright posture COP is generally located comfortably inside the

individual's base of support and stability limit; COP then varies in amplitude as the person sways (van Deursen & Everett, 2005).

Rodrigues et al. (2010) measured changes in COP for 18 healthy subjects over 4 conditions including performance of plantar flexion from standing, KMI of this task, VMI of another person performing the task and a control condition in which the individual was asked to mentally sing a well known song. Body sway was measured by working out the area of the ellipse in which displacement took place using a force platform. The mean frequencies, velocities and SDs of COP displacements were taken for anterior-posterior (AP) and medial-lateral (ML) paths. In addition, EMG was recorded from the right lateral gastrocnemius, the prime mover for the plantar flexion task, throughout all the conditions. The authors also made a calculation of instantaneous equilibrium points, which they defined as the locations of COP when the resultant horizontal force was zero. These were then interpolated to give the "rambling component" of COP displacement (Rodrigues et al., 2010, p. 744). The disparity between the trajectories of COP and rambling then provided a measure labelled "tremble". Thus, the COP values were decomposed into two components: ramble was felt to provide a clear measure of the body's centre of gravity (COG), the point at which the body's entire weight acts, while tremble was thought to provide an error value for COP and COG. This error measure is important as, during quiet stance, COG and ground reaction force are collinear (van Deursen & Everett, 2005).

Rodrigues et al. (2010) found that KMI led to significantly greater displacement of the COP ellipse as compared with VMI or rest. The effect of KMI on COP displacement was also significantly greater than for VMI. In addition, SD for the medial-lateral direction was also significantly larger for KMI than for VMI, although the SDs for both were not found to be significantly different when compared with a rest condition. The mean velocity of COP displacement was also significantly larger for the KMI task in the AP direction, when compared with control and VMI. Mean velocity and SD for tremble in the AP direction were also significantly greater for the KMI condition. Ramble, however, did not show any effect of MI modality. EMG did not vary significantly across the control or imagery conditions, although data from only 13 participants was used in the analysis. Raw root mean square values in

millivolts (mV) for EMG were nearly identical for VMI and KMI (Rodrigues et al, 2010).

In their discussion of these findings, Rodrigues et al. (2010), in common with some of the authors cited above, draw on Jeannerod's (2006) hypothesis of motor simulation. They suggest, in keeping with Jeannerod, that MI recruits similar cerebral regions to those engaged in executed movement. Further, they argue that KMI is more likely than VMI to invoke activation in motor areas, including M1, and speculate that this may then lead to a more effector specific activation of the appropriate body parts for the relevant task.

One further point considered by Rodrigues et al. (2010) in relation to the Jeannerod hypothesis is that the effects noted for KMI may have resulted from inadequate suppression of the motor command at the spinal level, leading to EMG activation which may have had an impact on postural mechanisms. The authors maintain, however, that this is unlikely, as no differences were noted in the EMG signal across the imagery or control conditions. They do, however, acknowledge that a subliminal activation may have been present which went undetected.

Rodrigues et al. (2010) also relate their findings to the concept of the forward model, suggesting that KMI may have produced a predictive forward model of the movement, which resulted in augmented activation of the gamma motor neuron. The gamma motor neuron innervates the muscle spindle and helps make adjustments to the excitability of the spindle as the muscle is stretched (Pearson & Gordon, 2000). Rodrigues et al. (2010) quote Gandevia, Wilson, Inglis, and Burke (1997) in support of this suggestion. However, Gandevia et al.'s (1997) findings do not fit satisfactorily with another of Rodrigues et al.'s (2010) results. This is because the earlier authors explicitly stated that they could not find evidence for isolated gamma motor neuron activity during MI without a corresponding increase in EMG. Rodrigues et al. (2010), however, did not find any evidence of raised EMG activity in either of the imagery conditions. In addition, Gandevia et al. (1997) used indwelling electrodes, which are likely to have produced more accurate results than the surface electrodes used by Rodrigues et al. (2010; Kamen & Gabriel, 2010). If Gandevia et al. (1997) were correct, it is unlikely, therefore, that there would have been any differential activation

of the gamma motor neuron system. Also, Gandevia et al.'s (1997) study did not control for imagery modality: the participants were simply asked to imagine the specified movements. While Rodrigues et al. (2010) do, therefore, provide convincing evidence for a significant effect of KMI on postural sway, it might not be the case that this resulted from differential activation of the gamma motor neuron. It may be argued, then, that the precise mechanisms by which KMI could produce such an effect remain an open question.

A further study which attempted to dissociate the behavioural effects of KMI and VMI was carried out by Guillot, Collet, and Dittmar (2004). These authors hypothesized that elite tennis players and gymnasts would rely primarily on different sensory modalities in their mental imagery training. They posited, in addition, that learning styles might vary according to the degree to which the two groups of athletes needed to distinguish details against a visual backdrop.

Guillot et al. (2004) made use of the theory of field dependence. This implies that the more field independent sportsperson would need to separate objects from the surrounding visual scene, while the field dependent athlete would not be required to do this. In addition the field dependent player would rely more on vision, while the field independent athlete would depend more on "gravitational or egocentric" input (Guillot et al., 2004, p. 192). The authors then made a further distinction between tennis and gymnastics on the basis of skill type: tennis, which takes place in an unpredictable environment to which the player must respond to produce a desired outcome, is an open skill; gymnastics, on the other hand, is a closed skill, in which the individual aims to reproduce a highly stereotyped motor pattern. Their experimental hypotheses were that tennis players would be more field independent and would benefit more from VMI, while gymnasts would be more field dependent and rely more on KMI.

Ten tennis players and ten gymnasts were tested. First, they were asked to complete a figure-ground distinction test. This was intended to test their degree of dependence on the visual field. They were then asked to imagine skill sets derived from their own sports using either VMI or KMI randomly. The real time needed to complete these tasks had been recorded prior to the test and the imagined time was

determined using autonomic response measurements. The ratio of KMI time to VMI time was then calculated. A ratio higher than one signified more time spent on VMI, a ratio below one indicated more time on KMI, while a ratio of one pointed to an equal amount of time spent on both.

Guillot et al. (2004) found that gymnasts, who were shown to be more field independent during the figure-ground test, had a MI ratio closer to one. The authors suggested that this indicated that the gymnasts were able to use KMI and VMI equally well. It was also found that five tennis players had a ratio higher than one, while four had a ratio lower than one, suggesting 50% of tennis players favoured KMI and 40% preferred VMI. Commenting elsewhere on these inconclusive results, Guillot, Debarnot, Louis, Hoyek, and Collet (2010) state that “it seems premature to draw final conclusions regarding the comparative effects of the different types and perspectives of imagery” (p.217). It would appear then, based on these findings, that the respective uses of VMI and KMI in a motor training programme are not at all obvious.

Féry (2003) conducted an experiment on 24 healthy individuals to attempt to determine if KMI or VMI were more suited to learning a drawing task. The kinaesthetic task involved learning a form by tracing it with their finger for exactly 20s; the visual task involved viewing the form only. Once the form was traced or viewed the participants were asked to mentally image it and then draw the form. In addition, a control group practised an unrelated mental task.

Féry (2003) hypothesized that the KMI group would perform better when asked to reproduce the length of time needed to trace the form, and that the VMI group would perform best when asked to reproduce the form as accurately as possible. It was found that on the accuracy task the VMI group performed significantly better on the first two trials of the task, although on the final trial the difference was only significant when compared with the performance of the control group. When reproduction of the duration of the task was analyzed it was found that the KMI group performed significantly better than the VMI group, though again only for the first two trials.

Féry (2003) conducted a second experiment in which 24 healthy volunteers had to trace a path in the same form as described above using a stylus. Each time the stylus touched the sides counted as an error and the participants had to complete the



task as accurately as possible within a time limit of 70 s. In addition the duration of the errors was measured. The experimental design was the same as previously, with one group using KMI of the task, one using VMI, and a control group which performed an unrelated mental task. It was found that the KMI group produced significantly fewer errors on the task. It was also revealed that the KMI group produced significantly shorter error durations when compared with VMI, though this was only true for trial one.

This pair of experiments does seem to demonstrate differential effects for learning with VMI and KMI. In particular, KMI would seem better suited to learning the timing of a motor sequence and the accurate motor control needed to reproduce the sequence. VMI, on the other hand, seems more appropriate for learning to reproduce the visual form of the stimulus. It should be underlined, however, that these results were not consistent across all trials, and that in the final trial the differences between KMI and VMI became non-significant. This may suggest that the learning effect over repeated trials became more important than the modality of the mental imagery technique employed by the imager.

### **3.3.9 The effects of KMI and VMI in stroke.**

Despite the research discussed in the preceding paragraphs suggesting that KMI and VMI might be dissociable phenomena on a number of levels, and speculation that one or the other may be more useful in stroke rehabilitation, little work has been devoted to making a direct comparison of the relative efficacy of KMI and VMI in motor recovery following stroke. Indeed, a search of the literature produced only one result. This is discussed below.

Kim, Oh, Kim, and Choi (2011) conducted a study of 15 individuals with lower limb hemiparesis following stroke. The participants' performance was measured with a clinical measure, the timed up-and-go test, and EMG was recorded from the affected quadriceps, hamstrings, gastrocnemius and tibialis anterior (TA) of the hemiplegic leg. In addition, kinematic data were recorded from the foot, knee, thigh and pelvis of the hemiplegic side. The patients were tested under four conditions: KMI and VMI alone, and KMI and VMI which incorporated an external auditory signal which specified the gait rhythm.

For the timed-up-and-go test the authors found a significantly faster performance for KMI with the external auditory cue when compared with VMI alone (Kim et al., 2011). In addition, the time to complete the task was also significantly shorter for VMI with the auditory cue, when compared with VMI alone. However, there were no differences found for VMI alone when comparing pre-test measures, post-test measures and measures taken after one hour. Similarly, there were no significant differences for KMI on these timed measurements. Indeed, the results seem to be rather random, with very high  $p$  values of .948 for VMI and .756 for KMI. It would seem, therefore, that on this clinical outcome measure KMI and VMI made no difference to task performance. Differences emerged only when imagery training was paired with the auditory cue.

The EMG recordings showed significantly higher activations for gastrocnemius during stance phase and for the hamstrings during the swing phase of gait for KMI with an auditory cue when compared with VMI alone (Kim et al., 2011). When a comparison for pre- and post-test and a follow-up measure one hour after testing was made, EMG values for the four muscles during the swing phase were significantly greater for VMI with the external cue and KMI with the external cue. However, for KMI alone the EMG values were only significantly greater following training for three muscles, with gastrocnemius being unaffected. VMI was only linked to significantly higher EMG in hamstring and TA (Kim et al., 2011). During the stance phase of gait the post- and follow-up EMG values were significantly higher in the hamstrings for VMI and for hamstrings and gastrocnemius for KMI (Kim et al., 2011). KMI with an external cue was linked to significantly higher EMG activation differences in all the muscles. VMI with the external cue was only linked to significantly greater activation in the hamstrings and gastrocnemius.

With regards to the kinematic measures, there was a significantly larger displacement for KMI alone when pre-, post- and follow-up measures were compared for the knee and ankle joint. VMI alone was not linked to significant differences in any of the joints. KMI combined with an auditory cue was linked to significantly greater displacement in the knee and ankle, whereas VMI with the cue was only linked to greater displacement in the ankle. In addition, there were significantly higher

displacements at the ankle joint on post- measures for KMI, KMI with an external cue and VMI with an external cue (Kim et al., 2011). There were, however, no differences in the hip angular displacement values for any of the conditions when pre-, post- and follow-up measures were compared for each condition. When a direct comparison was made between the conditions, the only significant difference was found to be that KMI with the external cue was linked to greater knee displacement than KMI alone.

The results of the study of Kim et al. (2011) do seem to suggest that KMI may be more effective in promoting gait pattern recovery following stroke. However, it might be argued that the results are not unambiguous. For the timed up-and-go test, for example, significantly faster times were only found for KMI when it was paired with an external auditory cue, suggesting KMI alone did not produce any significantly different outcomes. When EMG measurements were compared, KMI was linked to increased activation in three muscles, and VMI with increased activity in two muscles (Kim et al., 2011). This would suggest that, while KMI had a more widespread effect, the difference between the two modalities was one of degree. Further, it is not clear why the effects of the two modalities would be limited to specific muscles. It is interesting to note, in addition, that the findings of this study are in contrast to those of Rodrigues et al. (2010), who did not find any difference in EMG levels in gastrocnemius for VMI or KMI.

With regard to joint kinematics, Kim et al. (2011) found that KMI was associated with increased displacement at the knee and ankle, while VMI did not seem to have any effect on displacement values for any joint. However, the fact that KMI was linked to increased values on only two joints may require further explanation. Further, when a between-comparison was made of the results for each condition, the only difference found was that KMI with an auditory cue was linked to higher displacement at the knee than KMI on its own. The study of Kim et al. (2011) had the benefit of using clinical, kinematic and physiological measures in order to provide a more complete picture of the effects of the MI tasks. It may be the case, however, that, while an advantage is noted for KMI in some conditions, there is a degree of variability in the results which point to the need for further investigation.

### **3.4 Kinematic Measurements Used in Experiment Two**

#### **3.4.1 Correlation of MCP, PIP and distal interphalangeal joint paths.**

The importance of looking at inter-joint coordination has already been stressed in chapter two. It was hypothesized for experiment two that MI training would lead to a stronger negative correlation between the MCP and the PIP and distal interphalangeal (DIP) joint paths for the hemiplegic index finger. A negative correlation was predicted because the movement pattern specified by the lumbrical grip requires the MCP joint flexion to increase, while the PIP and DIP joints are increasing in extension. Furthermore, it was hypothesized that KMI would have a stronger effect on this dependent variable than VMI.

#### **3.4.2 Mean DIP and PIP extension and mean MCP joint flexion.**

The lumbrical grip is characterized by the extension of the DIP and PIP joints with simultaneous flexion of the MCP joints (Palastanga et al., 2006). As shown in chapter two, the ability to extend the fingers is taken as an important landmark in the rehabilitation of the hand post-stroke (Beebe & Lang, 2009). It has been regarded as one index of the degree of intactness of the corticospinal tract and also a predictive indicator of how far the entire upper limb may recover (Smania et al., 2007). It was therefore hypothesized that MI would be linked with greater extension in the DIP and PIP joints, and also greater flexion of the MCP joint. It was further predicted that KMI would have a stronger effect in this respect.

#### **3.4.3 Overall MT.**

On the basis of the results of experiment number one, outlined in chapter two, it was predicted that the use of MI would lead to a faster MT for all three phases of the grasp. Furthermore, also given the findings of experiment one, it was predicted that KMI would be more likely to promote such an increased MT.

#### **3.4.4 MT during the approach phase.**

The MT during the approach phase of the grasp, as the hand moved towards the object, was also measured as in experiment one. Here as well, it was felt that this dependent variable might provide a plausible way to investigate the effects of MI training at the motor planning level of the movement. It was hypothesized that MT during the approach phase, as the hand moved towards the object, would be faster

when using motor imagery. As previously, it was also predicted that this effect would be more marked for the group using KMI.

#### **3.4.5 Standard deviations (SDs) of holding phase MCP, PIP and DIP joint paths and velocities.**

It has been argued by some researchers that reaching movements are constrained by an end-point hand posture that is specified by the motor control system prior to the start of the reach (Rosenbaum et al., 2001; Meulenbroek, Rosenbaum, & Vaughan, 2001). Some of these authors have therefore concluded that the minimization of end-point variability may be a key control parameter for the motor system (Harris & Wolpert, 1998; Rosenbaum et al., 2001). Rosenbaum et al. (2001) have maintained, in addition, that one feature of reaching movements in hemiplegia is an increase in end-point variability. It was therefore hypothesized that MI, and KMI in particular, would be associated with reduced end-point variability as the hand was grasping the object. Two measures of end-point variability were taken: the SDs of the MCP, PIP and DIP joint paths during the holding phase of the movement, and the SDs of velocity for those joints during the holding phase.

#### **3.4.6 Number of movement units (MUs) for MCP, PIP and DIP joints during the approach phase.**

MUs have already been defined and justified as a useful dependent variable in chapter two. There, it was suggested that the number of MUs during the approach phase of the grasp might provide a measure of the number of corrections made during the hand's approach towards the object. This might then be taken as a further indication of the balance of feed-forward control and control based on sensory feedback. It was hypothesized that MI would be associated with a reduced number of MUs for the MCP, PIP and DIP joints during the approach phase of the movement. Again, the secondary hypothesis was that KMI would be linked to a greater reduction in MUs.

### **3.5 Links Between 1<sup>st</sup> and 2<sup>nd</sup> Experiments**

The recovery of activity in the intrinsic hand muscles is seen as being crucial in regaining the use of the hand following stroke (Champion, Barber & Lynch-Ellerington, 2009). The central focus of both experiments one and two, therefore, was

on the use of MI in the promotion of activation of the intrinsic hand lumbrical muscles affected by hemiplegia following stroke. Both experiments used the performance of a 'lumbrical grip' following mental training with MI as an adjunct to mainstream physical rehabilitation.

A shared concern of experiments one and two was the need to define the sensory modality used in MI and instantiate this in the MI training script. This was in response to a review of the literature which suggested that, although MI could be embedded in different sensory modalities, the sensory content of imagery had not been treated as an independent variable by some researchers (for example, Page et al., 2005; 2007). It was also noted that, while some authors had asserted that kinaesthetic imagery was likely to be more effective in sensorimotor rehabilitation (for example, Mulder, 2007), little work seemed to have been done to test this. Experiment two therefore added a task which employed visual imagery in addition to the KMI and rest conditions already used in experiment one. In this sense, experiment two developed a theme which had been partially addressed in the first study.

Experiments one and two both used optoelectronic techniques to capture the rotatory movements of distal joints in the hemiplegic hand. The model of the hand used in experiment one allowed the capture of movement in the MCP and PIP joints. This model was then developed in experiment two so that the distal interphalangeal joint (DIP) could also be captured, allowing a more complete analysis.

Data had been reduced in the first experiment using time normalisation, interpolation with splines and ensemble averaging. This allowed both the construction of curves showing representative angular movements for each group and statistical analysis. The technique was also used in experiment two. However, experiment one also revealed some methodological problems which were subsequently addressed in experiment two. These are discussed below.

The start and end of the hand movement had been ascertained in experiment one by visual inspection of the captured movement. Although these points were then verified by checking them against the curves for the joint paths and velocities, the initial identification depended upon the subjective judgement of the researcher. A more reliable method was therefore deployed in experiment two, based upon initial

scrutiny of the angular velocity curves.

A further methodological problem identified in experiment one was the lack of repeated measures in the design. As has been explained above, the importance of this was underlined by the between-subject variability revealed in experiment one. Repeated measures were therefore included in experiment two.

The inclusion criteria were similar in both experiments, although some different instruments were used. It was felt that a more reliable cognitive screen was required, and so the Mini Mental State Examination (MMSE) was used. In addition, experiment two also involved an assessment of imagery ability using the “Kinesthetic and Visual Imagery Questionnaire” (KVIQ) (Malouin et al., 2004, p. 178).

Similar kinematic measurements were calculated for experiments one and two. Mean MTs had been found to be significantly different for the control and experimental groups in experiment one. Individuals using KMI had shown a faster MT. It was therefore felt important to include this measure as a dependent variable in experiment two, in order to test whether VMI had the same effect. Review of the literature had pointed to the potential importance of analysing the interactions of the joints, which is thought to be impaired in post-stroke hemiplegia (Cirstea & Levin, 2000; Levin, 1996). Both experiments therefore involved a calculation of the correlation coefficients of the mean joint angles during the phase in which the hemiplegic hand was actually grasping the object. Movement variability was also analysed because this has been shown to be a central problem for individuals with stroke (van Vliet & Sheridan, 2007). Both experiments involved measurements of MUs as a measure of movement variability. However, the measurement of variability was taken further in experiment two by analysing the SDs of the joint angles during the holding phase. These measurements were undertaken in response to literature which suggested that the variability of the movement endpoint is likely to be increased by disordered motor control (Harris & Wolpert, 1998).

### **3.6 Method**

#### **3.6.1 Recruitment sites**

Participants were once again recruited from Hospital Universitario Major and Barrios Unidos hospital. The characteristics of these hospitals have already been

described in chapter two. One person was also recruited via a contact from the university community.

### **3.6.2 Recruitment process for experiment two.**

Recruitment for experiment two took place from March to December 2010. The recruitment process for experiment two was, in all cases but one, the same as that for experiment one. Potential participants were identified during daily visits to the hospitals described above, again by referring to the daily therapy lists, from ward managers or from occupational or physiotherapists based in the hospital. When approached in the hospital, the researcher made the same initial clinical judgements as described above for experiment one. One difference was that for experiment two it was sometimes agreed to conduct the baseline screening tests and deliver the physical treatment in the person's home. This was done because, during recruitment for experiment one, some patients were lost to the study as they had been discharged from the hospital before the researcher was able to complete the baseline screening; for example, if they had requested more time to look through the information document.

The one exception in experiment two was a participant who was recruited via a university contact. This person had been discharged from any stroke services prior to making the initial contact. In this case, the initial contact was made by telephone. This was because the internal postal service in Bogotá is considered to be unreliable, making a telephone call the most efficient way to make this first approach. Once verbal permission was given by this person, the researcher visited him at home and the process from that point on was the same as that already described for other participants.

As for experiment one, all participants were given plenty of time to read the information sheet. If extra time was requested a follow up telephone call was arranged at a convenient time. If they were interested in taking part in the study, the screening tests were conducted, as previously explained for experiment one. If the individuals met the criteria, they were then asked to sign the informed consent form, which all participants did.

### **3.6.3 Sampling process for experiment two.**

As can be seen from the discussion of the recruitment procedure the



sampling technique for experiment two was the same as that for experiment one. It was also therefore a non-probabilistic convenience sample. Some limitations of this are enlarged on in the discussion section.

### 3.6.4 Participant characteristics.

All participants had either had an ischaemic or haemorrhagic stroke. The participants' demographic details are given in table 3:3.

Table 3:3

#### *Participant Characteristics*

Patient	Age	Gender	Time since stroke	Affected side
1	48	F	2 months	L
2	64	F	3 weeks	R
3	79	M	20 days	L
4	64	M	3.5 years	R
5	45	F	3 weeks	R
6	55	F	11 days	R
7	55	F	3 weeks	R
8	64	M	1 month	R
9	51	M	2 months 3 weeks	R
10	74	F	3 weeks	L
11	73	F	2 weeks	R
12	43	M	3 months	R
13	74	F	2 months	L
14	76	M	4 months	R
15	41	F	8 days	L

The inclusion criteria were: (a) hemiplegia affecting the hand resulting in a loss of dexterity and/or power as a result of the stroke; (b) a score of one or more on the hand activities component of the Motor Assessment Scale; (c) a score of more than 20 on the Mini-Mental State Examination (MMSE)<sup>1</sup>; (d) a score of four or less on the

<sup>1</sup>Participant 11 scored 13 on the MMSE. This person had dysphasia. However, she was fully orientated to time, place and person and did not show any evidence of apraxia. She was also able to follow verbal three stage commands. It was felt therefore, in line with the World Medical Association declaration of Helsinki, that she was competent to give informed consent and that her performance on the MMSE

Modified Ashworth Scale; (e) an ability to mobilize and transfer independently without an aid; (f) normal or corrected to normal vision; (g) not partaking in any other clinical studies.

Four people were excluded from the study who did not meet the criteria. All individuals who met the inclusion criteria agreed to take part in the study. The scores on the screening tests, including the KVIQ, are shown in table 3:4. In addition, a description of each participant's lesion site, where available, is given in table 3:5.

As for experiment one, once screening tests had been completed and it was ascertained if the person was suitable for the study, informed consent was gained via the signing of the patient information sheet.

### **3.6.5 Payments.**

As for experiment one, no payments were made for taking part in the study. Any transport costs were met by the researcher.

### **3.6.6 Sample size and characteristics.**

To the best of the author's knowledge, the hypotheses in this experiment had not been tested previously. There were, therefore, no data available upon which to base a calculation of sample size.

Following baseline testing, a group of 15 patients between the ages of 41 and 79 ( $M = 60.6$ ,  $SD = 12.9$ ) were recruited. Nine were female and six were male. All completed the study.

### **3.6.7 Safety considerations.**

The procedures were the same as for experiment one. No adverse effects were noted by the researcher or reported by the patients during the course of the study.

### **3.6.8 Pilot study.**

The experiment was piloted with one person with stroke (Female, 39y). The aim of the pilot was once again to identify problems with the overall experiment

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could be explained by her communication difficulties. The informed consent form was then read to her by her next of kin. Her next of kin then witnessed as she signed the form and countersigned. During treatment she was subsequently able to follow all instructions appropriately.

Table 3:4  
*Participants' Scores on Screening Tests*

Patient	MAS Upper Limb	MAS Hand	MAS Advanced Hand	Ashworth	MMSE	KVIQ (visual component)	KVIQ (kinaesthetic component)
1	6	5	2	3	27	44	43
2	5	5	1	3	27	67	75
3	5	1	1	5	32	51	34
4	6	6	2	3	29	68	68
5	6	5	2	3	24	57	48
6	6	6	3	3	28	84	45
7	6	5	2	3	29	53	65
8	5	5	2	3	28	15	18
9	5	2	0	4	23	66	78
10	5	5	2	3	28	32	17
11	6	5	0	3	13	64	65
12	6	5	3	3	27	43	45
13	6	5	2	3	29	25	53
14	6	5	1	4	21	69	33
15	5	5	1	1	27	44	54

Table 3:5

*Description of Lesion by Group*

Patient	KMI group
1	Small hyperdense lesions in posterior bilateral white subcortical matter.
2	Left basal ganglia haemorrhage.
3	Bilateral occipital and right pre-frontal ischaemic lesions.
4	Radiologist's report not available.
5	Ischaemic lesions in left pre-central, frontal and parietal lobes.
	VMI group
6	Infarct in left pre-central, posterior putamen and post-central parietal region.
7	Small cortical foci, suggesting haemorrhage.
8	Hyperdensity in left corona radiata, extending to left posterior internal capsule and left thalamus.
9	Infarct in left medial and superior temporal lobes.
10	Ischaemic area in right frontal region.
	Relaxation group
11	Radiologist's report not available.
12	Left basal ganglia haemorrhage.
13	Radiologist's report not available.
14	Infarct in left posterior insula, frontal lobe and inferior cerebellum.
15	Hyperdensity in right frontal lobe, anterior and lateral fossa of left temporal and inferior frontal lobes.

design prior to commencing the study. The specific objectives were: (a) to check that the cameras could see all of the markers on the hemiplegic hand; (b) to determine if the delivery of the taped MI and relaxation scripts via headphones was feasible; (c) to trial the use of the randomisation software in an experimental situation; (d) to identify any other problems in the research design. Following this pilot it was decided to alter the research design so as to include repeated measures after treatment.

**3.6.9 Measures used.**

The Motor Assessment Scale (MAS) was used as a screening assessment of hand function. An assessment of upper limb muscle spasticity was carried using the

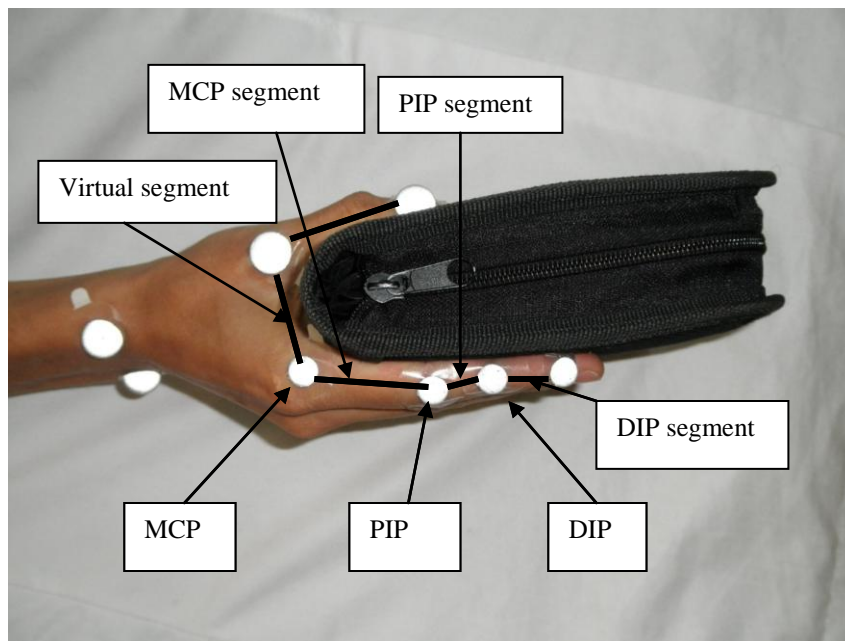
Modified Ashworth Scale. The details of both assessments have already been given for experiment one in chapter two. In addition, an assessment of cognitive function was carried out using the Spanish version of the Mini Mental Status Examination (MMSE).

Patients were also assessed performing KMI and VMI using a Spanish translation of the KVIQ. This was translated into Spanish from English by a native Spanish speaker who held the Cambridge Certificate of Proficiency in English, which is the highest level qualification for speakers of English as a second language awarded by the University of Cambridge, UK. Issues regarding the translation of this instrument are outlined further in the discussion section of this chapter.

All testing and treatment was carried out by the researcher, a band seven occupational therapist whose training and experience has already been described in chapter one.

#### **3.6.10 Joint kinematics and mechanical model of hand.**

Infrared reflective markers were placed on the index finger tip and the distal interphalangeal (DIP), the proximal interphalangeal (PIP) and the metacarpophalangeal (MCP) joints of the index finger of the hemiplegic hand. In addition, a marker was placed on the thumb MCP. Markers were also attached to the thumb tip and the ulnar and radial styloid processes, although these markers were not used in the mechanical model of the hand for subsequent kinematic analysis. The location of the markers is shown in figure 3:1.



*Figure 3:1.* Location of joint markers. The diagram also shows the biomechanical model of the hand.

Joint angles were defined in the same way as for experiment one, as the attitude of two bony segments in relation to each other. The bony segments of the index finger were labelled following the model outlined by Zatsiorsky (1998). This breaks the finger into a distal phalangeal segment, a proximal phalangeal segment and a metacarpophalangeal segment. The thumb was treated as one segment, defined by markers at the thumb MCP joint and the thumb tip. In addition, a virtual segment was defined which extended across the thumb web space and linked the thumb MCP joint and the MCP joint of the index finger.

For joint kinematics analysis the angles were defined as follows: (a) the distal interphalangeal joint angle (DIP) was defined as the vertex of the distal interphalangeal segment and the proximal phalangeal segment; (b) the proximal interphalangeal joint angle (PIP) was defined as the vertex of the proximal phalangeal segment and the metacarpophalangeal segment; (c) the MCP joint angle was defined as the vertex of the metacarpophalangeal segment and the virtual thumb web space segment. The biomechanical model of the hand is shown in figure 3:1.

### **3.6.11 Research design.**

A between-within design was used with measurements repeated of one factor. The between factor was 'group' (KMI x VMI x Relaxation). The within factor was 'time' (Pre-treatment x Post-treatment). Repeated measures were of each of the dependent variables.

### **3.6.12 Apparatus.**

The participants were required to grasp the same compact disc case already described in chapter two. The motion capture system and reflective markers were also identical to those already described in experiment one. The experimental set-up can be seen in figure 2:2 in chapter two. In addition the computer software used was the same as that already described in chapter one.

The video and commentary explaining the experimental task to the patients were presented via a laptop computer. The MI and relaxation tasks were delivered via headphones from a tape recording. The narrator of the recorded scripts was the same as that described for experiment one.

Motion capture data were exported as text files and stored on spreadsheets. Data reduction was accomplished using MATLAB version 7.4.

### **3.6.13 Therapy interventions.**

The mainstream physical therapy intervention for experiment two was based on the same protocol as for experiment one, which can be read in appendix 2:6. For experiment two, however, the mainstream therapy was not delivered solely in the hospital setting, but also in the patients' homes and in the laboratory. The therapy required no specialist equipment, and so the site of the intervention actually made very little difference. The motivation here was that, while the treatment location was restricted to the hospital, as in experiment one, participants were required to make a journey to take part in this stage of the research. In addition, as shown above, patients who were discharged home prior to giving informed consent were lost to the first experiment.

The timing of the therapy intervention was also the same, with each separate therapy session lasting for around 15-20 min. One difference was that only two therapy sessions were delivered prior to motion capture while three had been delivered

in experiment one. This was largely a time saving measure, as the sample size for this experiment was larger than for experiment one. Given that it had taken ten months to complete experiment one, it was felt that delivering three separate sessions of mainstream therapy may have been prohibitive with respect to time with this larger sample. Some limitations regarding the timing of the intervention are outlined in the discussion section of this chapter.

#### **3.6.14 Timing of MI treatment.**

The scripts used for KMI and guided relaxation were the same as those used for experiment one. The recordings lasted for 7 min 20s and 7 min 24s respectively. A new recording was made of the KMI script. The KMI recording began with a relaxation script based on guided mindful breathing which was repeated five times and with each repetition lasting on average 29 s; the KMI script was then repeated five times, with each repetition lasted an average of 39 s. The VMI recording also began with a relaxation script based around mindful breathing, repeated six times and with each repetition lasting for an average of 25 s; the VMI task itself was repeated five times and each repetition lasted an average of 46 s. The VMI script can be read in appendix 3:1. The relaxation script began with guided breathing once more, repeated three times, with each repetition lasting an average of 26 s; the relaxation task was repeated 6 times, with each repetition lasting on average of 29 s.

#### **3.6.15 Experimental procedure.**

Once the physical treatment sessions were completed reflective markers were placed on the person's hemiplegic hand, following the layout shown in figure 3:1. The person was seated in a comfortable posture with a desk placed in front of them throughout, as shown in figure 2:2 of chapter two. Each participant was seen separately on different days.

Each person then watched a video showing a hand grasping the black compact disc case using a lumbrical grip. The video was accompanied with a recorded commentary explaining that grasping the object with the lumbrical grip was the task which the participant had to perform for the experiment. A verbal description of the lumbrical grip was also given in the commentary. There were four alternative videos: two showing a female right and left hand performing the grip; two showing a male



right and left hand executing the task. The video to be viewed was selected by the researcher on the basis of the gender of the patient and which side was affected by the stroke. Thus, a woman with a left-sided hemiplegia would view the video of the female left hand, a man with a right-sided hemiplegia would view the video of the male right hand, and so on.

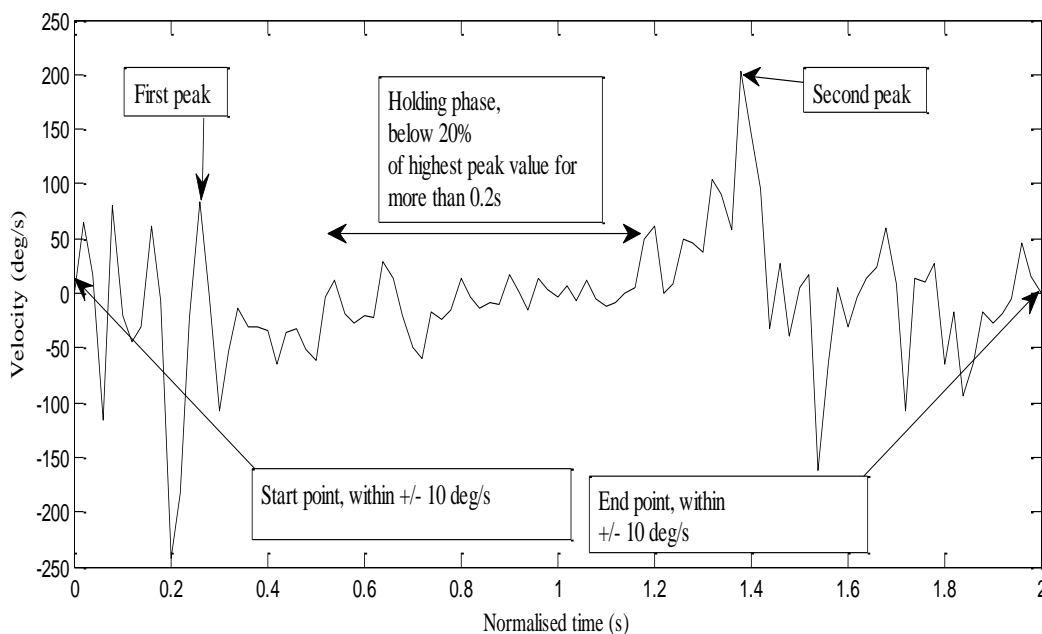
The participants were then allocated to one of three groups. These were the KMI group, the VMI and the Relaxation (control) group. Eleven of the participants were allocated using Random Allocation Software (Saghae, 2004). Four were not randomized. This occurred because the researcher was both administering the physical treatment sessions and allocating to group. The KMI and VMI groups were filled first by chance, through random allocation, while only one patient had by that point been allocated to the relaxation group. Since the sample size for each group was set at five, and since it was only possible to allocate patients individually as they consecutively took part in the experiment, it was therefore clear in advance that the final four patients would have to go to the relaxation group. The physical treatment given to these last four patients was, however, based on an identical protocol to the rest. In addition all stimulus materials for the experimental manipulation were presented in a standardised, pre-recorded, format. It is felt, therefore, that this breakdown in the randomisation process should not have posed a threat to the internal validity of the research. This issue is addressed further in the discussion.

Following allocation to group the participants were required to place the hand affected more by the stroke in a comfortable starting position on a desk in front of them. They were then required to reach and grasp the compact disc case using the lumbrical grip. Fourteen grasps were performed and these were recorded using the optoelectronic motion capture system already described in chapter two.

#### **3.6.16 Data analysis and reduction.**

The method used to label angles and calculate path, velocity and acceleration was the same as that already outlined for experiment one and explained in chapter two. However, the method used for identifying the start and end point for each movement was different in this experiment. Movement onset and offset points for each grasp were identified by looking at the velocity curve for the PIP joint. Firstly, two velocity

values within  $\pm 10$  degrees per second (deg/s) were identified. If the curve between these two points had two distinct peaks enclosing a secondary section where the velocity fell to below 20% of the peak velocity of the main curve for more than 0.2s, it was judged to represent the movement of the joint during one grasp. This was because the values of  $\pm 10$  deg/s were taken as points where the joint was nearly still, prior to and following reaching and grasping. The secondary section, where there was a drop in velocity for more than 0.2s, was then taken as the holding phase, in which the hand was actually grasping the object. One such movement cycle is shown in figure 3:2.



*Figure 3:2.* Plot of velocity (deg/s) profile for the PIP joint during one grasping movement for participant two, prior to KMI treatment. Data are interpolated and time normalised. Labels show landmarks used to distinguish the start and end of the grasp, the peak velocity and the holding phase.

Five such movement cycles were identified for the PIP joint. All data outside of these five movements were then removed from the columns of values for PIP angular path, velocity and acceleration, leaving five movement cycles for analysis.

The five movement cycles for the PIP joint were then used to identify the movement cycles for the MCP and DIP joints. This was done by placing the uncut columns of data for MCP and DIP angular velocity next to the five velocity vectors for the PIP joint. Velocity values of  $\pm 10$  deg/s that were located as closely as possible to the start and end points for the five movement cycles for the PIP joint were then identified. All other data were then removed, giving five movement cycles for the MCP and DIP joints which coincided closely with those of the PIP joint. The uncut joint path and acceleration data columns for each joint were then placed next to the column containing the five velocity vectors for that joint. All values falling outside the values matching those of the five velocity profiles were removed, giving five angular path and acceleration profiles for each joint. The theoretical rationale for the method used to identify the movement cycles is explained in the discussion section of this chapter.

Wherever possible, the five movements were selected from the middle of the sequence of 14 movements initially captured, in order to control for initial practice time and the possible effects of fatigue towards the end of the grasping period (Schmidt & Lee, 2005). However, artefactual issues occasionally meant that all of the five central movements were not adequately captured, and in these cases it was therefore necessary to select movements from nearer the start or end of the 14 movement sequence. In addition, for patient nine, artefactual problems meant that only three clear movement cycles could be identified for the DIP path, velocity and acceleration profiles prior to VMI treatment. Analysis for this patient's DIP joint in this condition was therefore carried out on three movement cycles only.

The data reduction techniques of time normalisation, interpolation with cubic splines and the construction of ensemble averaged data were used in this experiment. This has already been discussed in detail in chapter two for experiment one. Five columns of equal length for each of the MCP, PIP and DIP angular paths, velocities and accelerations during each of the five movements<sup>2</sup> were created for each movement. These columns were then averaged for each person to form an ensemble average for path, velocity and acceleration for each joint for that individual. Following

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<sup>2</sup> With the exception of participant nine, as noted above.

this, these ensemble averages for each of the five groups were then themselves averaged. This gave a group ensemble average for path, velocity and acceleration for each joint for each of the three experimental groups.

The calculation of group ensemble averages allowed the identification of the phases of the grasping movement for each group. The method used to achieve this was the same as that already described for experiment two. This process is illustrated in figure 3:3 for the ensemble average curve of the PIP joint path and velocity of the KMI group prior to treatment. It can be seen that the phase changes identified from the curve for joint path, shown in the top panel, coincide with velocity values which are close to zero.

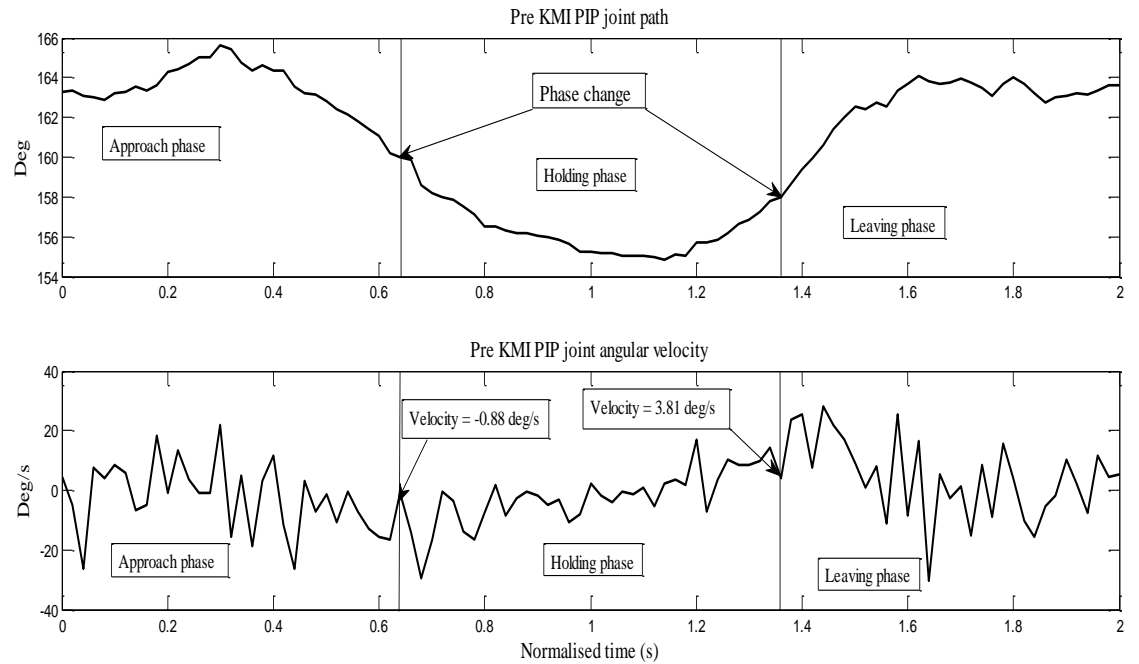
### **3.7 Results**

#### **3.7.1 Participant flow.**

Nineteen participants were assessed for eligibility. Four were excluded as they did not meet the inclusion criteria. Fifteen completed the study. The flow of participants through the study is shown in figure 3:4.

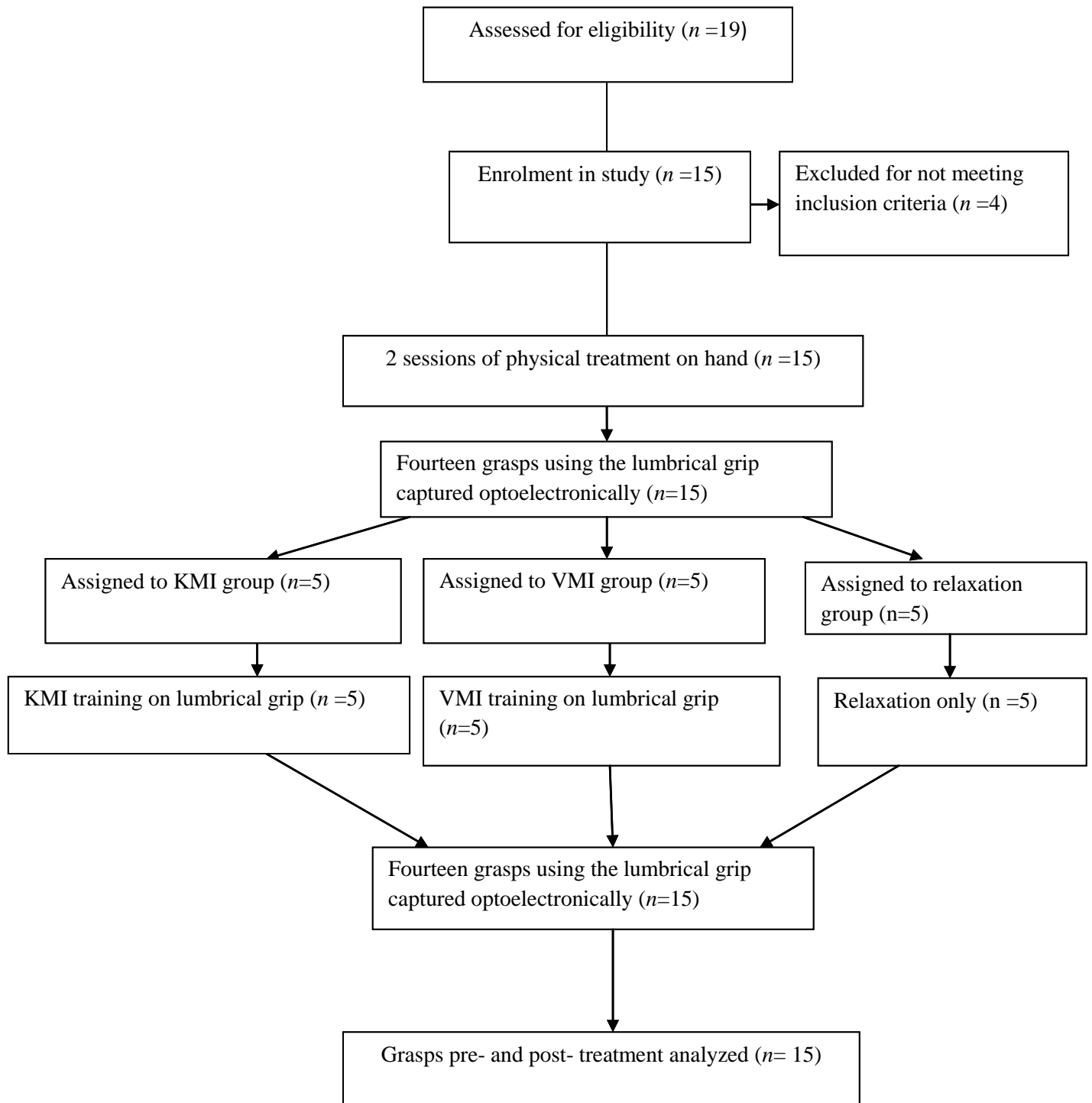
#### **3.7.2 Statistical analysis.**

The dependent variable measures were compared using 3 x 2 (Group [KMI, VMI, relaxation] x Time [pre-treatment, post-treatment]) between-within factorial analysis of variance (ANOVA). There were repeated measures of the factor 'Time'. To control for multi-sample sphericity, results were subject to the Greenhouse-Geisser correction. Any significant main or interaction effects were subjected to post hoc testing using Tukey's Honestly Significant Difference (HSD) test. In addition, testing was carried out using Pearson's product-moment correlation coefficient ( $r$ ). The threshold value for statistical significance for all tests was set at  $p = < .05$ .



*Figure 3:3.* Plots of PIP joint path (upper plot) and PIP joint angular velocity (lower plot) prior to KMI treatment, showing the phase changes of the grasp and the angular velocity values at those points.

Figure 3:4. Participant flow through study



### 3.7.3 KVIQ results for experiment two.

The results for the KVIQ were compared for significant differences using a one way ANOVA. There were no significant differences for the visual component between the groups at baseline,  $F(2, 12) = 0.271, p = .767$ . Nor were there any significant differences for the kinaesthetic component at baseline,  $F(2, 12) = 0.259, p = .776$ . The results are shown in figure 3:5.

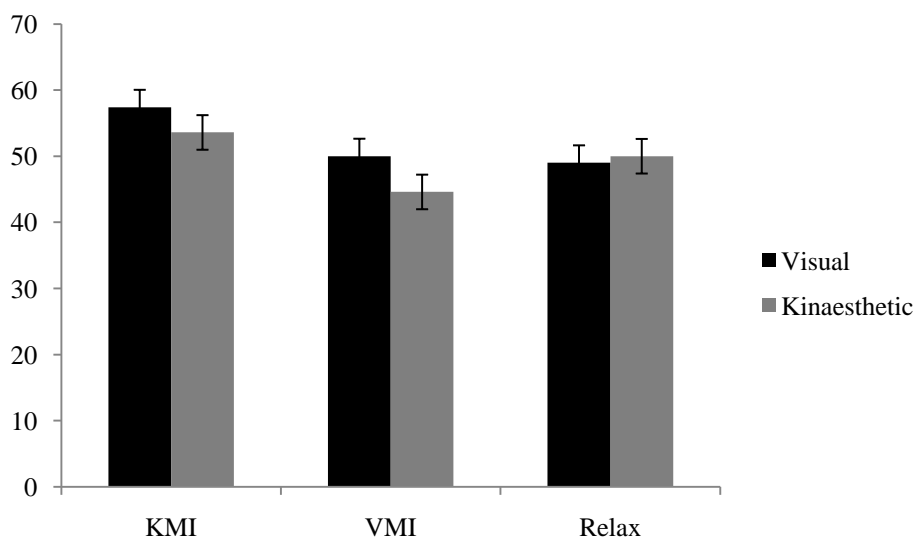


Figure 3:5. Mean scores for KVIQ

### 3.7.4 Motor Assessment Scale for experiment two.

The results for the hand component of the MAS were compared at baseline using a one way ANOVA. There were no significant differences noted for the upper limb component of the MAS,  $F(2,12) = 0.75, p = .493$ . Nor were there any significant differences for the hand component of the MAS,  $F(2, 12) = 0.23, p = 0.798$ . Furthermore, there were no significant differences between the scores for the advanced hand component of the MAS,  $F(2,12) = 0.214, p = .81$ . The results are illustrated in figure 3:6.

It was therefore the case that none of the groups differed significantly at

baseline with relation to visual or kinaesthetic motor imagery ability or upper limb or hand function.

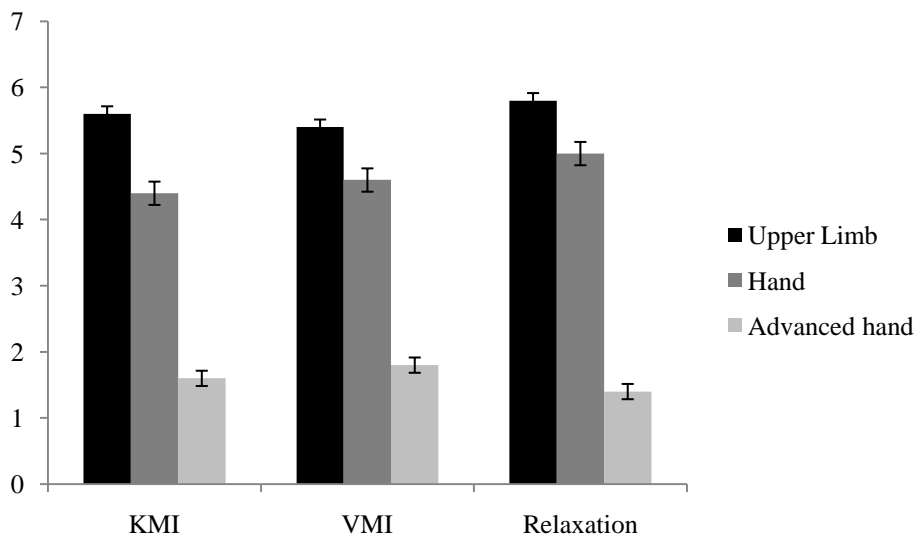


Figure 3:6. Mean scores on MAS components

### 3.7.5 Correlation of MCP, PIP and DIP paths during the holding phase.

The correlations of the mean values of the MCP, PIP and DIP joint paths during the holding phase of the movement were compared before and after treatment. The correlation of the MCP and DIP joints prior to VMI treatment was not statistically significant,  $r = -.82$ ,  $p = .09$ . However, the correlation of the MCP and DIP joints following VMI treatment was statistically significant,  $r = -.95$ ,  $p = .01$ . Importantly, this was a negative correlation, in line with the experimental hypothesis. One anomalous finding was that the correlation between the MCP and DIP joint for the relaxation group was statistically significant prior to treatment,  $r = -.92$ ,  $p = .03$ , though non-significant following treatment,  $r = -.67$ ,  $p = .22$ . The correlation matrices for all joints are shown in table 3:6. In addition, the pre- and post-VMI MCP and DIP values are shown in a scatter plot in figure 3:7.



Table 3:6.

*Correlation Coefficients (r) of Joint Angular Paths During the Holding Phase Before and After Treatment*

	Pre-KMI (N=5)			Post-KMI (N=5)		
	MCP	PIP	DIP	MCP	PIP	DIP
MCP	–			MCP	–	
PIP	-0.64	–		PIP	0.49	–
DIP	-0.13	0.01	–	DIP	0.60	0.66

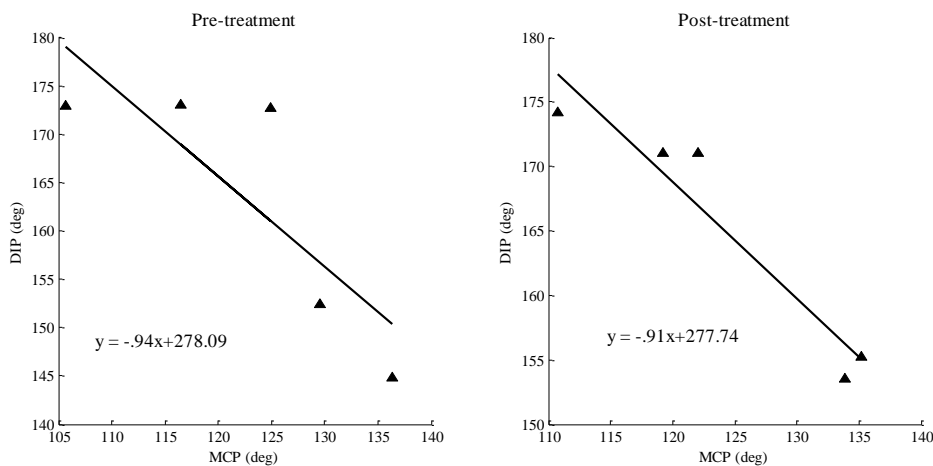
  

	Pre-VMI (N=5)			Post-VMI (N=5)		
	MCP	PIP	DIP	MCP	PIP	DIP
MCP	–			MCP	–	
PIP	-0.56	–		PIP	-0.70	–
DIP	-0.82	-0.55	–	DIP	-0.95*	0.54

	Pre-relax (N=5)			Post-relax (N=5)		
	MCP	PIP	DIP	MCP	PIP	DIP
MCP	–			MCP	–	
PIP	-.82	–		PIP	-.61	–
DIP	-.92*	0.64	–	DIP	-.67	-.09

\* $p \leq 0.05$ .



*Figure 3:7. Scatterplots of mean values for MCP and DIP joint angular paths during the holding phase, pre- and post- VMI treatment, showing best fit lines and linear equations.*

### 3.7.6 Mean PIP, DIP and MCP extension during the holding phase.

#### *MCP.*

The mean values for the MCP joint path during the holding phase were compared for significant difference. The results for the main effect of time were not statistically significant,  $F(1, 12) = 0.125, p = .73$ , neither were the results for the main effect of group,  $F(2, 12) = 0.607, p = .561$ , nor the interaction effect of time and group,  $F(2, 12) = 1.372, p = .291$ .

#### *PIP.*

The PIP joint path mean values were not statistically different for the main effect of time,  $F(1, 12) = 3, p = .109$ , nor group,  $F(2, 12) = 0.332, p = .724$ . Neither was there a significant interaction effect for time and group,  $F(2, 12) = 1.083, p = .37$ .

#### *DIP.*

The DIP joint path mean values were not significantly different for the main effect of time,  $F(1, 12) = 0.076, p = .788$ . The results for main effect of group were not significant either,  $F(2, 12) = 1.58, p = .246$ , nor were those for time and group interaction,  $F(2, 12) = 0.413, p = .671$ .

The mean and standard deviations of the joint paths for MCP, PIP and DIP before and after treatment for each group are shown in table 3:7.

Table 3:7.

*Mean (standard deviation) Extension (deg) of Finger Joints During the Holding Phase*

	KMI		VMI		Relaxation	
	Pre	Post	Pre	Post	Pre	Post
MCP	115.81(107)	117.95(12.12)	122.59 (11.9)	124.22(10.29)	118.09 (10.09)	115.64(11.29)
PIP	158.32(9.57)	157.77 (9.7)	155.62(12.41)	150.03(18.36)	152.96 (13.98)	151.4 (10.55)
DIP	171.21(5.65)	171.3 (2.79)	167.78(10.22)	167.42(9.4)	163.73 (4.27)	162.86 (7.76)

### 3.7.7 Standard deviation (SD) of MCP, PIP and DIP joint path during the holding phase.

#### *MCP.*

The results for SD of the MCP joint path during the holding phase were not

significantly different for time,  $F(1, 12) = 0.02, p = .89.$ , nor for group  $F(2, 12) = 0.125, p = .884.$  Neither was there a significant interaction effect for the two factors,  $F(1, 2) = 2.927, p = .092.$

### **PIP.**

The SDs of the PIP joint path for the holding phase did not show a significant main effect for time  $F(1, 12) = 0.061, p = .81,$  nor group  $F(2, 12) = 1.112, p = .36,$  nor a significant interaction effect for time and group,  $F(2, 12) = .665, p = .375.$

### **DIP.**

The SDs for the DIP joint path did not show a significant main effect for time,  $F(1, 12) = 0.047, p = .831,$  or interaction of time and group,  $F(2, 12) = 0.16, p = .831.$  However, there was a significant main effect of group,  $F(2, 12) = 5.456, p = .021.$  Post hoc testing showed that the mean SD of the DIP joint path for the relaxation group was significantly higher than the KMI or VMI group.

All mean (SD) MCP, PIP and DIP SDs of angular path values for the holding phase are given in table 3:8.

Table 3:8.

*Mean (SD) of Standard Deviation of Joint Angular Paths (deg) During the Holding Phase*

	KMI		VMI		Relaxation	
	Pre	Post	Pre	Post	Pre	Post
MCP	2.38(1.32)	2.23(1.56)	2.29(1.69)	1.55(0.98)	1.75(0.87)	2.77(1.97)
PIP	1.39(1.14)	2.04(1.76)	1.97(1.42)	1.8(1.52)	3.36(2.65)	3.09(2.15)
DIP	1.06(0.26)	0.98(0.47)	1.29(0.88)	1.08(0.73)	3.1(1.86)	3.24(2.07)

### **3.7.8 SD of MCP, PIP and DIP velocity during the holding phase.**

#### **MCP.**

The SDs for the MCP angular velocity during the holding phase did not show a significant main effect for time  $F(1, 12) = 2.731, p = .124.$  Neither was there a significant main effect for group,  $F(2, 12) = 1.055, p = .378,$  nor a significant interaction effect of time and group,  $F(2, 12) = 0.993, p = .399.$

**PIP.**

The SDs for the PIP angular velocity during the holding phase did not show a significant main effect of time,  $F(1,12) = 0.061, p = .81$ , nor of group,  $F(2, 12) = 1.112, p = .36$ . There was no significant interaction effect of time and group,  $F(2, 12) = 1.066, p = .375$ .

**DIP.**

The SDs for DIP angular velocity during the holding phase did not show a statistically significant main effect of time,  $F(1, 12) = 0.285, p = .603$ . However, there was a significant main effect of group,  $F(2, 12) = 8.265, p = .006$ . Post hoc testing revealed that the relaxation group had a significantly higher SD of angular velocity for the DIP joint during the holding phase. The interaction for time and group was, however, not significant,  $F(2, 12) = 0.709, p = .512$ .

The mean (SD) of the SDs of holding phase angular velocity for MCP, PIP and DIP for each group before and after treatment is shown in table 3:9.

Table 3:9.

*Mean SD (SD) Angular Velocity (deg/s) for MCP, PIP and DIP During the Holding Phase*

	KMI		VMI		Relaxation	
	Pre	Post	Pre	Post	Pre	Post
MCP	2.38(1.32)	2.23 (1.56)	2.29(1.69)	1.55(0.98)	1.75(0.87)	2.77(1.97)
PIP	14.58(5.2)	23.45(15.21)	15.93(9.72)	23.07(15.85)	40.2(14.38)	32.85(11.25)
DIP	15.17(3.84)	23.66(9.67)	15.48(10.1)	16.95(3.2)	56.28(36.05)	52.88(22.45)

**3.7.9 Mean MT.**

Mean MT for the whole grasping movement was calculated for each participant. These values were compared for significant difference using a between-within ANOVA. The main effect of time was significant,  $F(1, 12) = 6.294, p = .028$ . This shows that mean MT pre-treatment for all groups was significantly longer before treatment, at 2.91s, than after treatment, at 2.35s. The main effect of treatment was not significant,  $F(2,12) = 1.589, p = .244$ . Neither was the interaction effect of time

and treatment significant,  $F(2,12) = 1.378, p = .289$ . However, when the results for the individual groups were compared using a two tailed  $t$  test for dependent samples it was found that the mean MT prior to KMI treatment was significantly longer than that after KMI treatment,  $t(4) = 3.639, p = .022$ . These results are shown in figure 3:8. The differences before and after VMI were not significant  $t(4) = 0.59, p = .587$ , neither were those for the relaxation control condition,  $t(4) = 0.794, p = .472$ .

With regard to these significant findings for KMI it was of interest to ask whether the significantly shorter MT following treatment was simply an effect of maturation, in other words had the participants simply improved their MT due to practice performing the reach? To test this the first two MTs of each person prior to KMI were placed in a set of data along with the first two MTs following treatment. This set of data was called 'early', indicating that it represented the first two movement times for each patient in each condition. The final two MTs for each patient before and after KMI treatment were then placed in a single data set, labelled 'late', indicating that these were the final two MTs for each patient in each condition. These two data sets were then compared for significant difference using a paired samples two tailed  $t$  test. It was found that the difference between the two was not significant,  $t(19) = 1.33, p = .2$ . These results suggested that the significantly shorter movement time noted following KMI treatment was indeed likely to be related to the KMI treatment and not to an effect of practice.

#### ***3.7.10 MT during the approach phase.***

The above results for the MT were based on the overall MT. It was of interest to ask therefore if the MT was different during the different phases of the movement. A calculation was therefore made of the MTs during the approach phase only.

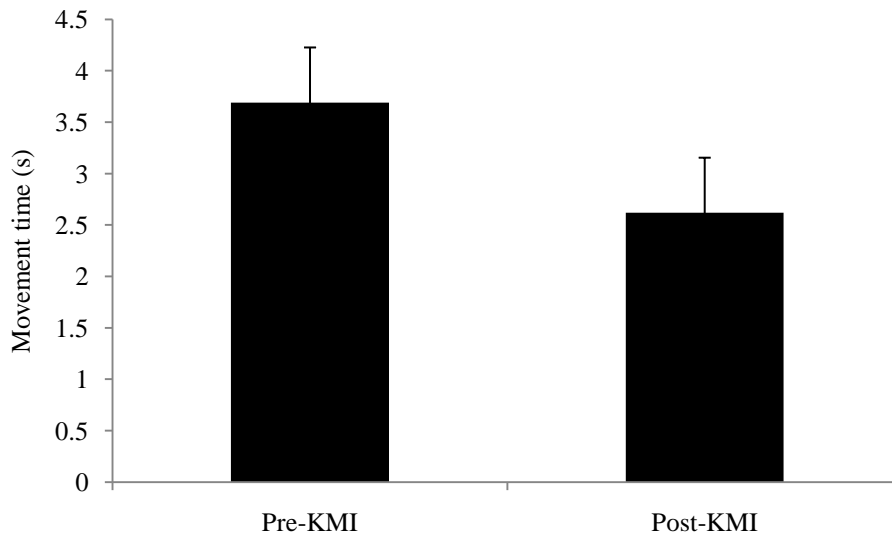


Figure 3:8. Mean overall MT for the KMI group

#### ***MCP MT.***

There was no significant main effect for time,  $F(1,12) = 1.175, p = .3$ . Neither was there a significant main effect of treatment,  $F(2, 12) = .746, p = .495$ , nor a significant interaction of time and treatment,  $F(2, 12) = .392, p = .684$ .

#### ***PIP MT.***

The MT during the approach time did not show a main effect of time,  $F(1, 12) = 1.968, p = .186$ . However, there was a main effect of treatment,  $F(2,12) = 4.215, p = .041$ . A one way ANOVA was therefore conducted on the single factor 'treatment'. This showed that the VMI group had significantly faster MT (0.55s) than either the KMI group (0.69s) or the relaxation group (0.66s),  $F(2, 27) = 4.421, p = .022$ . Further post hoc testing using a paired samples  $t$  test, however, showed that there was no significant difference for the VMI group before and after treatment,  $t(4) = 0.862, p = .437$ .

#### ***DIP MT.***

The MT during the approach showed no significant main effect of time  $F(1, 12) = .975, p = .343$ . Neither was there a significant main effect of treatment,  $F(2, 12) = 2.386, p = .134$ , nor of interaction for time and treatment,  $F(2, 12) = 1.127, p = .356$ .

#### **3.7.11 Movement Units (MUs) during the approach phase.**

**MCP.**

The number of MUs for the MCP joint just missed significance for the main effect of time,  $F(1, 12) = 3.883, p = .072$ . The main effect of group was not found to be significant,  $F(2, 12) = 1.162, p = .346$ . The interaction effect of group and time were not found to be significant,  $F(2, 12) = 1.162, p = .346$ .

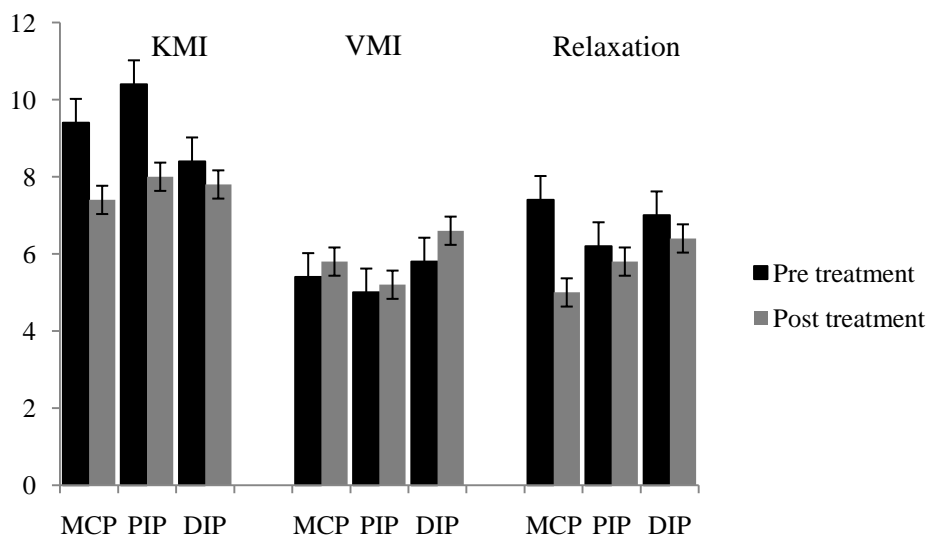
**PIP.**

The number of MUs for the PIP joint showed no significant main effect for time  $F(1, 12) = 1.798, p = .205$ . However, there was a significant main effect for group,  $F(2, 12) = 3.765, p = .054$ . Post hoc testing showed that the KMI group had a significantly higher number of MUs. There was, however, no significant interaction effect for time and group,  $F(2, 12) = 1.479, p = .267$ .

**DIP.**

The number of MUs for the DIP joint did not show a significant main effect for time,  $F(1, 12) = .037, p = .851$ , nor for group,  $F(2, 12) = 0.601, p = .564$ . Neither was there a significant difference for interaction of time and group,  $F(2, 12) = 0.45, p = .648$ .

The mean number of MUs before and after treatment for MCP, PIP and DIP for each group is shown in figure 3:9.



*Figure 3:9.* Mean number of movement units during approach phase for each group. Error bars show standard error.

### **3.8 Discussion**

#### **3.8.1 Restatement of hypotheses.**

It was hypothesized that practice with MI would be associated with a stronger negative correlation between the mean MCP and PIP and DIP joint paths during the holding phase of the movement. This primary hypothesis was partially supported, in that the MCP and DIP joint paths during the holding phase, which did not show a statistically significant negative correlation prior to VMI training, showed a significant negative correlation after training. The secondary hypothesis, that only KMI would be more likely to produce a stronger negative correlation, was not, however, supported.

It had also been hypothesized that the mean extension of the PIP and DIP joints during the holding phase of the movement would be greater following MI training, while the MCP joint angle would actually be smaller as the joint increased in flexion. This pattern of extension at the PIP and DIP, with flexion at the MCP would be typical of the lumbrical action. Secondly, it was hypothesized that KMI would be more effective in promoting this pattern. Neither of these hypotheses were, however, supported.

Two measures of endpoint variability were calculated. These were the SDs of the MCP, PIP and DIP joints and the SDs of the velocity of these joints, both measures taken during the holding phase of the movement. It was hypothesized that MI training would be linked to a reduction in the magnitude of the SDs for path and for velocity, as a result of a reduction of endpoint variability. As before, the secondary hypothesis was that KMI would exert a stronger effect, and lead to a greater reduction of SD magnitude. Neither the primary nor the secondary hypotheses were supported for these dependent variables.

Finally, it was predicted that there would be a reduction in MUs during the approach phase of the movement following MI training. Again, a secondary hypothesis was made that KMI would be linked to a higher reduction in MUs than VMI. These hypotheses were not, however, supported by the results.

The following discussion looks at each of these hypotheses in turn. Firstly, the correlation of the MCP, PIP and DIP joints will be examined, followed by the magnitude of the angles, the end-point variability and the MUs on approach to the



object.

### **3.8.2 Correlation of MCP and DIP joint paths.**

The finding of significant negative correlation of the MCP and DIP joint paths for the holding phase, following VMI training, points to improved inter-joint coordination in line with the constraints of the lumbrical grip. The KMI group showed a non-significant negative correlation prior to treatment for PIP, DIP and MCP, but the correlation became positive following treatment, and remained non-significant. The relaxation group showed a significant negative correlation for MCP and DIP before treatment; following treatment the negativity was maintained, but the correlation was non-significant.

The significant post-treatment result for the VMI group is made more interesting by the fact that this group included participant nine, the most impaired of the fifteen patients. He was one of only two in the whole sample to score zero for the Advanced Hand Activities section of the Motor Assessment Scale and also scored four on the Ashworth scale, indicating that he was one of the people with the highest level of hypertonicity. It might have been predicted that this individual's performance would have affected the group mean, leading to a poorer performance on the lumbrical grip task, but this was not the case. One explanation might be this patient's high scores on the KVIQ, where he had the fifth highest score for the visual component and the highest score for the kinaesthetic component. This may indicate that he was able to perform MI effectively.

The secondary hypothesis, that KMI would be more linked to a strong negative correlation between the finger joints, was not, however, supported. This hypothesis had been made on the basis of the work of Rodrigues et al. (2010), who found that KMI produced higher levels of postural displacement than VMI, suggesting that KMI might have a stronger effect on task performance. It may be important here to consider the nature of the training task in the current research in more detail. The first point is that the performance of the lumbrical grip task from a seated position was unlikely to have involved any major postural perturbation. The findings of Rodrigues et al. (2010) regarding the effects of KMI on postural displacement may therefore have limited applicability to this experiment. A second factor to take into account concerns the type

of motor skill involved in the lumbrical grip task. The reproduction of the lumbrical grasp might be classified as a closed skill; that is, one which involves the replication of a pre-specified motor pattern within a predictable environment (Guillot et al., 2004; Schmidt & Lee, 2005). Arguably, therefore, the key factor in the “constraint hierarchy” (Meulenbroek et al., 2001, p.137) of motor planning would then have been the pre-specified postural form of the hand as it grasped the object, a form which had been presented visually via a video. It is possible that a visual modality may therefore have been more suitable for training on this task. This is explored further below.

### **3.8.3 Possible advantages of VMI for healthy individuals and those with stroke.**

It has already been shown in the introduction that Féry (2003) was able to show some evidence that individuals who used VMI performed better on a task which involved reproducing an accurate representation of a visually presented form, when compared with those who had used KMI. Other studies have demonstrated, in addition, that external visual imagery, as was used in the current experiment, may be advantageous when the trained action focuses on reproducing a pre-specified movement pattern, as in the lumbrical grip task. White and Hardy (1995), for example, found that a group using external visual imagery showed significantly higher memory and retention scores for a series of gymnastics moves than a group who used internal visual imagery. Hardy and Callow (1999), meanwhile, reported an experiment which found that karate players scored significantly higher when learning a new karate form using external visual imagery. In both of these sports the emphasis is on the exacting replication of a pre-determined sequence of moves with a highly stereotyped form. White and Hardy (1995, p. 171) have, furthermore, drawn attention to the concept of the “image of the act”, referring to the imaging of a stable mental representation of the form of a desired movement. It may be that VMI helps to fix such an image of the act, and in the case of the present research acted as a useful adjunct to the physical training of hand lumbrical activation. It may be argued, therefore, that in closed motor skill tasks which emphasize reproduction of a postural form, such as the task in the present experiment, VMI is a more appropriate form of mental training.

It could be countered that the studies referenced in the above paragraph were

all of healthy volunteers, and that the findings therefore have limited applicability to people with stroke. However, Malouin et al. (2004) have shown that, in a test of imagery ability, stroke patients scored significantly higher for VMI than KMI. This pattern was not found in a healthy control group, which showed no significant differences in its ability using either modality. Furthermore, in Malouin et al.'s (2004) study, the stroke group's mean score for VMI was actually higher than the mean score for VMI in the healthy group; the mean KMI score, however, was lower in the stroke group than the healthy controls. In addition, the healthy group showed a significant correlation between their scores for VMI ability and their scores for KMI ability, whereas the stroke group did not show any correlation between these two scores. Thus, the stroke group scored better for VMI, and in addition their score did not seem to be related to general imagery ability, rather it seemed that they had an active advantage for using the visual modality. Furthermore, the stroke patients in Malouin et al.'s (2004) study showed a stronger, and highly significant, correlation between their scores on a test of visual-spatial working memory and their level of motor recovery. The correlations between motor recovery and verbal and kinaesthetic memory were also significant, though not to the same extent (Malouin et al., 2004). The authors proposed that their stroke group worked better with VMI than KMI, and that they further benefited if they had a lower level of impairment in visual-spatial working memory, leading to greater motor recovery.

A further factor that might be considered is that visual imagery may be a more readily accessible form of imagery in the first place. Certainly, one of the themes of the introduction to this chapter was that, while authors have been able to demonstrate clear evidence of retinotopic activity in BA 17 and 18 during visual imagery (Guillot et al., 2009; Klein et al., 2004; Kosslyn et al., 1999; Kosslyn et al., 2006), it has been more difficult to present unambiguous evidence that KMI is linked to unique activation of cerebral motor regions (Guillot et al., 2009). It may be important to take into account, in addition, that, while the neural mechanisms of vision are "relatively well understood" (Kosslyn et al., 2006, p. 135), the precise relationship between the elemental physiology of movement and its neural co-ordination remain uncertain (Jeannerod, 1997; Latash, 2008). This may provide some explanation as to why it has

proved challenging to identify unique KMI-linked brain regions. It may also mean that KMI is perhaps more difficult to generate in a clearly localizable way than VMI, even in the healthy brain, and would arguably be even more challenging for a stroke patient with cerebral motor region damage. This may provide an additional explanation for the significantly improved joint correlations noted for the VMI group alone in the present research.

### **3.8.4 Relationship between lesion site and imagery ability.**

Malouin et al. (2004) maintain that the ability to produce different categories of mental imagery may be related to lesion site. This would certainly fit with theoretical approaches, from the early work of Bastian (1887) to the contemporary work of Jeannerod (2006), which stress that visual or motor imaging involves the activation of brain regions which would be normally be involved in the act of perception, or in action planning and execution. From this perspective it would therefore be the case that damage to a motor region may lead to difficulty in imaging a motor act, while damage to a visual region would lead to impaired ability to produce visual imagery.

Unlike Malouin et al.'s (2004) study, the present research had the advantage of providing some data regarding the patient's lesion sites. As table 3:5 shows, the VMI group did not have lesions affecting any primary or secondary visual areas. Neither did they show any evidence of visual-spatial deficit at baseline. Lesion data was available for only four members of the KMI group. However, table 3:5 shows that three of the group had lesions affecting motor regions. Even for participant number one in this group, whose report results were not so clear, and number four, whose radiology report was unavailable, the loss of power and dexterity in the affected hand would point to a lesion in a cerebral motor region. The same may be said for the relaxation group. It may be the case, therefore, in line with Jeannerod's (2006) hypothesis of motor simulation, that the KMI group had difficulty generating an image based on the sensation of the lumbrical action, due to damage in their motor regions. The VMI group, however, may still have been able to produce a visual image of the postural form of the lumbrical grip, as their visual cortices remained intact. Thus, not only might VMI have been more suited to learning the task used in this experiment, as

argued above, but the patients may have had a greater capacity to work with this imagery modality in the first place.

### **3.8.5 Movement time (MT).**

It was found that the mean MT, measured for the whole grasp, was significantly shorter following KMI training, and that this was not the case for the VMI or the relaxation group. Furthermore, the initial overall MTs and the final overall MTs before and after KMI treatment were collapsed into single data sets and compared for significant difference. It was found that there was no significant difference between these two sets of data. This suggests that the faster MT following KMI treatment was not merely an effect of practice, but was indeed related to the use of KMI.

These findings therefore help confirm that already outlined for experiment one, that MT seemed to be faster when using KMI. This also seems to confirm the results of Féry (2003), who found that, in a healthy group of participants, KMI was linked to significant improvement on variables linked to the timing of a movement, while VMI was associated with significant improvement on the reproduction of the form of a pattern. Féry (2003) commented that a possible reason for these findings was that movement timing was likely to be dependent upon the bodily perception of relationships of mass and force. As such, he suggested, KMI was likely to be a more useful imagery strategy.

MT was also calculated for the approach phase of the movement. Here, a faster MT for one of the groups following treatment may have indicated a more specific effect during the planning phase of the movement. However, no significant differences were found for any of the groups. This reinforces the point already made in chapter two that the effect of KMI may have been at a very general level. It does not, however, seem to have had any influence upon what would be a component of the movement which would presumably involve higher level planning mechanisms.

One issue here, of course, is the fact that KMI seemed to have a significant effect on overall MT but not any of the other dependent variables. It was argued above that this may be explained by the damage to motor regions experienced by the patients following stroke making it more difficult to generate a kinaesthetic image of the

movement. At first sight the finding that KMI promoted a faster MT seems incompatible with this. It will be recalled, however, that the timing of the movement was not addressed in the KMI training script. Furthermore, the focus of the KMI training was on the central segment of the movement, the point at which the hand actually grasped the CD case. However, the measurement of overall MT was taken from the start to the end of the whole reaching and grasping movement. These factors may point to a far more diffuse and general effect of KMI. It is possible that the patients could still have utilized KMI in this way, despite not being able to generate a more fine-grained kinaesthetic image of the lumbrical grasp. Overall, the findings do seem to confirm Féry's (2003) finding that visual and kinaesthetic modalities of imagery may be useful for different kinds of training.

### **3.8.6 Disruption to MI in stroke.**

It was suggested above that the patients in the KMI group may have not been able to produce clear kinaesthetic imagery as a result of damage to motor areas of the cortex. Experiments using mental chronometry seem to support this analysis, suggesting that cortical damage may produce deficits in imagery linked to the functions of the affected region. Sirigu et al. (1996), for example, compared patients with parietal lesions and healthy controls on their ability to accurately estimate hand movement times from imagined movements, when compared with their time to execute those movements. The patients' mental estimates did not match the execution times, while those of the healthy controls did. Sirigu et al. (1996) also noted that the inaccuracies were effector-specific, in that the patients' mental estimates of movement times for more proximal joints matched their executed times closely. This suggested that the disruptive effects of lesions to the parietal cortex on imaging movement times for the hand may well have been linked to this region's known role in modulating the visual-motor transformations involved in grasping (Castiello, 2005; Jeannerod, 1997). In addition, Stinear et al. (2007) found that patients with lesions in the right primary motor cortex showed mental chronometry which diverged from the patterns noted for healthy individuals or those with left hemisphere lesions. If it is the case that KMI is more closely linked to activation of motor regions than VMI, as researchers have suggested (Guillot et al., 2009; Stinear et al., 2007; Rodrigues et al., 2010), then it may

also be reasonable to assume that lesions in these areas would lead to a deficit in KMI ability. Conversely, patients with an intact visual cortex may not be impeded when using VMI. This may help to explain the advantage noted for VMI in the present study for the more specific effects of joint interaction and the far more general, non-specific effects on overall movement time noted for KMI.

The application of virtual lesions using TMS has also provided evidence that the condition of the cortex may affect imaging ability. Ganis, Keenan, Kosslyn, and Pascual-Leone (2000), for example, found that magnetic stimulation of the hand area of M1 slowed response times in a mental rotation task, which is thought to be underpinned by visual mental imagery (Kosslyn et al., 2006). In addition, the slowing was not so marked when the subjects mentally rotated the image of a foot, pointing to specific involvement of the hand region of M1 in the imagery process (Ganis et al. 2000).

TMS has also been used to assess the ability of KMI to provoke MEPs, which are taken as one measure of cortical excitability (Kamen & Gabriel, 2010). Battaglia et al. (2006), for example, looked at the effects of KMI of a sequence of hand movements on increasing M1 excitability in patients with cerebellar lesions. They found that in healthy controls, the effect of KMI was facilitatory for both hemispheres, as it was for the motor region ipsilateral to the lesioned cerebellar hemisphere in the patients. However, the excitatory effect of KMI on M1 contralateral to the cerebellar damage was significantly lower than either the healthy group or the ipsilesional motor region. Again, this suggests that a neural lesion may actually interfere with the ability to work effectively with MI.

Stinear, Fleming, Barber, and Byblow, (2007) carried out an experiment on twelve people with stroke who had M1 lesions, six with right hemisphere and six with left hemisphere damage, alongside eight healthy controls. They found that KMI of a right thumb movement during TMS of M1 was linked to raised MEPs in the unaffected right abductor pollicis brevis (APB) of the left hemisphere patients. APB MEP levels in the hemiplegic hand were not, however, affected. In addition, KMI of the right hand did not make any difference to the MEPs in either hand for the right hemisphere group (Stinear et al., 2007). Furthermore, KMI of both hands led to raised

MEP levels in the APB of the intact hand only for the left hemisphere patients and did not affect either hand of the right hemisphere patients. This study therefore suggests that the efficacy of KMI may be affected both by the lesion laterality and also by the presence of hemiplegia.

Other researchers, working with the mental chronometry paradigm, have also noted that, while mental MT estimates of neurological patients may match actual MTs, they may still reflect their impaired movement capabilities. González, Rodríguez, Ramirez, and Sabaté (2005), for example, found that KMI and execution of a finger sequence were linked to significantly slower mental and executed MTs in patients with cerebellar lesions than in healthy controls. Sabaté, González, and Rodríguez, (2007), meanwhile, compared KMI and actual MTs in healthy individuals and persons with different neurological and skeletal pathologies (Sabaté et al., 2007). These authors found that real MTs were slower for all of the patients, compared with the controls, and that the patients' executed and imagined times correlated strongly.

fMRI has also revealed altered patterns of cortical activation during MI in stroke patients, when compared with healthy individuals. Sharma et al. (2009) used a finger movement task, followed by first person MI of the task. The authors are not explicit about the sensory modality of the imagery employed, however it may be inferred from their description that it was kinaesthetic. It was found that KMI for the stroke group was linked to less lateralized activity and involved motor regions of both hemispheres.

Another study by Sharma, Baron, and Rowe (2009a) focused on the connections between cerebral regions following sub-cortical stroke and in healthy controls. During KMI the stroke group showed increased connectivity between the left (ipsilesional) pre-frontal cortex (PFC) and left pre-motor cortex (PMC), and the left PFC and SMA, when compared with the controls. They also showed significantly decreased connectivity between the left SMA and left PMC. The key point is that, although displaying comparable regional activation levels during MI, the stroke patients showed altered patterns of connectivity within the frontal cortex.

In summary, it may be the case that KMI is both underpinned by the activation of motor regions, and, as suggested by González et al. (2005) and Sabaté et al. (2007),



is also constrained by the conditions of the musculoskeletal apparatus. It would seem unlikely, then, that an individual could successfully produce a fine grained kinaesthetic image of a movement in the context of a damaged cerebral motor system. Furthermore, they may also have difficulty using KMI to image a movement which they could not, in reality, perform. In the present study it may therefore have been the case that the KMI group patients were simply not able to produce an adequate kinaesthetic image of the lumbrical grip. However, the fact that their visual cortices remained intact might have meant that they could still generate a visual image of the target hand posture. Furthermore, it could still be the case that KMI may have had a very diffuse and non-specific effect on the tasks performance, leading to a faster gross MT.

The evidence presented here suggests that stroke patients with damage to motor regions may have difficulty employing MI as a viable training strategy. It may be useful here to briefly discuss emerging hypotheses which help to conceptualize such difficulties. This is done below.

### **3.8.7 Theories of impaired imagery ability in stroke.**

González et al. (2005) have proposed that the cerebellum may be part of a monitoring system which detects changes in the physical capacity of the individual and adjusts the internal representation of movements accordingly. Thus, a slower executed MT will result in slower mental MT. Sabaté et al. (2007) developed this notion, suggesting that pre-movement planning mechanisms may adapt to the post-lesion conditions of the motor apparatus, and that this is reflected in the mechanisms of MI. In so far as KMI can be considered as part of the pre-movement planning system, it would therefore be likely that it is adjusted to fit the conditions in the intrinsic muscles, rather than vice versa, as was predicted in the original hypothesis of the present research.

Sharma et al. (2006, p. 1943), meanwhile, have coined the term “chaotic motor imagery” to describe a condition in which persons suffering from stroke may have impaired acuity or timing of imagery. If these authors (González et al., 2005; Sabaté et al., 2007; Sharma et al., 2006) are correct, it may be reasonable to suppose that these processes would severely limit the effectiveness of KMI in a situation in which

there is damage to motor regions. This may help explain the failure of KMI to promote improved correlation between the finger joints of interest in the present research.

### **3.8.8 Physiological considerations regarding MI and the lumbrical grip.**

One outstanding question is why, given the significant negative correlation noted for the MCP and DIP joints following VMI treatment, the same pattern was not observed for the MCP and PIP joints? This requires further explanation, as the lumbrical action is marked by simultaneous flexion of the MCP and extension of the PIP and DIP joints and one may therefore have predicted that the same effects would be noted at the PIP joint (Palastanga et al., 2006).

It may be useful here to recall the physiology of some of the muscles involved in the lumbrical grip. Firstly, the lumbricals themselves have their point of insertion predominantly in the distal phalangeal section of the dorsal digital expansion, with far fewer fibres having a middle phalangeal insertion (Palastanga et al., 2006). It may be the case, therefore, that increased activation of the lumbricals would exert a stronger effect at the DIP joint, producing more extension at this joint than at the PIP. Table 3:7, which shows the mean extension of the joints during the holding phase, seems to confirm this. It will be noted that the mean extension of the DIP joint following VMI treatment was around 17° greater than for the PIP joint. Indeed, the extension of the DIP joint was greater than that of the PIP for all of the groups in all conditions.

It is important, in addition, to take into account the action of the extrinsic hand muscles that form part of the lumbricals' working environment. The dorsal digital expansion, for example, is formed from the distal portion of the extrinsic extensor digitorum muscle (Palastanga et al., 2006). This muscle's main function is the extension of the MCP joint (Palastanga et al., 2006) and, as Long (1968) pointed out, it may not be strong enough, acting on its own, to extend the PIP and DIP joints against the powerful pull into flexion exercised by the flexor digitorum profundus (FDP). According to Long (1968) the key role of the lumbricals is, therefore, to work against FDP, in order to facilitate extension in the interphalangeal joints. In hemiplegia, it has been found in the upper limb that, while the relative loss of strength in the finger extensors may be less than that in the finger flexor group, the absolute power of the extensors is still lower (Colebatch & Gandevia, 1989). Therefore, where

the lumbrical muscles cannot work to counteract the action of the flexors, the hand may adopt the characteristic flexed posture seen following neurological damage (Bobath, 1978; Carr & Shepherd, 1998; Champion, Barber, & Lynch-Ellerington, 2009; Davies, 1985).

It has been argued here that the force exerted by the lumbricals on the PIP joint may not be as great as that on the DIP joint. If that were the case, then the force of the extrinsic flexors, which would have been co-activated with the lumbricals during the holding phase (Long, 1968), may have been harder to resist at the PIP joint, resulting in a more marked flexor pattern for that joint. This would then explain the lack of a significant negative correlation between that joint and the MCP, as was found for the DIP and MCP.

It is also important to take into account a further factor which might work strongly against the lumbrical at the MCP. This concerns the flexor digitorum superficialis (FDS) and the flexor digitorum profundus (FDP). FDS is a powerful two-headed muscle which inserts into the volar aspect of the middle phalanx, exerting its force on the PIP joint (Palastanga et al, 2006). FDP is a more slender single-headed muscle which inserts into the distal phalanx, and whose main function is the flexion of DIP (Palastanga et al., 2006). Since FDP is a weaker muscle, the pull into flexion may not have been felt as strongly at DIP when compared with PIP, which was being acted upon by the stronger FDS. This may further explain the co-ordinated contraction pattern in keeping with the lumbrical action at MCP-DIP, but not at MCP-PIP following VMI treatment.

It was also pointed out above, that the relaxation group showed a statistically significant negative correlation for the MCP and DIP joint paths prior to listening to the guided relaxation script, but that following relaxation the correlation was no longer significant. This would suggest that this group was able to partially produce a lumbrical grip following physical training and watching the video explaining the grip. The same limitations noted for the VMI group regarding the PIP joint described above also seem to have applied in this case. However, it may be the case that the relaxation task then disrupted this. One possibility is that a degree of co-contraction, for example at extensor digitorum and the lumbricals, had been necessary to achieve the pattern in

the first place. Relaxation may then have made this more difficult to achieve when tested again post-treatment.

The following section contains a discussion of the other measured dependent variables in the study. Following this a number of methodological issues are examined. Some theoretical implications of the research are then discussed.

### **3.8.9 MCP, PIP and DIP joint path.**

None of the treatments were found to be linked to a significant difference for the mean DIP and PIP extension, or the MCP flexion during the holding phase of the grasp. At first sight, this finding seems at odds with the finding of a statistically significant negative correlation for the MCP and DIP joints following VMI treatment. The key question is why significant differences were not also noted for MCP and DIP joint magnitudes following VMI treatment? The explanation may lie in the fact that this variable was a measure of isolated joint angles. This measurement may not have been adequate to capture the precise way in which the effect of VMI worked. This can be understood at a biomechanical, physiological or motor theoretic level. Each of these is discussed in turn below.

Biomechanically, the MCP, PIP and DIP joints can be thought of as a serial kinematic chain, temporarily closed by the object being grasped during the holding phase (Zatsiorsky, 1998). According to biomechanical theory these linked joints can be conceived of as acting as a unit in a “functional symmetry”, guided externally by the constraints of the instruction to perform the lumbrical grip (Zatsiorsky, 1998, p.112). From this perspective, it is likely that any effect of MI training would not have been apparent at the level of the isolated joint, but at the level of the interrelationship between the joints.

At the physiological level, it has already been shown how the lumbrical muscle acts at all three joints simultaneously. Indeed, the uniqueness of this muscle lies in its ability to produce flexion at the MCP and extension at the interphalangeal joints in a single contraction (Palastanga et al., 2006). From this point of view, therefore, it is also unlikely that, if VMI were targeted at activation of the lumbrical muscle, the effect would be noted at the level of an individual joint.

In motor theoretic terms it may also be the case that MI would not be found to

act by increasing or decreasing the size of isolated joint angles. This is because MI, as suggested by Jeannerod (2006), may share the same mechanisms as pre-movement motor planning. If this is the case, then it is likely to operate by specifying the patterns of interactions between joints, rather than the activation levels of individual muscles. Motor schema theory, (Schmidt, 1975) and generalized motor program theories (Schmidt, Zelaznik, Hawkins, Frank, & Quinn, 1979; Schmidt & Lee, 2005) predict that the motor program would encode invariant movement patterns, and that variables such as the relative forces of muscles in the synergy would be determined centrally by the program. The absolute activation levels of the separate muscles and the absolute magnitude of individual joint angles would then be parameters which were subordinated to this general program. From this perspective, it might be also predicted that the influence of MI training would be exerted at the level of the pattern of joint interaction, rather than individual joint angles.

#### **3.8.10 Movement variability in stroke.**

Variability in the movement profiles of the hemiplegic upper limb in stroke patients has been found to be higher than that of the less affected upper limb (Levin, 1996) and than that of healthy individuals (Cirstea et al., 2003). Certainly, in the present study, high levels of variability were noted across all of the movement profiles and treatment conditions for each group, as can be seen in tables 3:7 and 3:8 and also graphically for the PIP joint of the KMI group prior to treatment in figure 3:3. This may be seen as a physiological problem but might also be analyzed in relation to putative motor control processes. Some possible physiological explanations for this increase variability are discussed below. This is then followed by a motor theoretic account of movement variability. Following on from this, the outcomes on the variables in the current experiment which were used to examine motor variability are discussed.

#### **3.8.11 Physiological explanations for post stroke movement variability.**

There may be a number of physiological explanations for the increased variability of movements in the hand following stroke. Post-stroke tremor specifically affecting the hand has been noted as a fairly widespread problem and has been linked specifically to lesions in sensory and motor areas (Alarcón et al., 2004; Ferbert &

Gerwig, 1993; Kim, 1992; Kim & Lee, 1994). In addition, tremor may not be detectable visually, as one study found that it was only made apparent on EMG readings (Ferber & Gerwig, 1993).

A further feature of the post-stroke syndrome is an altered pattern of reciprocal innervation (Sherrington, 1947; Gjelsvik, 2008). This may lead to difficulties coordinating the agonist-antagonist balance when activating the fingers, leading to a more variable movement path for the joint. There may be altered patterns of descending control, axonal sprouting, damage to the recurrent inhibition of the Renshaw cell system, disruption of the Ia inhibitory interneuron and the emergence of plateau potentials, which might all lead to an increase in neuronal firing (Gjelsvik, 2008; Gracies, 2005; Gracies 2005a; Pearson & Gordon, 2000). There may also be a down regulating of inhibitory neurotransmitter receptors for several weeks following neurological injury (Nudo, 1999). Furthermore, peripheral changes in the structure of the muscle can increase stiffness and make smooth movements more difficult to achieve for the patient (Gracies, 2005). Finally, altered patterns of descending neural control may lead to less finely graded movements, particularly in the fingers (Gracies, 2005).

Cicinelli et al. (2006) have highlighted the issue of hyperexcitability in the motor cortex following stroke. These authors suggest that, as there is reduced sensory feedback from less active muscles following the initiation of a motor program, then the “gain” of the motor mechanisms has to be increased, leading to increased excitability (Cicinelli et al., 2006, p. 252). Furthermore, they argue that damage to the primary motor cortex (M1) may lead to the recruitment of secondary motor regions which have fewer and less sensitive connections at the level of the spine than M1. The increased excitability may also, therefore, be an adaptive response to the problem of generating an effective motor command through the descending control systems. It is reasonable to suppose that the high levels of variability in joint path, velocity and acceleration noted in all patients who took part in the present study may have been a result of any one or a combination of these factors.

**3.8.12 Standard deviations (SDs) of MCP, PIP and DIP joint paths and velocity.**

The present study took the kinematic variable of the SDs of the angular paths of MCP, PIP and DIP joints as one measure of variability. In addition, a second kinematic variable which measured variability was the SD of velocity of these joints. Both variables were measured for the holding phase of the grasping movement only, as this was taken as the endpoint of the movement. These measures thus provided a plausible picture of the endpoint variability of the grasping movement. However, no significant differences were found following any of the mental training tasks. Some possible explanations for this are explored below.

Firstly, it will be recalled that significant results which affected specific aspects of the production of the lumbrical grip had been previously found only when using VMI. Since the reduction in endpoint variability was likely to involve the modulation of the degree of force used in the resting hand posture, then it might be the case that KMI was more suited to such a task. However, as explained above, cortical damage to motor areas may have restricted the use of KMI for these patients, making the modulation of force levels using MI more difficult.

A further point is that a reduction in endpoint variability was not specified in the MI tasks. It was presumed that any improvement in motor control which might be reflected in a reduction in endpoint variability, would take place at a non-conscious level. It is not clear whether this assumption was warranted, and it may therefore be the case that future research should explicitly include an instruction regarding variability in the MI training. It may also be the case that there were aspects of the physical performance of the task and the MI task which may have been contributing to the variability of the endpoint measures. This point requires further explanation and is considered firstly from the neurophysiological perspective and then from the standpoint of motor theory.

### **3.8.13 Increase in sense of effort and movement variability.**

Evidence suggests that individuals with paralysis or altered sensory feedback in the upper limb may use extra effort to accomplish a motor task, and that their sense of effort is then used as a reference by the motor control system (Gandevia & McCloskey, 1977) Subjective accounts of stroke have consistently stressed the exaggerated voluntary effort needed to contract a paralyzed muscle (Brodal, 1973;

Jeannerod, 1988). Bertrand, Mercier, Shun, Bourbonnais, and Desrosiers, (2003) and Simon, Kelly, and Ferris (2009) have also presented evidence suggesting that, as a result, individuals with stroke may have difficulty gauging the amount of force needed to perform a task.

It may be the case that the participants in the present research experienced a heightened sense of effort on attempting to complete the task by reaching and grasping repeatedly with the hemiplegic limb. This would have been because of the greater perceived level of force needed to move the limb. In addition, it may have actually been further exaggerated by the concentration demanded in the MI training. This increased effort would presumably be accompanied by augmented central neuronal drive. Schmidt et al. (1979) and Harris and Wolpert (1998) have both argued that such increased drive can lead to increased variability in the path and endpoint of an upper limb movement. It may be possible, therefore, that the MI task and the repeated reaching and grasping movements may have actually led to an increase in variability in the finger joints as the hand grasped the object. If this were the case, it seems unlikely that MI could have acted to reduce endpoint variability.

#### **3.8.14 Number of movement units (MUs) during approach.**

In the current study the number of MUs for the MCP, PIP and DIP joints during the approach phase of the movement was taken as a plausible measure of the balance of feedback and feed-forward error correction as the hand approached the object to prior to grasping. It was hypothesized, in line with Lin et al., (2009), that more efficient motor control would be distinguished by a higher level of feed-forward guidance. It was predicted, therefore, that MI treatment would be associated with a reduced number of MUs, as there would be less shifting between the two forms of error correction and more reliance on feed forward control, allowing for a smoother movement trajectory.

No significant differences were found between the MUs following treatment in any of the conditions. These results are the same as for experiment one. It is interesting to note that Lin et al. (2007) also failed to find any significant differences for the number of MUs following treatment with constraint induced movement therapy. The theoretical background behind the choice of this kinematic variable has



already been discussed in chapter two. It might be the case, however, that this kinematic variable is not a valid measure of the control strategies being used in a reaching movement. The appropriateness of the use of MUs as an index of motor control strategy in a study of this sort therefore remains an open question.

### **3.9 Methodological Issues Raised by Experiment Two**

This study has also raised a number of methodological issues in relation to researching hand movements in adult hemiplegia. These are discussed below.

#### **3.9.1 Measuring movement phases.**

The challenges posed when trying to identify the start and end points of the grasping movement in this study were similar to those already outlined for experiment one. However, it was still important to identify a reliable way in which to determine a single movement start and endpoint. In experiment one this was done by visually identifying the start and end points of the movement from the images of the markers on the computer screen. A different approach was adopted for the present experiment. This is explained below.

Reference was made to literature examining the onset and endpoint of the EMG signal, which has also been described as “notoriously noisy” (Latash, 2008, p. 142). Walter (1984) pointed to the problems inherent in applying a single computer algorithm to such data. He maintained that the start and end of the movement are rarely abrupt and that it is likely that the researcher will need to resort to visual inspection of the signal as an adjunct to computerized detection. The use of a standard algorithm would then lead to a misleading impression of objectivity for a process in which there was in fact substantial human input (Walter, 1984).

Walter (1984) proposed, instead, an interactive method which involves the researcher firstly identifying visually the topology of a representative waveform for the movement. The computer is then used to identify the temporal sites for the landmarks of interest which define this waveform, such as initiation and termination points. Kamen and Gabriel (2010), also writing with reference to detecting the initiation of a movement via the EMG signal, comment that “ironically... manual detection remains the gold standard against which new algorithms are tested” (p. 113).

The interactive method outlined by Walter (1984) and Kamen and Gabriel

(2010), derived as it is from the exigency of working from highly variable data, was considered to be applicable to the identification of start and end points of hand movement in the present research. The exact method has already been described in the methodology section of this chapter. Below, a number of possible methodological objections are dealt with.

First, it might be objected that measuring the start of the hand movement from the PIP joint of the index finger was unwarranted. For example, measurements were also available of the thumb, the grip aperture and the wrist, and any of these movements could have been taken as a start point. However, Wing and Fraser (1983) have shown that the movement of the index finger is the key factor in shaping and guiding the overall grasp. As the focus of this study was on the grasp itself, rather than the transport phase of the movement (Jeannerod, 1988), it was felt that the PIP joint therefore provided an appropriate reference point from which to define the start and end of each movement grasping cycle.

A further objection might be that the identified start and end points, at velocity values of  $\pm 10$  deg/s, were based on an arbitrary figure. However, the very high levels of variability in the movement meant that it was not possible to identify zero velocity points at which the index finger was completely still. It was felt that the kinematic landmark of  $\pm 10$  deg/s did, however, provide a reasonable approximation to such a point. Furthermore, other published studies that have measured upper and lower limb movements in a stroke population may be open to similar criticism. Levin (1996), for example, identified a start and end point as 10% of peak shoulder velocity, Cirstea et al. (2003) also used a cut-off of 10% of peak velocity, and Menegoni et al. (2008) suggested a cut-off velocity of 50mm/s. However, none of these authors provided further empirical or theoretical justification of these measurements. Hansen, Elliot, and Khan (2007), in a study of healthy individuals, identified movements with a finger velocity of 10 mm/s for more than 20 ms continuously as the start point of the movement; again, no further justification was provided. It would seem, then, that other studies in this area might be charged with using arbitrary measures to identify start and end points of movement. The present study, however, which focused on far more complex rotatory finger movements in hemiplegia, had the advantage of

providing a clear theoretical justification for the kinematic landmarks chosen.

### **3.9.2 Sample size.**

As stated in the introduction, the lack of previous research in this area made the calculation of an optimal sample size difficult. Published studies of MI in the rehabilitation of the hemiplegic upper limb generally have used samples of thirteen (Page et al., 2001), eleven (Page et al., 2005) and thirty two patients (Page et al., 2007). It was felt, therefore, that, given the time and resource limitations of this study, a sample size of fifteen was appropriate. A larger sample size is always desirable, of course, and should be aimed at in future work.

### **3.9.3 Limitations of sampling process.**

As can be seen from the discussion of the recruitment procedure the sampling technique for experiment two was the same as that for experiment one. It was also therefore a non-probabilistic convenience sample. The limitations of such a sample are the same as those already described for experiment one in chapter two.

### **3.9.4 Limitations of physical treatment protocol.**

For experiment two many of the same limitations apply to the delivery of the physical therapy protocol as have been highlighted for experiment one. As explained with regard to the earlier study, the application of a standardised treatment programme may not be appropriate as all patients will respond differently to physical treatment.

The timing of the interventions was also a limiting factor for experiment two. Again, the exact timing of each session was not adequately controlled, though all were between 15-20 min. Furthermore, the timing between the physical therapy sessions was not controlled with sufficient rigour. For example, in some cases two sessions were delivered on the same day, with a break of 15-25 minutes in between. Furthermore, some individuals received the final session of therapy in the laboratory; a break of one hour then took place before MI was used and motion capture undertaken. It is possible that the closer proximity of physical treatment and motion capture may have given those individuals a performance advantage. In the future, these aspects should be more carefully controlled as timing may have therefore entered the study as an extraneous variable.

### **3.9.5 Number of repetitions of the task.**

No definite protocol exists as to the optimal number of repeated movements needed to ensure that a representative ensemble averaged profile can be constructed for an upper limb movement (Kontaxis et al., 2009). Kontaxis et al. (2009) state that this decision is dependent on factors which include the task to be performed and the disability level of the actor. These authors do, however, suggest a minimum of three repetitions. Experiment one had calculated ensemble averages from ten grasps. It was felt that, given the comments of Kontaxis et al. (2009) that such a high number was not necessary for the present research. However, given the high levels of variability encountered in the upper limb movements of stroke patients (Cirstea et al., 2003; Levin, 1996), it was decided to include five repetitions in the analysis. It will be noted that, in the one case where this was not possible, that of patient nine's DIP measurements prior to treatment, the number of repetitions taken still met the necessary minimum requirements identified by Kontaxis et al. (2009).

### **3.9.6 Range and length of times since stroke used in sample.**

This was a varied sample, with a wide a range of times since stroke onset. However, motor control studies of patients suffering from hemiplegia have used samples with comparable ranges of time since stroke: in Cirstea and Levin's (2000) study the period since onset ranged from two to fifteen months; Cirstea et al.'s (2003) study used a sample whose time since stroke ranged from three to fifteen months; Levin (1996) used a sample in which the time since onset ranged from 6 months to 7.2 years; in Lin et al.'s (2007) study of constraint induced movement therapy the time since stroke ranged from 13-26 months.

Studies of MI in hemiplegia have also used widely varied samples: in Page et al.'s (2005) study, time since onset ranged from 15-48 months; in Page et al.'s (2007) experiment, the time ranged from 12-174 months; Sharma et al.'s (2009a) study used a sample whose time since stroke was from 7days -703 days; in Gonzalez et al.'s (2005) experiment the time ranged from 6-53 months. Based on previous work, therefore, it would seem that the range of times since stroke used in this study (8 days-3.5 years), should not have presented a problem.

It might also be objected that patient four in the present study, who had 3.5 years since the event, was unlikely to benefit from MI training. It is certainly the case

that post-stroke recovery has been found to level off after six months (Nudo, 1999). However, it has also been demonstrated that motor recovery following rehabilitation is still a possibility some years after the initial event (Woldag & Hummelshein, 2001). Furthermore, this seems to be supported by MI literature. Malouin et al. (2004)'s sample, for example, included a patient who had had their stroke 4.5 years previously and these researchers were still able to show a significant improvement in motor function following mental training. Page et al. (2005) demonstrated a significant improvement in stroke patients who were on average at 23.8 months post stroke. In Page et al.'s (2007) study, highly significant improvements were recorded in a sample whose average post-stroke time was 42 months, with a maximum time of 14.5 years. It does not, therefore, seem unreasonable to have included a patient who was 3.5 years post-stroke in the present study.

### **3.10 Limitations of instruments used.**

#### ***3.10.1 General points relating to psychometrics and translation of instruments.***

Hambleton and Bollwark (1991) have highlighted a number of threats to validity which can arise when a research instrument is translated from its source language for use with a population from a different cultural and linguistic background. First, the relevance of the instrument to the culture of the target population may not be clear. At the same time, the target population's response to questioning and the presence of a researcher might be different to that of the original population for whom the instrument was intended. The possible difficulties which might arise, for example, as a result of the presence of a researcher from a wealthier country in a resource poor context, have already been explored in this thesis. Researchers have proposed a number of solutions to these problems. Some of these are discussed below.

The technique of back-translation has been recommended by several authors (Hambleton & Bollwark, 1991; Sireci, Yang, Harter & Erhlich, 2006; Ros-Morente, Vilagr a-Ruiz, Rodriguez-Hansen, Wigman & Barrantes-Vidal, 2011). Sireci et al. (2006) described how this was accomplished in their own work. First, a team of translators translated the instrument into the target languages and these were then translated back into the source languages by another group of translators who had not

seen the original text. These translations were then compared with the original and any disagreements adjudicated by a designated third person. Following this process a final version was approved. Hambleton and Bollwark (1991) have suggested that a post-translation probe may also be conducted, involving a sample of the target population being asked to explain how they interpreted the translated instrument. The examiner can then make a decision about the validity of the translation. As the authors point out, however, this is a process open to subjective interpretation and may be biased by the interaction of the examiner and the participant (Hambleton & Bollwark, 1991).

It remains the case, then, that the subjective judgements involved in the translation process may undermine the validity of the instrument. Hambleton and Bollwark (1991) have pointed out, in addition, that translators may rely at times on intuitive guesses as to an appropriate translation for a word or term. This may lead to error which can then be masked by the process of back-translation. These authors therefore recommend that the back-translation method is complemented by statistical techniques. For example, individuals proficient in both languages could undergo both versions of the test some time apart and the degree of correlation of their responses tested using Pearson's  $r$ , or any differences in their responses tested with a dependent samples  $t$  test (Hambleton & Bollwark, 1991). Sireci et al. (2006), for example, wished to find out if the iterative process of translation and back-translation had improved the validity of a Likert scale and used analysis of covariance (ANCOVA), regression analysis and a dependent samples  $t$  test to test the responses. Sireci et al. (2006) found that, over time, the responses of different linguistic groups which had been matched by other demographic factors began to show fewer differences. This suggested that the validity of the instruments had indeed been improved by the process of repeated back-translation and revision.

### ***3.10.2 Limitations regarding the use of the KVIQ***

In the present research The Kinaesthetic and Visual Imagery Questionnaire (KVIQ) was used in order to assess the ease with which participants could use motor imagery. The KVIQ was originally validated on people with stroke in a Canadian context (Malouin et al., 2007). Although the research took place in Quebec, it seems that it was the English version of the KVIQ which was validated. For the purpose of

the current study the English version KVIQ was first translated into Colombian Spanish by an individual who was a native speaker who was also proficient in English. The translation was then checked by the researcher, who looked for terms which might diverge from the English source and check that the responses to the Spanish version would be the same as those required in the English KVIQ. This is the most basic type of validity check recommended by Hambleton and Bollwark (1991). A full back-translation by an independent translator and a subsequent check against the original text were not, however, carried out. This was because of limitations on time and resources, making the recruitment of an appropriate translator difficult. This is discussed further below.

The translated KVIQ had not had its validity and reliability demonstrated in a Spanish language context. It may not be certain, therefore, that the Spanish speaking participants were responding in the same way to all parts of the instrument as would an English speaking group. Hambleton and Bollwark (1991) do, however, point out that a reasonable test of validity is if the instrument includes a requirement for the examinee to produce a particular movement. If the movement is executed as expected, then this is a sign of the validity of the translated test. In the case of the initial steps in KVIQ, the respondent was required to enact some simple movements upon instruction prior to imagining them. All participants performed the actions as was expected by the researcher, indicating that this part of the KVIQ at least was reasonably valid. The next part of the KVIQ, however, required the person to imagine the movement in a kinaesthetic or visual modality. Such interior processes are, of course, hard to monitor, and present a general challenge in MI research (Neuper et al., 2005). It may be the case that the compliance of the participants with the MI process was made even more difficult to judge when the instrument was being administered in the researcher's second language, making the issue of validation even more important. Hambleton and Bollwark (1991) do point out, however, that, if an instrument makes reference mainly to etic, that is, culturally non-specific concepts and terms, it may be the case that it will also display increased cross-population reliability and validity. The KVIQ arguably tests abilities which would be evenly distributed across all human populations and, to the best of the author's knowledge, does not make explicit

reference to specific cultural phenomena. Furthermore, the language of the KVIQ is both simple and repetitive and does not require complex verbal responses, making the translation process less open to interpretation. In this sense it is hoped that the imagery component of the translated KVIQ can be regarded as reasonably valid.

### **3.10.3 Randomization issues.**

As stated in the introduction, it was not possible to randomize the final four patients recruited to the study. This can be explained with reference both to the particular needs of the participants and the procedures necessitated by the research protocol. First, issues to do with patient safety and comfort, for instance transport to and from the laboratory and the importance of giving full attention to the person before, during and after the visit, meant that the participants had to be seen separately on different days. Second, the administration of a specialised physical treatment protocol prior to the experimental treatments meant that the author, a trained occupational therapist, had to both give treatment and allocate to group. It was clear in advance, therefore, once the random allocation process had led to the KMI and VMI groups being filled, which group the remaining patients would be allocated to.

It should be stressed that all participants received identical physical treatment based on the protocol outlined in the appendices. Also, the explanation of the task was provided via a pre-recorded video in order to minimize any possible bias in the way the task was presented. In addition, the KMI, VMI and relaxation treatments were all pre-recorded and each member of the group listened to an identical recording. It is felt, therefore, that experimenter bias should not have entered the study as a confounding variable.

### **3.11 Conclusion**

It was found that VMI was linked to a significant post-treatment negative correlation between the paths of MCP and DIP joints during the holding phase of the movement, during which the grasp was actually executed. It may be the case that VMI is actually more suited to the type of training task used in this study, which emphasized achievement of a resting posture, rather than aiming at a target. KMI, meanwhile, was associated with a faster overall MT. Since this variable was not specified in the MI script, it was suggested that this points to a much more general role



for KMI, perhaps equivalent to attentional processes. It was also argued that the different outcomes that were associated with VMI and KMI may have been linked to individual's cerebral lesion site.

**Appendix 3:1.****Visual imagery task script.**

"Picture your weak hand. Imagine you are looking at it, but keep your hand quite still. Now imagine you can see your fingers moving, but do not really move your hand, keep it quite still. Imagine you can see your knuckles beginning to bend, but at the same time imagine you see your fingers staying quite straight. Imagine you can see your hand in the position you have practised in therapy and watched on the video, which is with your knuckles bent, but your fingers straight out. Careful not to really move your hand, just imagine you can see it moving."

Repeated six times

## Chapter 4

### **Is There a Raised EMG Signal During KMI of Thumb Abduction in Stroke Patients with hemiplegia affecting the hand?**

#### **4.1 Experiment Three Research Question**

Is there raised EMG signal during KMI and VMI of thumb abduction in stroke patients with hemiplegia affecting the hand?

##### **4.1.1 Experiment Three Hypotheses**

1. Individuals with hemiplegia following stroke will be more likely to show a raised EMG signal while using MI to imagine movement in abductor pollicis brevis (APB).
2. KMI is more likely to lead to a raised EMG signal in APB in people with hemiplegia following stroke.

#### **4.2 Literature Search**

##### **4.2.1 Aim and objectives of literature search for chapter four.**

The aim of the literature search for chapter four was to identify evidence that the issue of whether MI causes peripheral muscular activation is subject to debate amongst researchers. A key objective was to show that this controversy has involved discussion about the presence and role of the electromyogram (EMG) signal during motor imagery.

##### **4.2.2 Inclusion and exclusion criteria.**

As described above for chapter three this was a scoping study with broad search criteria. Only studies of adult humans were included. All studies were published in English. In addition, there was no exclusion criterion based on the presence of stroke and studies of neurologically intact individuals were also included. There were no exclusions based on type of outcome measure. Furthermore, there was no exclusion criterion based upon study methodology.

##### **4.2.3 Characteristics of included studies.**

All studies except for one employed surface EMG. The one exception used indwelling electrodes.

#### 4.2.4 Search terms used.

MeSH terms used in MEDLINE, CINAHL and PSYCinfo included: electromyography; cerebrovascular accidents; arm; hand. Free text terms included: motor image\*; stroke; upper limb.

#### 4.2.5 Search strategy employed.

An example search strategy, from Web of Science, is shown in table 4:1.

Table 4:1

*Search strategy for Web of Science (Science Citation Index Expanded) for chapter four November 2011 – January 2012*

Search terms	Search limits			Hits
	Web of Science Categories	Search type	Document type	
("Mental Image*" OR "Motor Image*") AND "Electromyography"	Neurosciences Clinical neurology Psychology Neuroimaging	Topic	Article	16
("Mental Image*" OR "Motor Image*") AND ("Electromyography") AND ("stroke" OR "cerebrovascular accident")	Neurosciences, Clinical neurology Psychology Sports sciences Behavioral science	Topic	Article	1

#### 4.2.6 Internet sites used.

The website of the European Union project of Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM) was used to obtain guidance on the placement of surface EMG sensors.

#### 4.2.7 Grey literature.

The inventory and recommendations of SENIAM were used as a guide to sensor placement. In addition, technical manuals on electromyography were extensively consulted.

#### 4.2.8 Limits of the search.

For Web of Science the search categories were extended to include journals from the field of sports science. Otherwise, the search limits were the same as those used for earlier searches.

#### **4.2.9 Critical appraisal tool.**

Only one study was identified which used a group of individuals with stroke. However, this study did not fit the definition of a clinical trial. It was therefore felt inappropriate to attempt assessment of the study using the PEDro scale.

#### **4.3 Introduction**

It is fairly well established that mental practice using MI (MI) can improve task performance, both in healthy individuals and stroke patients (see, for example, Amemiya, Ishizu, Ayabe, & Kojima, 2010; Page et al. 2007). However, there has been continuing discussion about the precise mechanisms which underpin MI. One unresolved issue involves the presence or absence of peripheral muscular activation as measured by EMG. Disagreement also exists as to the significance and function of any raised EMG signal which might be observed during MI. A brief historical introduction to this controversy is given below, followed by discussion of more recent evidence.

As long ago as 1905 James argued that, while an imagined movement must involve inhibition of motor output, this inhibition was not total. In his view, therefore, every imagined movement would involve some low level muscular activation. He does not seem, however, to have regarded this peripheral activity as being the explanation for the motor priming effect of MI. He believed that this probably occurred as a result of a sudden release of tension as the motor inhibition gave way (James, 1905).

Other early work on MI, however, stressed that the EMG signal recorded during imagery had a functional significance and aided task performance by providing proprioceptive feedback. This was labelled the 'psychoneuromuscular' (PNM) position in the 1960s by Richardson (as cited in Lutz, 2003, p. 149). One source of evidence for PNM came from the work of Jacobson (1930a; 1930b). In one experiment (Jacobson, 1930a), three healthy individuals were asked to imagine bending their arm, or to visualize bending the limb. It was found that the instruction to imagine the arm bending led to an increased electrical signal in the biceps. The command to visualize the movement on the other hand, led to increased action potentials in the ocular muscles. In another experiment, Jacobson (1930b) asked an

individual with an above elbow amputation to imagine bending his left, amputated, hand. Increased action potentials were recorded in the remaining left biceps and in the intact right hand. Jacobson concluded that “mental activity is not confined to closed circuits within the brain... neuromuscular regions participate” (1930a, p. 121).

Since the 1980s, however, PNM has been largely displaced by a theory of “central representation”, which has drawn on the hypothesis of motor simulation (Jeannerod, 2006; Mulder et al, 2004, p. 212). In this analysis MI is thought to rely on an internal mental model of the required action. This internal representation on its own can be used as a training resource and does not require peripheral activation.

In one early paper which is representative of this theoretical tradition and which has already been discussed in the introduction to this thesis, Decety et al. (1989) argued that the central nervous system mechanisms involved in movement planning and preparation were the same as those used in mental imagery of the movement. It was this overlap, these authors suggested, rather than peripheral muscular activity, which explained the effectiveness of mental imagery training. Following this, Decety and Jeannerod (1996) proposed that that the mental representation of a movement in the absence of execution should involve blocking of the motor signal at some point in the cortical-spinal system. This last point clearly distinguished their position from PNM and was to form a constituent part of the developing analysis of central representation.

As shown in the introduction to this thesis, Jeannerod (2001) went on to promulgate a more far reaching theory of simulation. This suggested that an imagined action corresponds to a covert, preparation stage of activity. Jeannerod (2001, p.106) hypothesized that the imagined action might invoke a “subliminal” activation of the motor system, but that this would be accompanied by a simultaneous inhibition of the action command. Jeannerod (2006) later put forward a more developed model of the hypothesis of motor simulation. Here, he restated that the ability to imagine a movement necessarily implied a dual process of central activation and spinal inhibition. He proposed, in addition, that any peripheral activation noted during MI would, therefore, be a result of inadequate containment of the motor signal at the brainstem or spinal stage.

A prediction which flows from Jeannerod's hypothesis is that any peripheral muscular activity noted in an MI trial is a result of a breakdown in this process of inhibition, and has no functional relationship with MI. From the central representation perspective, therefore, any heightened EMG during MI is merely an epiphenomenon of MI and would have no practical significance. Again, this stands in direct contradiction to the earlier PNM approach which saw the muscular contraction as a condition of effective MI training.

As shown in chapter one, Jeannerod's hypothesis has proven highly attractive and is widely referenced as a starting point in MI research papers (for example, Mulder et al., 2004; Li et al., 2004; Li 2007). Despite this, the PNM position has maintained some influence. As would be expected, one moot issue in the debate centres on the presence and/or significance of peripheral muscular activity in MI. This has often focused on the experimental evidence for the presence of an EMG signal which can be linked to MI. The following section contains discussion of a number of papers which touch on this issue. It will be argued that results have sometimes been contradictory and also occasionally open to conflicting interpretation.

#### **4.3.1 Evidence for EMG during MI.**

One of the most frequently cited experiments to examine the role of EMG activation in MI is that conducted by Yue and Cole (1992). The paper has generally been referenced in support of a central representation approach. Mulder, de Vries, and Zijlstra (2005, p. 345), for example, commented that Yue and Cole (1992) were "not able to show activation of peripheral systems in parallel with motor imagery", an interpretation repeated by Dickstein, Gazit-Grunwald, Plax, Dunskey, and Marcovitz, (2005), Lutz (2003), and Mulder (2007). However, closer analysis of Yue and Cole's findings with regard to EMG activation might not support this analysis. The experiment and results are discussed below.

Thirty healthy individuals were randomized to three groups. The first group exercised to produce a maximal voluntary contraction (MVC) of the hypothenar muscles of the left hand, involving abduction of the little finger. The second group practised MI of this task, while a third control group did not practise at all. Baseline EMG readings were taken from the abductor digit minimi (ADM) before and after the

training period. Force was measured at baseline using a transducer and measured again after training. EMG and force measurements were also taken for the contralateral, untrained, hand.

The authors found that for the exercise group force production increased by 29.75% for the trained hand and 14% for the contralateral hand. For the MI group force production increased by 22% for the trained hand and 10% for the untrained. In addition, the results for the MI group seemed to have been attained without any increase in muscle bulk, as electrical stimulation of the ulnar nerve following training did not lead to any increase in muscle twitch.

While the increase in force may not have involved a muscle fibre increase, the claim that EMG played no role in the MI training is harder to maintain. One point is that, although no significant increase in EMG signal was noted following MI, there was still an absolute increase of 21.73%. Yue and Cole (1992) do, however, state in their discussion that hardly any EMG activity was noted during MI training. As evidence they display readings taken from one participant only. Although there was no increase in EMG for this individual, the case is not necessarily representative of the whole MI group. The reading was taken from a participant who showed one of the lowest force increases in the hypothenar muscles following MI, and the only one to show no increase in force production in the contralateral hand. An alternative explanation for the lack of EMG signal during MI may be simply that the individual was not complying with MI. It is certainly the case that other researchers have found a lack of compliance or inability to perform MI within the healthy population, and have excluded individuals from their studies on that basis (see, for example, Sharma, Jones, Carpenter, & Baron, 2008). This is not therefore an unreasonable alternative explanation for this individual's results. Without seeing results for the whole sample for EMG output during MI training, therefore, it is difficult to judge the assertion that there was no peripheral activation.

Other authors have, however, maintained an emphasis on a functional role for EMG in MI. Gandevia, Wilson, Inglis, and Burke (1997), for example, were concerned to investigate the relationship between MI, muscle spindle activation and EMG. Their experimental hypothesis was that MI might work to improve motor skill



by stimulating fusimotor activity which could then provide afferent feedback to the motor system. Twelve healthy individuals were tested over four different conditions. These included imagination of weak extension and flexion of the specified limb, imagination of flexion and extension of the limb at specified strengths and speeds, MI of complex activities and MI of self-selected activities.

In Gandevia et al.'s (1997) first experiment the experimental conditions were accompanied by microneurographic readings of fusimotor activity taken from the peroneal or radial nerve. In a second experiment the same activities were practised, with EMG readings taken from flexor carpi ulnaris (FCR) and extensor carpi radialis (ECR). In both experiments recordings were also taken when at rest. The participants were instructed to keep the limb still during all imagined movements.

The authors found, firstly, that all of the muscle spindles were active during MI. However, the second experiment also showed that fusimotor activity could not be dissociated from EMG activation. It was found that, during MI, EMG bursts increased significantly from a mean of 1 to a mean of 2.1 every minute. The authors commented that the strength of the EMG signal was also positively correlated with the strength or speed of the imagined movement and argued that "this definitely links the occurrence of EMG bursts to the act of rehearsing the movements" (Gandevia et al., 1997, p. 262).

Gandevia et al. (1997) concluded that MI did not activate the muscle spindle without also activating EMG. It was felt to be unlikely, therefore, that enhanced feedback from the muscle spindle alone could explain the training effects of MI. However the increased EMG signal did point to heightened recruitment of motor neurons during MI. The authors argued, therefore, that MI may involve unintentional subliminal movement practice. This activation would then provide feedback to the CNS and may help explain the signals noted in the motor cortex during MI training. In particular, they suggested, it may help map voluntary movement instructions onto specific muscles.

It is interesting to note that Gandevia et al.'s (1997) interpretation of their results shows some overlap with Yue and Cole's (1992) analysis. The earlier authors also acknowledged that MI training must be linked to increased activation of the motor

neurons. However, in Yue and Cole's (1992) analysis this was believed to follow from an alteration in the central motor program. This led in turn to changes in the balance of inhibitory and excitatory mechanisms at the spinal level, facilitating increased motor neuron recruitment. Gandevia et al. (1997) however, took the view that "imagined movements produce qualitatively similar, but quantitatively lesser, drive to muscles than overt movements" (p. 265). Thus MI effects were not explicable with reference purely to a central motor program. Rather, they were explained by peripheral activation which was reflected in a raised EMG signal. A very important point about Gandevia et al.'s (1997) study is that it is the only experiment on MI and EMG that has used indwelling electrodes, which are more likely to provide accurate recordings of the electrical activity in deeper and tonic muscle fibres (Guillot, Lebon, & Collet, 2010). The results of the study are, therefore, particularly persuasive.

In a later commentary on the work of Gandevia et al., (1997), Gandevia (1999) speculated that the co-activation of the gamma and alpha motor neurons indicated that very low level activation of the muscular apparatus during MI may provide feedback which can then prime motor performance. Referring to the "exquisite sensitivity" of receptors in the spindle and tendon, he comments that it would be surprising if the peripheral activation noted by Gandevia et al. (1997) during MI did not have some functional role in providing afferent feedback which supported subsequent motor performance (Gandevia, 1999, p. 168). This comment is an implied criticism of the motor simulation/central representation position that any raised EMG activity noted during MI is likely to be no more than a failure of the inhibitory impulse which would normally accompany MI.

Guillot et al. (2007) also explored the presence of raised EMG in MI, locating their study in the context of the debate between PNM and central representation approaches. Thirty healthy individuals were tested across four different weight lifting conditions. Each condition involved both executing and imagining the movement. In the first, heavy concentric, condition, the participants were required to lift a weight as fast as possible. In the light concentric condition they had to lift the weight at 50% of their maximal strength. The participants were also required to produce an isometric contraction and lower the weight with an eccentric contraction. EMG readings were

taken from muscles including the long head of biceps, brachioradialis, triceps, flexors carpi radialis and ulnaris and the anterior deltoid. A goniometer was also used to ensure that the elbow did not move during the MI conditions.

Guillot et al. (2007) found that there was significantly greater EMG activity in all of the muscles during the MI conditions than at rest. They proposed, therefore, that not only does MI activate the prime mover in a task, in this case the long head of biceps, but also activates the antagonist, fixator and synergistic muscles. The authors argue that this finding points to the “central and peripheral functional equivalence” of MI and real movement (Guillot et al., 2007, p. 25).

Guillot et al. (2007) also found that the level of EMG signal during MI was task specific. The signal was significantly stronger in the heavy concentric condition, weaker in the light concentric condition and weakest in the eccentric MI task. As a real concentric task would require a larger activation of motor units than an eccentric task this suggests that the EMG response during MI was scaled to the degree of force which would be involved in the executed task. In addition, only the long head of biceps showed a significantly higher EMG signal during a correct performed MI trial.

Guillot et al., (2007) were, however, cautious in their interpretation of these results. They stated, for example, that the raised EMG signal found during MI, which was also scaled to the imagined contraction type, may have a functional role in MI. Of course, this would not be predicted by the central representation position, which, as shown, sees EMG activity as an epiphenomenon resulting from poor suppression of the motor command. On the other hand, Guillot et al. (2007) point out that findings from other studies have not shown increased EMG activity during MI. They therefore maintain that MI training effects are unlikely to be based upon afferent feedback from peripheral activation, as suggested by PNM and also by authors such as Gandevia et al., (1997).

Despite Guillot et al.'s (2007) theoretical conclusions, it might be argued that the results of their study are not unequivocally supportive of the central representation approach. The strength of the EMG response during MI, for example, was linked to the type of muscle contraction involved in the imagined task. It seems unlikely, as the authors acknowledge, that this correlation would have no functional significance.

Another problematic finding was that, despite the lack of any clear connection between MI accuracy and EMG activity in most muscles, there was a statistically significant connection between EMG magnitude and MI accuracy in the long head of biceps, which was the agonist muscle in the task. This may also, therefore, intimate towards a more functional link between MI and EMG intensity.

Lutz (2003) carried out a similar study to that of Guillot et al. (2007). This author's aim was to investigate the source of MI training effects in relation to two hypotheses. In the first, "inflow processing" hypothesis, MI would be based on proprioceptive feedback from peripheral muscular activation; in the second, "outflow processing" hypothesis, MI would be ascribed to activation of a central motor program, with an EMG signal being a consequence rather than the cause of MI (Lutz, 2003, p. 149). These two standpoints are equivalent to the PNM and central representation positions respectively.

Lutz (2003) carried out two experiments on healthy individuals. In the initial experiment 80 novice dart throwers were assigned to one of two groups. In the first group 40 individuals were required to practise MI of dart throwing, while the second group practised a control task based on imaging a neutral mental scene. Both groups then practised dart throwing over a two day period. EMG measurements were taken from biceps, the medial triceps and frontalis muscles for all conditions and groups. It was found that MI of dart throwing was linked to significant EMG increase only in the biceps, and that this was not correlated with task performance or retention.

In the second experiment one hundred and four healthy novice dart players were randomized to groups using relaxation prior to MI or practising a distraction activity. Once again they then either practised MI of dart throwing or the control imagery task. Following this, they practised dart throwing and then took part in a task retention test within 26 hours. In this second trial significantly greater EMG activation during MI was found only in the frontalis. Again, there was no significant correlation between task performance and retention and EMG activation.

Lutz (2003) commented that, although MI was linked to significantly higher EMG signal in two specific muscle groups in both experiments, the central representation analysis was likely to be correct. This was because, first, there was no

connection between EMG activation and error or retention scores. Second, the EMG activation was limited to single muscles and did not follow the triphasic pattern of agonist-antagonist-agonist bursts which would be seen in an executed movement (discussed in Jeanerod, 1988). Overall, then, the author concludes that the augmented EMG signal noted in MI probably 'leaked' from the central motor program and was unlikely to have a functional role in performance.

A study undertaken by Jowdy and Harris (1990) also sought to clarify the role of EMG activation during MI training. These authors were particularly interested to find out if there was a connection between the level of skill performing a task and EMG activity during MI. In this experiment 14 healthy individuals who were skilled jugglers were compared in an MI task with 15 unskilled jugglers. Both groups were then asked to perform MI of juggling while EMG readings were taken from the right bicep. Jowdy and Harris (1990) hypothesized that the more skilled jugglers would show higher levels of activation during mental imaging.

Jowdy and Harris (1990) found a highly significant increase in EMG amplitude during the MI tasks for both groups. This is a figure for the pooled mean difference score for both groups. Taking the skilled group alone the mean score for MI and a resting baseline condition only seem to have differed by a magnitude of  $0.477 \mu\text{v}$ . This suggests that most of the significant difference for EMG levels during imagery was attributable to the results for the unskilled group, who showed an increase of  $0.529 \mu\text{v}$ . Furthermore, although the authors state that there was no significant difference between the results of the skilled and unskilled group, it is also shown that the results of the ANOVA carried out on these values revealed that they differed at  $p = <.05$ . The authors had predicted that the more skilled jugglers would show a higher EMG level during MI, arguing that this would have also been predicted by the PNM model (Jowdy and Harris, 1990). The fact that the opposite turned out to be the case leads the authors to state that the relevance of the raised EMG noted during MI remains unclear. However, it might be argued that the raised EMG noted during imagery for the unskilled group, which may have even been statistically significant, functioned as an adjunct to practice as the skill was being learnt. Overall, the key finding of relevance to the present discussion is that MI could be shown to have link to heightened EMG

signalling.

#### **4.3.2 Bioinformational theory.**

One interesting point of theoretical departure in the debate about the role and presence of raised EMG during MI is based on bioinformational theory. This rejects the notion of the mental image as an analogue structure (Lang, 1979). Rather, the image is seen as being structured from a network of coded propositions (Callow & Hardy, 2004). These might include a stimulus proposition that reveals an object's visual properties and a response proposition that specifies the appropriate muscular response to the stimulus (Callow & Hardy, 2004). Lang (1979) also stated that the image might encompass a motor program. These concepts do not seem to have had any purchase in clinically based studies of MI and EMG. Their main influence seems to have been on research in the field of sports psychology. Two such studies are discussed below.

Bakker, Boschker, and Chung (1996) conducted a study with 22 healthy individuals. They were asked to execute unilateral weightlifting tasks using weights of 4.5kg or 9kg. They were then asked to perform one of two mental imagery tasks. The two imagery tasks were based on a stimulus proposition, in which they visualised the weights and the lifting task, and a response proposition, in which they imagined the feel of the weights. EMG was measured from the biceps bilaterally, with sensors placed on the origin and insertion points of the muscle belly. The authors hypothesized that the stimulus response proposition task would be linked to higher EMG activation when compared with baseline. In addition, they predicted that the EMG response would be stronger for the active arm during the imaging task.

Bakker et al. (1996) found that the EMG response was significantly larger during the imagery task which utilized the response proposition. The EMG response was also found to be significantly stronger in the active arm during imaging. In addition, the EMG response for the active arm was more selective. Here, the difference between imaging for the two weights was significant for the active bicep but not for the passive muscle. It was also noted that there was a higher EMG signal in the active arm when lifting the heavier weight and when using the stimulus proposition imagery. However, this interaction effect just failed to reach statistical

significance (Bakker et al., 1996). The key findings of interest here are that the raised EMG signal appeared to be specific to the muscle involved in the imagery task, as well as being closely related to the content of the task in relation to the heaviness of the weight and to whether the imagery was based on a stimulus or response proposition.

Another experiment influenced by bioinformational theory was carried out by Hale (1982). Interestingly, this study was presented as essentially a re-run of Jacobson's (1931a) study, which was discussed at the beginning of this introduction, using a larger sample size and more modern recording techniques. Hale's (1982) study involved 48 healthy males, half of whom were experienced weight lifters, half of whom were not. The individuals were required to mentally image a series of biceps curls while lifting a weight. They were required to perform either KMI or VMI, presented in a counterbalanced order. Hale (1982) equated KMI with the response proposition and VMI with the stimulus proposition, as outlined by Lang (1979). EMG readings were taken from the dominant bicep.

Hale (1982) subtracted the mean resting baseline measure of EMG amplitude from the mean EMG amplitude during the MI conditions. These formed a series of mean difference measurements which were then compared. It was found that the mean difference for the KMI condition was significantly larger than that for the VMI condition. The author concluded that KMI was therefore more likely to result in a higher efferent outflow. This, he argued, was in line with Lang's (1979) prediction regarding the muscular response proposition. Furthermore, the results were seen by Hale (1982) as a tentative confirmation that the beneficial effects of mental imagery practice may be based on low level muscle activation as measured by the EMG signal.

Another study from the sports psychology perspective is that of Harris and Robinson (1986). In this study sixteen beginning and fourteen advanced karate students were tested while imagining right lateral arm raises with EMG readings being taken from the middle deltoid of both arms. They used both internal and external imagery in the MI task. Internal imagery was defined in relation to the imagined sensation of movement and external imagery as simply imagining watching themselves performing the movement (Harris & Robinson, 1986). The right deltoid

showed significantly higher levels of EMG activation for both imagery types and for both participant groups. In addition, the more skilled students showed significantly higher EMG in the right arm, although there was no difference in EMG in the left deltoid based on skill level. The authors concluded that EMG activation was both specific to the task being imagined and also linked to skill level. Once again, these results seem to call into question the view that a heightened EMG signal has no functional significance during MI practice.

#### **4.3.3 Some authors' complete rejection of the PNM position.**

Some of the authors mentioned above have found evidence of EMG during MI but have disagreed about its functional significance. Other authors have, however, gone farther in their rejection of the PNM position. Mulder (2007), for example, maintains that MI involves the mental rehearsal of a movement with all motor output inhibited voluntarily. He goes as far as to argue that "if MI results in peripheral activation...then we actually cannot talk about MI in the strictest sense" (Mulder, 2007, p. 1274). According to this definition the findings of the MI experiments described above would be invalid. This more radical position has been made operative in the work of other researchers who simply exclude any MI trials in which EMG is noted from their analysis (for example Li et al., 2004; Li, 2007). Mulder and his colleagues have explored these issues experimentally. Two of these studies are described below.

Mulder, Zijlstra, Zijlstra, and Hochstenbach (2004) conducted two trials designed to test the PNM and central explanations of MI. They conjectured firstly that, if MI worked by stimulating the muscle to give afferent feedback, then it may help in learning a totally novel task. On the other hand, they suggested that if MI was a purely central process, based on activation of a motor program, then there would have to be some pre-existing knowledge of the specified training task for MI to be effective.

Their sample consisted of 37 healthy individuals who were unable to abduct the big toe on its own, and 40 healthy people who were able to partially perform this task (Mulder et al., 2004). Both of these groups were assigned to mental practice, physical practice or no practice groups. During mental practice EMG readings were taken from abductor hallucis. The procedures were the same for both experiments,



with the difference that only the prior toe abductors were included in the second trial.

It was found that only the group who could already abduct the big toe improved with mental practice alone. In addition this group did not show significantly different levels of improvement to the group of toe abductors who used only physical practice. The absolute improvement in range of motion was, however, smaller for the MI group. It was also noted that mental practice did not produce any EMG output, although data are not provided for this in the paper.

Mulder et al. (2004) concluded that the results supported a central representation hypothesis. This was based on the assumption that the participants were accessing a pre-existing motor program for a previously experienced activity as an aid to training. They argued that the group with no prior experience of the movement would not have had such a motor program, and therefore did not benefit from MI. In addition, the authors suggest, the lack of any EMG activation demonstrates that they were not able to generate proprioceptive feedback through imagined movement which could aid their performance. This final claim underlines their rejection of PNM. The lack of any data for EMG signals, however, makes this conclusion difficult to assess.

These themes were revisited by Mulder, de Vries, and Zijlstra in 2005. This experiment was based on the proposition that the results of their 2004 paper may have been explained by the fact that their training task was novel, requiring little force production. They therefore chose to use a more common exercise task which required substantial force.

The participants were two groups of healthy males, one group who had experience of performing squat exercises, another group who had not. For the control condition the participants sat quietly for five minutes. They then watched a video of a man performing squats while carrying 12.5kg weights. Following this they were told to imagine the task from a first person perspective. They then had to actually perform the squat exercises themselves. Following a period of rest they then imagined the activity once more. Throughout the conditions, EMG readings were taken from vastus medialis and rectus femoris and from the heart. A thermistor was also used to measure respiration in breaths per minute.

Mulder et al. (2005) found no significantly different levels of EMG activation

in any of the non-performance conditions for the leg muscles or for the heart. However, significant differences in respiration rate were found for the non-performance conditions when compared with the resting control condition. Mulder et al. (2005) suggested that these findings support a central patterning interpretation. In addition they introduce the idea that MI may work by producing a kind of “pseudo-proprioception” (Mulder et al., 2005, p. 349). This view is based on Jeannerod’s (2006) hypothesis of motor simulation and posits that the centrally generated image provides feedback that can be utilized in action execution.

This paper does seem to confirm Mulder et al.’s earlier (2004) proposition that there is no peripheral EMG activity associated with MI. However, as the authors point out, these results are in contradiction with the findings of other researchers. Mulder et al. (2005) conclude that further work would be needed to understand the possible motivational and emotional factors which might be involved in the inhibition of motor output.

#### **4.3.4 EMG during MI in stroke.**

It will be noted that the majority of studies which have been referenced above refer to work that has been conducted on healthy individuals. However, the ongoing debate about the role of the EMG signal in MI has been addressed with regard to stroke patients by Dickstein et al. (2005). These authors asserted that “the definition of imagery as a mental task neither implies nor disregards the likelihood of occurrence of recordable muscle activation” (Dickstein et al., 2005, p. 476). They were concerned to look at levels of EMG activation in relation to MI in lower limb hemiparesis following stroke. In this study six people with stroke, four of whom had strokes affecting the basal ganglia and two of whom had parietal lesions, took part. In addition a control group of nine healthy individuals participated.

Dickstein et al’s (2005) participants were required to repeatedly stand on tip toe, and also perform MI of this task. EMG recordings were taken from the medial gastrocnemius, the prime mover in this activity, and rectus femoris. The only control to ensure that movement did not take place during MI was provided by one of the researchers watching the participants during MI performance.

EMG activity during MI was noted in three of the basal ganglia patients and

three of the healthy participants. The authors do not state whether these EMG signals were significantly different for these six individuals when compared with the resting baseline readings. The EMG signal during MI was, however, significantly larger for the three healthy volunteers when compared with the stroke patients. In addition the EMG signal was significantly lower for the MI condition than in the execution condition. Also, the EMG activation in the medial gastrocnemius was significantly higher than for the rectus femoris. There was no EMG activity noted during MI for the other nine subjects (Dickstein et al., 2005).

Dickstein et al. (2005) concluded that there was no straightforward relationship between the EMG signal and MI based on two findings. First, there was no consistent pattern for activation across the two groups. Second, the levels of activation noted for MI were significantly lower than during the execution condition. The authors do, however, support a central explanation of MI along with Jeannerod's (2006) concept of concomitant motor inhibition.

It might be argued, however, that some of Dickstein et al.'s (2005) speculative conclusions do not entirely fit with Jeannerod's (2006) hypothesis. The authors suggested, for example, that the EMG signal noted in some of the MI trials might be part of an error detection strategy used by those individuals as part of motor preparation. The authors also argued that some of their participants were using muscular contractions as an adjunct to MI. Furthermore, it was found EMG signal during MI was significantly larger in the medial gastrocnemius, the agonist muscle in this task. This may be compared with Lutz's (2003) finding that the EMG recording in MI was significantly higher in the agonist bicep. It will also be recalled that Guillot et al. (2007) noted a significant link between the size of the EMG signal in the prime mover in their training task, also the bicep, and accuracy of MI. Taken together, these results may point to an active role for the EMG signal in MI, in keeping with PNM but rejected by proponents of central representation.

The above discussion shows there are a range of views about the relation of EMG and MI. Jacobson (1930a) provided evidence which supported the idea that peripheral activation allowed subliminal practice which explained the effectiveness of MI training. This perspective was adopted later by Gandevia et al. (1997). It was

shown, in addition, that a number of researchers working from a sport psychology perspective have found not only increased EMG signalling during MI, but also that the pattern of activation appears to be task and muscle specific (Bakker et al., 1996; Hale, 1982; Harris & Robinson, 1986; Jowdy & Harris, 1990). However this view has more recently been marginalized by the hypothesis of motor simulation, which sees EMG as essentially a functionally irrelevant leakage from a central motor program (Jeannerod, 2006). This position has influenced the arguments of some authors who have found evidence of EMG activity in MI but have denied it any functional significance (Dickstein et al., 2005; Guillot et al., 2007; Lutz, 2003). It was suggested, however, that some of the evidence presented by these authors would not seem to support such a theoretical position. Other authors have gone further and argued, or presumed in practice, that MI actually precludes EMG activation (Mulder et al., 2004, 2005; Li et al., 2004; Li, 2007). It also has been argued above that some of the evidence presented by those supportive of the motor simulation hypothesis is open to other interpretations (Yue & Cole, 1992). Furthermore, it was suggested that a fuller presentation of evidence in two papers would make the authors' conclusions easier to assess (Mulder et al., 2004; Yue & Cole, 1992).

#### **4.4 Hypotheses Regarding the EMG Signal During MI in individuals with stroke.**

##### **4.4.1 Movement inhibition may be more difficult in post-stroke hemiplegia.**

As noted above, the hypothesis of motor simulation has provided a key reference point in discussions about MI (Jeannerod, 2006). An important component of this hypothesis is the notion of the inhibition of the motor signal, which is proposed as the means by which muscular activation is blocked during mental movement practice. Any raised EMG activation is therefore viewed as a result of a breakdown in this process. Evidence explored in the following section suggests that such inhibition may be particularly problematic in persons with hemiplegia following stroke.

Hemiplegia has been linked to a collection of signs and symptoms grouped together under the heading of the "upper motor neuron syndrome" (UMN) (Carr & Shepherd, 1998, p. 185). UMN has traditionally included positive features, in the sense of unwanted additional movements, and negative features, which describe

functions which are lost to the patient (Carr & Shepherd, 1998). One of these positive features is the overactivity of the muscle, or hypertonicity, the neural constituent of which has been labelled spasticity (Raine, 2009). Spasticity has its origin in altered supra-spinal and spinal control mechanisms (Gracies, 2005). One aspect of this altered control is the poor inhibition of movement at both these levels. Some of the mechanisms implicated in this are discussed below.

Following stroke there may be a transfer of descending control from the damaged corticospinal pathway to other subcortical paths, such as the vestibulospinal (Gracies, 2005). These paths are less specific in their targets and may therefore produce unwanted collateral movements in addition to that specified by the motor command (Gracies, 2005). The intact corticospinal fibres may also re-branch onto unsuitable motor neurons, again producing superfluous movements in relation to a given signal. In addition, newly vacated regions of an axon release the growth protein GAP-43, leading to a sprouting of new axonal terminals from neighbouring neurons onto the damaged area (Gjelsvik, 2008). This can disrupt the established innervation, leading to excessive reflex reactions to sensory input and increased hypertonicity (Gjelsvik, 2008; Gracies, 2005).

Evidence also suggests that the glycinergic Renshaw interneuron, responsible for the recurrent inhibition of the alpha motor neuron and controlled via the corticospinal system, may itself become inhibited. This can lead to disruption of the inhibitory 1a interneuron, affecting the process of reciprocal inhibition of the antagonist muscle and causing co-contraction during attempts to move a joint and increased stiffness (Gracies, 2005b). In consequence, then, damage to the Renshaw cell may reduce the selectivity of movement (Gjelsvik, 2008). The alpha motor neuron may also undergo a change in resting membrane potential. A plateau potential may be established in which the cell membrane is sustained in a higher than normal state of depolarization, leading the cell to fire in response to a lower than normal excitation (Gjelsvik, 2008; Pearson & Gordon, 2000).

#### **4.4.2 There may be higher EMG in hemiplegic muscles.**

All of the above factors may lead to pathologically raised activity in the motor units and the muscle, and, consequently, a heightened EMG signal. Researchers have

indeed found evidence of abnormal EMG response in patients with upper motor neuron lesions (for example, Fleuren et al., 2006). Given the disruption of motor inhibition in UMN, described above, it might be predicted, in line with the motor simulation hypothesis, that MI would be linked to raised EMG output in the targeted muscles of stroke patients. This could be tested by comparing the EMG responses of healthy individuals and stroke patients during MI. To the best of the author's knowledge, the only MI trial to have compared stroke patients with neurologically intact individuals with regard to EMG activation is that of Dickstein et al. (2005), which has been described above. One limitation of this paper is, however, the authors' failure to clearly define the sensory modality of the MI task used. The problem with this omission is explained below.

It has already been explained in chapter two of this thesis that MI can be based on kinaesthetic or visual modalities. Furthermore, it has been shown that KMI and VMI may have very different effects. It may therefore be important to define the sensory modality used in MI experiment looking at the EMG signal during MI. One paper in particular is germane to the present discussion. This is discussed below.

Stinear et al. (2006) sought to test the respective roles of KMI and VMI in promoting motor cortical excitability. They used TMS to assess the excitability of cortical representation in areas corresponding to ADM and abductor pollicis brevis (APB). Twenty healthy individuals were tested under four different conditions which included a VMI and a KMI task. Ten of the group was then tested using TMS. It was found that only the KMI task led to an increase in excitability above resting level in the cortical area corresponding to APB (Stinear et al., 2006).

Stinear et al.'s (2006) study would suggest that only KMI promotes cortical excitability. EMG can be taken as one measure of cortical excitability (Li et al., 2004; Li, 2007). It may also therefore be the case that only KMI would be linked with a raised EMG signal. From this point of view it would be incumbent on the researcher to carefully define the sensory modality of imagery deployed in the experiment.

#### **4.5 Links Between 2<sup>nd</sup> and 3<sup>rd</sup> Experiments**

The hypotheses for experiment three flowed directly from experiment two. Experiment two had shown that VMI and KMI may have different effects on motor

behaviour during the rehabilitation of the hemiplegic hand. It was therefore of interest to compare the influence of these two modalities on the levels of muscular activation which might underpin such behavioural effects.

The hypotheses of the third experiment were also based on the body of theoretical work which has contributed to or developed out of the hypothesis of motor simulation (Jeannerod, 2006). In this sense, there was a theoretical link between the two experiments, as the research question guiding experiment two was also based implicitly upon these insights.

The focus on the intrinsic hand muscles affected by hemiplegia was also maintained in experiment three. However, the different technical approach adopted meant a shift away from the focus on the lumbricals. The objective was to capture recordings of the electromyogram signal via surface electromyography (SEMG). The lumbricals would have been very difficult to access using this technology for two reasons. First, they are covered by a fibrous aponeurosis (Palastanga et al., 2005); second, it would be difficult to distinguish an EMG signal in the lumbricals against the background signal of the FDP from which they originate. For experiment three, therefore, SEMG recordings were taken from the APB and the ADM.

The research question for experiment two originated from a gap identified in the literature concerning VMI and KMI. Similarly, the review of literature carried out as part of the process of designing experiment three suggested that, although one study had identified that KMI had a different effect on the EMG signal when compared with VMI (Hale, 1982), this had not been tested on individuals with stroke. This then provided the basis of the research question for experiment three.

Another link between the two studies is the question of the effects of the different MI modalities on variability in movement patterns. The way this was done for experiments one and two has already been described above. In experiment three, this was addressed by the collection and analysis of data on the SDs of the EMG signal in the different experimental conditions.

One point of departure in experiment three, in addition to the technique of data collection, was the use of a neurologically intact participant group. The objective here was to test if any effects on the EMG signal in any of the imagery, relaxation or

voluntary contraction conditions were linked to the presence of hemiplegia, or were noted in both groups equally.

## **4.6 Method**

### **4.6.1 Recruitment process.**

In experiment three, nine of the ten participants with stroke had previously taken part in experiment two. Eight of these had been recruited via the Hospital Universitario Major and Barrios Unidos hospitals. The ninth was recruited via the university community, as explained in chapter three. Following participation in experiment two each person was informed that a third experiment would be conducted, and that if they wished they might be contacted by telephone in the future. If they gave verbal consent to this, then they were informed that they might be contacted about the next experiment. Participants were then contacted by telephone during the recruitment period for experiment three. If they expressed continued interest in taking part, they were visited at home. Once ten participants with stroke had been recruited, recruitment was stopped.

Participant number four had not taken part in the earlier experiment. He had initially been approached at one of the hospital sites for experiment two. However, he did not fit the criteria for the previous study as he had reduced mobility. He gave verbal consent at that time to be contacted about any future research. Since he did not need to mobilise to take part in experiment three, he was approached to take part.

The recruitment of participants from amongst the community to which the researcher has most immediate access is common practice in motor control studies (see, for example, Hansen et al., 2007). In the case of experiment three, the age matched healthy individuals were initially recruited from among personal and professional contacts of the researcher. Recruitment was then based on “snowballing” from these initial contacts (Patrick, Pruchno, & Rose, 1998, p.297). This is a technique in which one participant nominates another potential candidate, and obtains permission from that person for the researcher to contact them. It has been identified as an efficient and cost effective way to recruit a sample in health research (Streeton, Cooke, & Campbell, 2004; Patrick et al., 1998). There are, however, disadvantages identified with this recruitment method, which are outlined in the discussion section of



this chapter.

The initial contact with the neurologically intact individuals was made by telephone. As has been pointed out above, this is considered a more secure method of communication in Bogotá than mail. Furthermore, the use of initial telephone contact has been used in other published studies, such as that of Patrick et al. (1998).

Following discussion with the local supervisor, it was felt that, as the experience of the participants would be substantially the same as in the previous experiments, the same information sheet could be used for experiment three as was used for the earlier experiments. Again, plenty of time was given and any questions answered. More time was given if requested. All participants signed to indicate informed consent. All participants completed the study. No participants received any payments for taking part in the study. Data collection took place during February 2011.

#### **4.6.2 Note on the sampling process.**

As for the previous experiments, this was a non-probabilistic convenience sample, which did not follow the rules of random sampling. The limitations of this approach to sampling are discussed further on in this chapter.

#### **4.6.3 Participant characteristics.**

Ten people with hemiplegia affecting the hand were recruited. Their demographic details are provided in table 4:2. Their time since stroke has been adjusted from that shown in experiment two.

In addition, ten individuals who had not had a stroke, age-matched to within 5 years of the stroke patients, were recruited. None of the healthy group had any medical condition which would have prevented participation in the study. All were over 18 years of age. All had normal or corrected to normal vision and hearing. Their demographic details are given in table 4:3.

Table 4:2.

*Stroke Participant Characteristics*

Stroke	Age	Gender	Time since stroke	Affected side	VKVIQ	KKVIQ
1	55	F	11 months	R	53	65
2	64	M	7 months	R	15	18
3	48	F	9 months	L	44	43
4	67	M	5 months	R	68	52
5	64	M	3 years 11 months	R	68	68
6	73	F	3 months 2 weeks	R	64	65
7	76	M	7 months	R	69	33
8	45	F	6 months	R	57	48
9	55	F	7 months	R	84	45
10	51	M	8 months	R	66	78

VKVIQ = visual component of KVIQ, KKVIQ = Kinaesthetic component of KVIQ

Table 4:3.

*Healthy Participant Characteristics*

Healthy	Age	Gender	Hand dominance	VKVIQ	KKVIQ
1	59	F	R	17	17
2	60	M	R	68	85
3	53	F	R	84	51
4	64	M	R	17	17
5	68	M	R	72	85
6	69	F	L	18	31
7	72	M	R	85	51
8	43	F	R	64	74
9	60	M	R	55	45
10	50	M	R	31	37

VKVIQ = visual component of KVIQ, KKVIQ = Kinaesthetic component of KVIQ

#### 4.6.4 Sample size.

The hypotheses in this experiment had not, to the best of the authors' knowledge, been tested in an earlier experiment. Consequently, there were no data upon which to base a calculation of sample size. The intended and actual sample size of the study was twenty. As indicated in table 4:2, ten participants with stroke were recruited, with a mean (SD) age of 59.8 (10.57). Five were male and five female. Table 4:3 shows that ten healthy individuals were recruited to the study, with a mean (SD) age of 59.8 (9.09) years. Six were male and four female. Table 4:4 shows the baseline assessment details of stroke participant number four, who had not been included in experiment two.

Table 4:4.

*Stroke Participant Four Baseline Assessment Details.*

	Motor Assessment	Motor Assessment		
Motor Assessment	Scale hand	Scale advanced		
Scale upper arm	movements	hand	Ashworth score	MMSE
4	2	0	3	27

#### 4.6.5 Pilot study.

Experiment three was piloted using students and staff from the University of Rosario community. Six people were tested. Their age range was 18-50. Four were female. As for experiments one and two, the aim was to confirm the feasibility of the research design. The objectives were: (a) to check that the surface EMG sensors were located appropriately on the hand, done by asking the person to contract the target muscle against resistance and identifying the location of the belly of the muscle; (b) to check that the recorded task instructions were understood appropriately by the participants and that they performed the tasks as instructed; (c) that the DataLog device was channelling the spoken instructions and the EMG signals simultaneously. It was not necessary to make any further changes to the design following this pilot.

#### **4.6.6 Measures used.**

As in the experiments previously described in this thesis, The Motor Assessment Scale (MAS) was used as a screening assessment of hand function in stroke participant number four. In addition, the Modified Ashworth Scale was used again in order to assess muscle tone in this patient. The healthy group was not tested with these scales and the remainder of the stroke group was not retested. Stroke participant four and the healthy group were also assessed using the Spanish translation of the KVIQ. The results can be seen in tables 4:2, 4:3, and 4:4. The details of these assessments have already been explained in chapter two. All testing was carried out by the researcher.

#### **4.6.7 Research design.**

A between-within design was used with measurements repeated of one factor. The within factor was ‘treatment’, on which repeated measures were made. The between factor was ‘group’ (Stroke x Healthy).

#### **4.6.8 Apparatus.**

EMG and electrogoniometry data were captured using the DataLog MWX8 (Biometrics, UK). Surface EMG was recorded via two electrodes (Biometrics, UK). These were active electrodes with inbuilt amplifiers. In addition, the electrodes had two inbuilt filters. These were a low pass filter with a cut off frequency of approximately 450 Hz and a high pass filter with a cut-off point of about 15 Hz. The action of the two filters together acted as a band-pass filter, allowing signals of middle frequency bands to pass. The electrodes had a high input impedance of  $10^{15}$  ohms. This, in addition to the fact that these were active electrodes, meant that an electrolyte gel was not needed in order to improve conductivity (Kamen & Gabriel, 2010). However, the skin was cleaned with pure alcohol prior to attachment of the electrodes. The electrodes were then fitted using purpose designed double-sided adhesive tape.

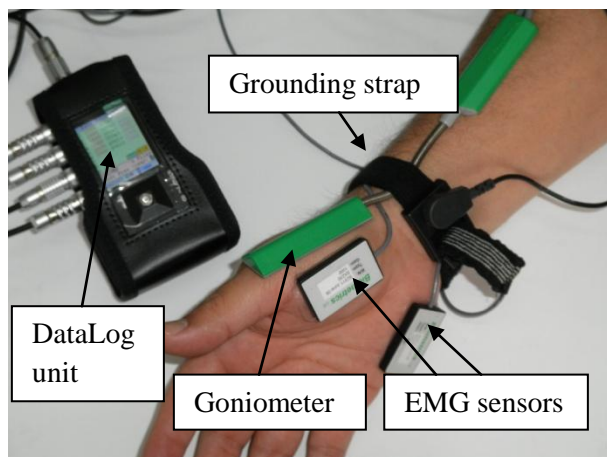
An electrogoniometer (Biometrics, UK) was also used to measure the movement of the thumb. This was also connected to the DataLog unit. A ground strap (Biometrics, UK) was also looped around the participants’ wrists, and then connected back to the main unit. The location of these items, as well as the DataLog unit, is shown in figure 4:1. All videos and recorded instructions were watched or listened to

with headphones via a lap top computer that was also connected to the DataLog unit. When performing maximum voluntary contractions (MVCs), participants abducted their thumbs against a block of specially prepared smooth wood which was held in place by the researcher.

Captured EMG data were stored in DataLog files and opened using Biometrics DataLog software (Biometrics, UK). These were then exported as ASCII files for offline processing in spreadsheets.

#### **4.6.9 Procedure.**

All procedures were carried out in the participants' homes. The procedure was as follows. The location of the muscle belly was first identified. This was done according to the method recommended by Palastanga et al. (2006). While the individual was seated comfortably in front of a table, the hemiplegic thumb in the patients and the non-dominant thumb in the healthy group was abducted against resistance and APB was identified by the researcher via palpation. Following this, the little finger of the same hand was abducted against resistance and ADM was identified using the same procedure. The electrodes were then attached at the central part of the muscle bulge. This is a commonly used technique in sensor placement in surface EMG studies (Hermans, Freriks, Disselhorst-Klug, & Rau, 2000). The sensors were attached as explained above. The ground strap was looped over the wrist of the hemiplegic hands of the stroke patients and the non-dominant hands of the healthy group. An electrogoniometer was attached with double-sided adhesive tape to the thumb PIP and the radial aspect of the forearm. The positioning of all these items is illustrated in figure 4:1.



*Figure 4:1.* Position of EMG sensors, grounding strap and goniometer. The DataLog unit is also shown.

Participants were first asked to make themselves comfortable in sitting with their hands resting in pronation down on their lap. They then listened to a short guided relaxation script through headphones. This had been recorded onto a laptop computer. During this period, recordings were made from the EMG electrodes and the goniometer. This recording provided the resting baseline measurement. Following this they watched a video which showed a thumb performing MVC against the block of wood. This was accompanied by a recorded commentary explaining the MVC task. The video watched depended upon the hand to be tested and the gender of the participant. Thus, a female participant whose left thumb movements were being tested watched a video of a female left hand performing the task, and so on.

The participants then placed their hemiplegic/non-dominant hand in pronation on the table in front of them. Following this, they listened to recorded instructions to perform a maximum voluntary contraction (MVC) of the hemiplegic/non-dominant thumb. The participants performed these tasks against the block of wood and EMG and goniometric data were acquired at the same time. This recording provided a baseline MVC.

The participants then listened to instructions to perform KMI and VMI. These were presented in a counterbalanced order, so that some performed KMI first and VMI second, while with other participants the order of presentation was reversed. Again, EMG and goniometry readings were taken during these MI treatments. In addition,

each MI task was followed by a further relaxation and a further MVC. EMG readings were also taken during each of these conditions.

#### **4.6.10 Timing of scripts.**

The initial relaxation task recorded script lasted for 1 min 59 s for both the stroke participants and the neurologically intact group. As stated above, there were four different videos; the average length of these was 30 s. The MVC task script for the stroke group lasted 1 min 49 s, while that for the healthy group lasted 1 min 44 s. The VMI script for both groups lasted for 2 min 26 s. The KMI script for the stroke group lasted for 2 min 52 s, while that for the healthy group lasted for 2 min 35 s.

The text for all the scripts is given in appendix 4:1.

#### **4.6.11 Data analysis and reduction.**

The root mean square (RMS) values of the EMG data were calculated using the Biometrics DataLog software (Biometrics, UK). The RMS values of the goniometry data were also calculated. The data were then prepared for export as ASCII files and offline processing. A mean value and SD value were then calculated for each participant for each condition for EMG and goniometry. These values were then used in statistical analysis.

### **4.7 Results**

#### **4.7.1 Participant flow.**

The flow of the participants through the experiment is shown in figure 4:3.

#### **4.7.2 Statistical tests.**

All factorial ANOVAs discussed in the results were subject to the Greenhouse-Geisser correction in order to control for multi-sample sphericity. Any significant main effects were subjected to post hoc testing using Tukey's Honestly Significant Difference (HSD) test. Further post hoc testing was carried out using paired and independent samples *t* tests. In addition, testing was carried out using Pearson's product-moment correlation coefficient (*r*). The threshold value for statistical significance for all tests was set at  $p < .05$

#### **4.7.3 KVIQ scores for experiment three.**

The visual component of the KVIQ scores for the stroke group and the health group in experiment three were compared for significant difference using a two tailed *t*

test. No significant difference was identified,  $t(18) = 0.726$ ,  $p = .477$ . Nor was there any significant difference for the kinaesthetic component of the test,  $t(18) = 0.224$ ,  $p = .825$ . The mean values are shown in figure 4:2.

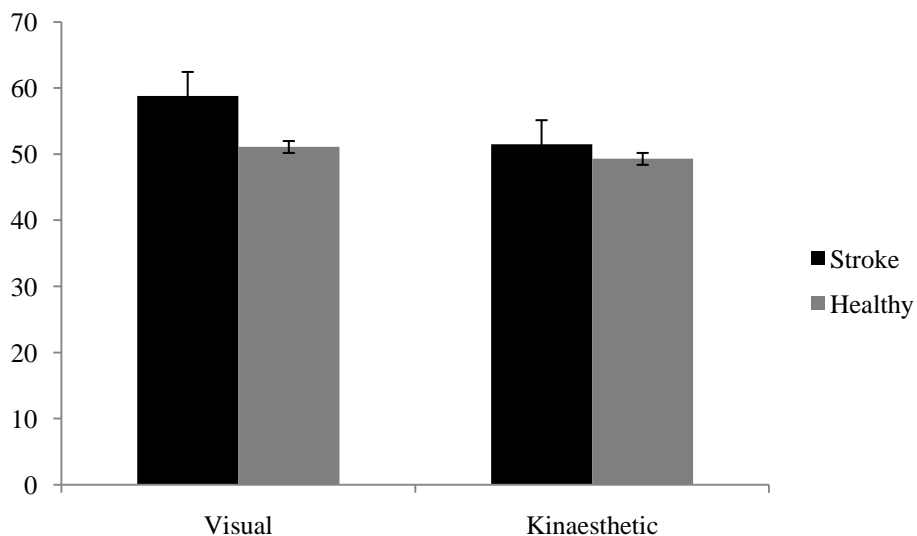


Figure 4:2. KVIQ scores for experiment three.

#### 4.7.4 Results for APB

##### 4.7.5 Resting baseline, VMI and KMI conditions.

First, it was desired to know if there was any difference in the EMG activity of the APB muscle during the MI conditions, when compared with the resting baseline condition. In addition, it was wished to know if there were any differences between the stroke and the healthy groups. The mean levels of EMG activation during the resting baseline and VMI and KMI conditions were therefore compared using a 2 x 3 (Group [stroke, healthy] x Treatment [resting baseline, VMI, KMI]) between-within factorial ANOVA. The mean (SD) values can be seen in table 4:5. There was a significant main effect of treatment,  $F(2,18) = 5.642$ ,  $p = .023$ .



Figure 4:3. Flow of participants through study.

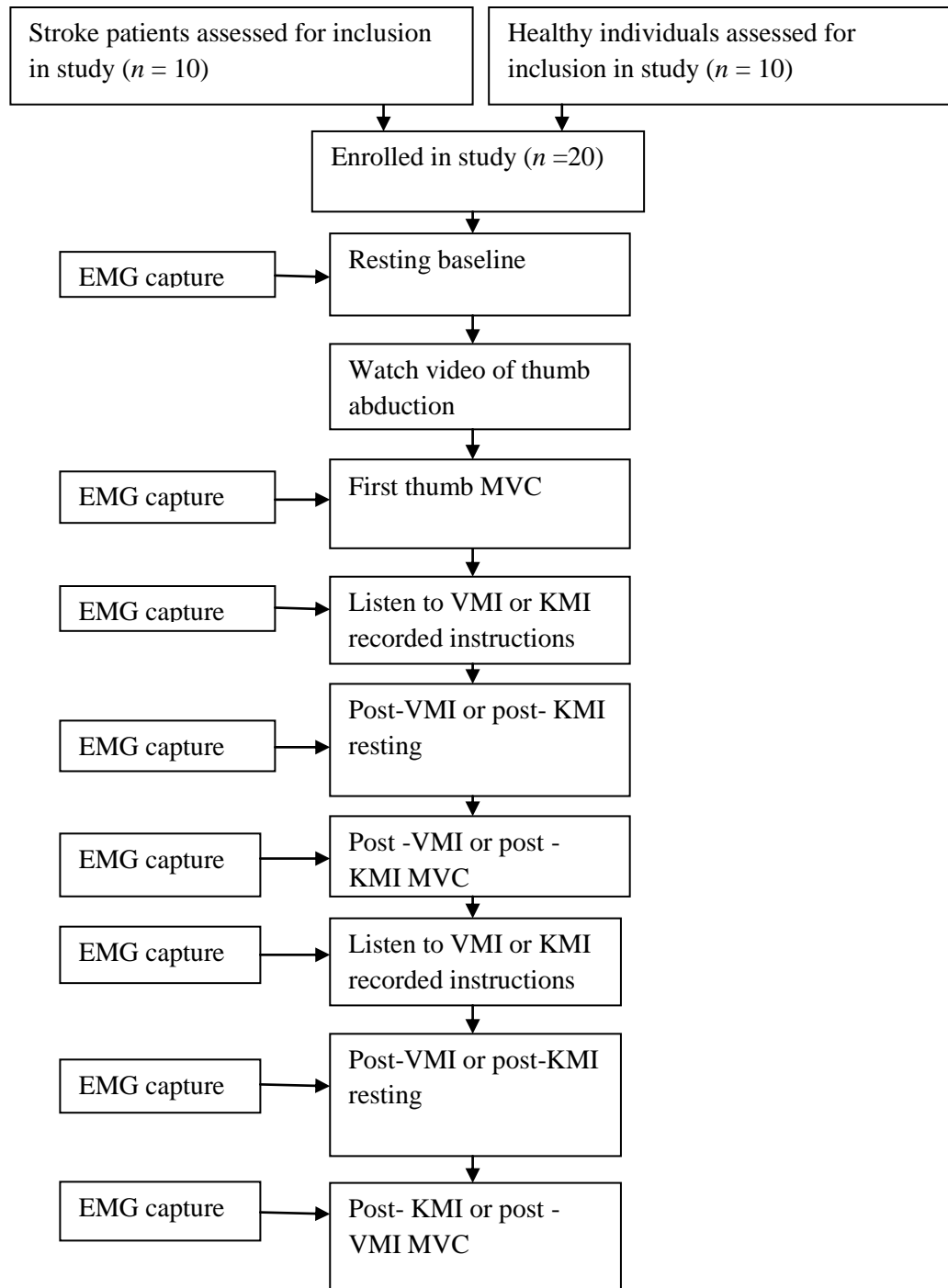


Table 4:5.

*Mean (SD) EMG (mV) for APB*

	Stroke	Healthy
Resting baseline	1.513 (0.088)	0.445 (0.089)
MVC 1	1.612 (0.314)	0.918 (0.666)
VMI	1.239 (0.089)	0.395 (0.098)
Post-VMI rest	1.121 (0.328)	0.337 (0.184)
Post-VMI MVC	1.478 (0.346)	0.929 (0.65)
KMI	1.219 (0.084)	0.397 (0.085)
Post KMI rest	1.219 (0.388)	0.345 (0.192)
Post KMI MVC	1.507 (0.343)	0.926 (0.643)

A further one way ANOVA was therefore carried out on the stroke group alone for the factor ‘treatment’, however no further significant differences were revealed,  $F(2, 9) = 0.321, p = .728$ . A one way ANOVA was also carried out on the healthy group alone for this single factor. No further significant differences were found here either,  $F(2, 9) = 0.120, p = .887$ .

Further post hoc testing was carried out to compare the EMG levels for the stroke group during the resting baseline and each of the MI conditions separately using a two-tailed paired samples  $t$  test. This showed that there was no significant difference for the stroke group between the resting baseline condition (1.513mV) and the VMI (1.239mV) condition,  $t(9) = 1.877, p = 0.093$ . However, there was a significant difference between the resting baseline condition and the KMI (1.219mV) condition,  $t(9) = 2.857, p = 0.019$ . This indicated that there was significantly lower EMG activity in APB during KMI (1.219mV) when compared with the resting baseline condition. These findings are shown in figure 4:4. This was not the case for the healthy group, where post hoc comparison of the resting baseline EMG (0.445mV) and EMG during KMI (0.397mV) did not reveal any significant difference,  $t(9) = 1.156, p = .278$ . Nor was there a significant difference between the resting baseline and the VMI (0.395mV) condition for the healthy group,  $t(9) = 1.06, p = .317$ .

There was also a significant main effect of group,  $F(1, 18) = 9.527, p = .006$ . A one way ANOVA on the factor ‘group’ showed that the EMG activation for the stroke group was significantly higher than the healthy group during the baseline resting period,  $F(1,18) = 13.245, p = .002$ , during VMI,  $F(1,18) = 7.821, p = .012$  and during KMI,  $F(1, 18) = 6.952, p = .017$ . These results are also illustrated in figure 4:4. There was, however, no significant interaction effect of training and group  $F(2, 18) = 2.773, p = .107$ .

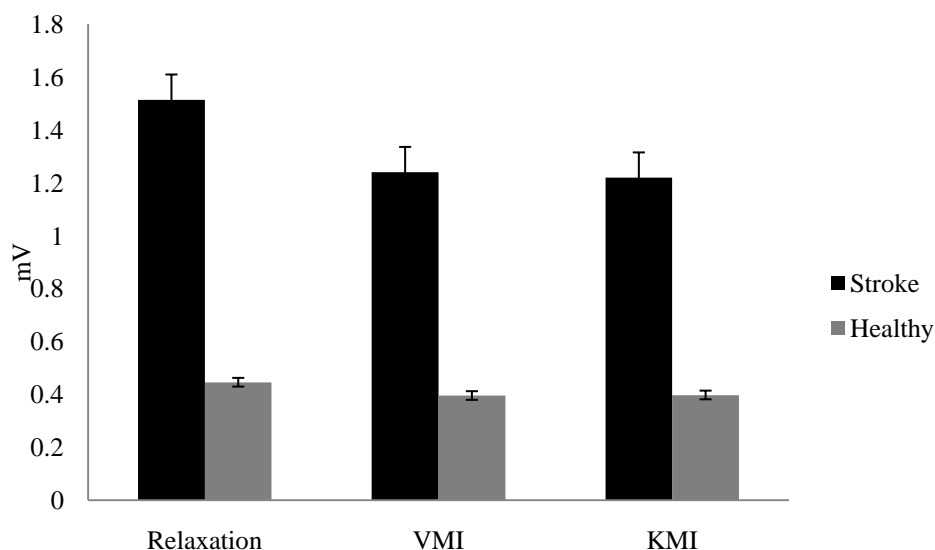


Figure 4:4. Mean EMG activation levels during relaxation, VMI and KMI conditions for APB.

#### ***4.7.6 Resting baseline compared with post-VMI relaxation and post-KMI relaxation.***

Here, it was wished to know if there was any difference between the initial resting baseline, prior to any MI treatment, and the readings taken while the participants were resting following KMI and VMI. As before, comparisons were also made between the stroke and healthy group in this respect. The mean levels of EMG activation during the resting baseline, the post-VMI relaxation and the post-KMI

relaxation conditions were consequently compared using a 2 x 3 (Group [stroke, healthy] x Treatment [resting baseline, post-VMI resting, post-KMI resting ]) between-within factorial ANOVA.

The mean values can be seen in table 4:5. There was a significant main effect of treatment,  $F(2, 18) = 9.371, p = .001$ . A one way ANOVA was therefore carried out on each group separately for this single factor. For the stroke group, the results were not significantly different,  $F(2, 9) = 0.51, p = .602$ , neither were those of the healthy group,  $F(2,9) = .716, p = .497$ .

Further post hoc testing was carried out in order to compare the resting EMG following each of the MI conditions separately with the initial resting baseline. Here, it was found for the stroke group that the resting EMG following VMI (1.478mV) was significantly lower than for the resting baseline (1.513mV),  $t(9) = 2.872, p = .018$ , as was the resting baseline following KMI (1.219mV),  $t(9) = 2.914, p = .017$ . For the healthy group the post VMI (0.337mV) resting baseline was also significantly lower than the initial resting baseline (0.445mV),  $t(9) = 2.306, p = .047$ . However, the resting baseline following KMI (0.345mV) was not significantly different to the initial resting baseline,  $t(9) = 1.75, p = .114$ .

It was found here as well that there was a significant main effect of group,  $F(1, 18) = 10.23, p = .005$ . A further one way ANOVA on the factor 'group' showed that the stroke group had significantly higher EMG activation during the resting baseline,  $F(2, 9) = 13.245, p = .002$ , during the post-VMI relaxation period,  $F(2,9) = 7.821, p = .012$ , and also during the post-KMI relaxation period,  $F(2, 9) = 6.952, p = .017$ . These results are shown in figure 4:5. There was no significant interaction effect of training and group,  $F(2, 18) = 2.808, p = .074$ .

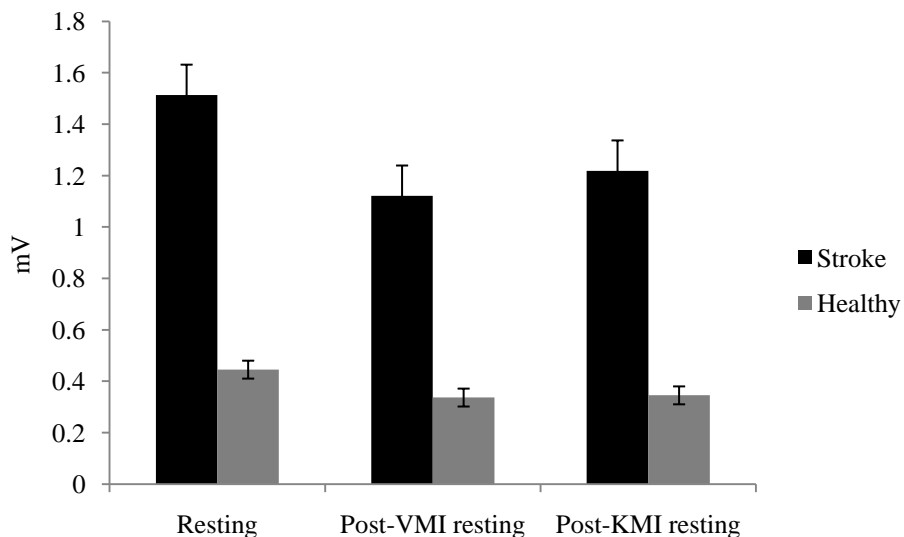


Figure 4:5. Mean EMG activation during resting baseline, post-VMI resting and post-KMI resting for APB.

#### 4.7.7 MVC following resting baseline, post-VMI MVC, post-KMI MVC.

A comparison was also made of the initial MVC, taken after the resting baseline and before any MI training, and the MVCs which were recorded following VMI and KMI. The main objective here was to find out if the MI conditions had had a facilitatory effect on the EMG levels during MVC. In addition, it was also desired to know if there were any differences between the stroke and healthy groups. The mean EMG values of the initial MVC, the MVC following VMI and the MVC following KMI were therefore compared using a 2 x 3 (Group [stroke, healthy] x Treatment [initial MVC, post-VMI MVC, post KMI MVC]) between-within factorial ANOVA.

The mean values can be seen in table 4:5. There was no significant main effect of treatment,  $F(2, 18) = 1.113, p = .340$ . Neither was there a significant interaction effect of treatment and group,  $F(2, 18) = 1.544, p = .227$ . However, there was a significant main effect of group,  $F(1,18) = 6.048, p = .024$ . A one way ANOVA was therefore conducted on this factor. This showed that the EMG activation for the stroke group was significantly greater than that for the healthy group during the initial MVC,  $F(1, 18) = 8.534, p = .009$ ; during MVC following VMI,  $F(1, 18) = 4.582, p = .046$  and during MVC following KMI,  $F(1, 18) = 4.918, p = .04$ . These results suggested

that the MI conditions were not linked to a stronger EMG signal during MVC, and that the key factor in determining the higher EMG activation was the presence of stroke.

These results are shown in figure 4:6.

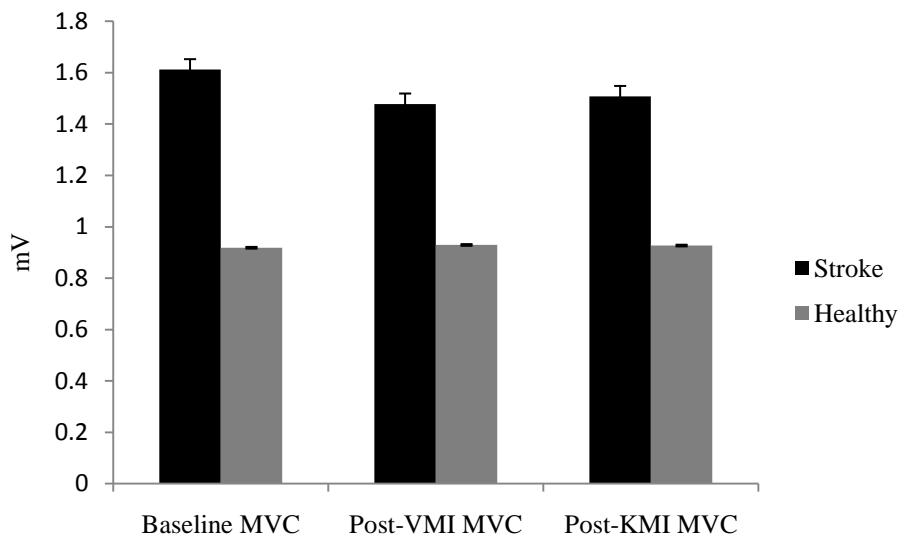


Figure 4:6. EMG activation levels for initial MVC, post-VMI MVC and post-KMI MVC for APB.

#### **4.7.8 Standard deviations (SDs) of APB EMG during resting baseline, VMI and KMI.**

It was desired to know whether the reduction in EMG activation following the onset of MI treatment was linked to changing levels of variability in the signal during MI. The SDs of the mean EMG signal were therefore compared using a 2 x 3 (Group [stroke, healthy] x Treatment [resting baseline, VMI, KMI]) between-within factorial ANOVA.

There was no significant main effect of treatment,  $F(2, 18) = .118, p = .847$ . Neither was there a significant main effect of group,  $F(1,18) = 0.0007, p = .932$ , nor was there a significant interaction effect of training and group,  $F(2, 18) = .031, p = .97$ . The mean values are shown in table 4:5. These results suggested that the changes in EMG signal during MI were not connected to the SD of the signal. The mean SDs for both groups in these conditions is shown in figure 4:7. In addition, the mean EMG values for the stroke group APB are plotted alongside their SDs in figure 4:8. It can be

seen that there is very little change in the SD throughout the conditions.

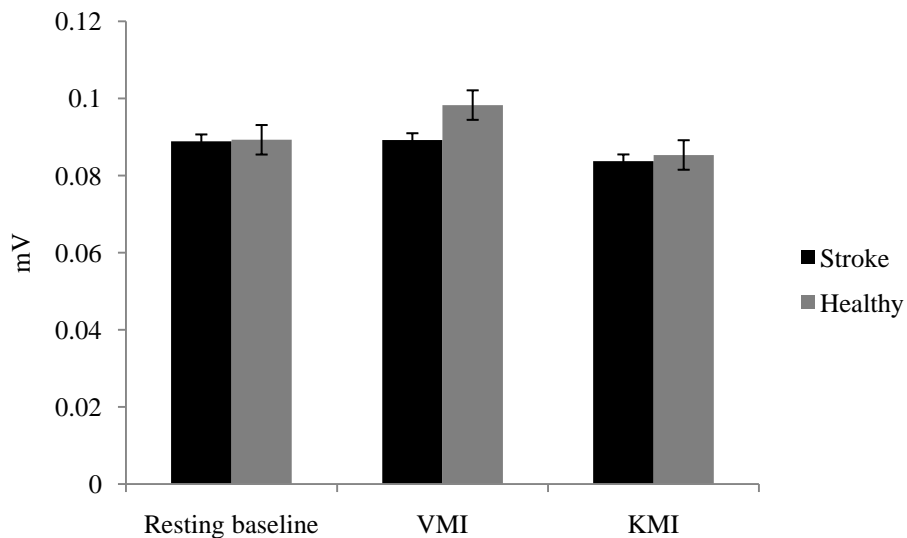


Figure 4:7. Mean SDs during resting baseline, VMI and KMI for APB

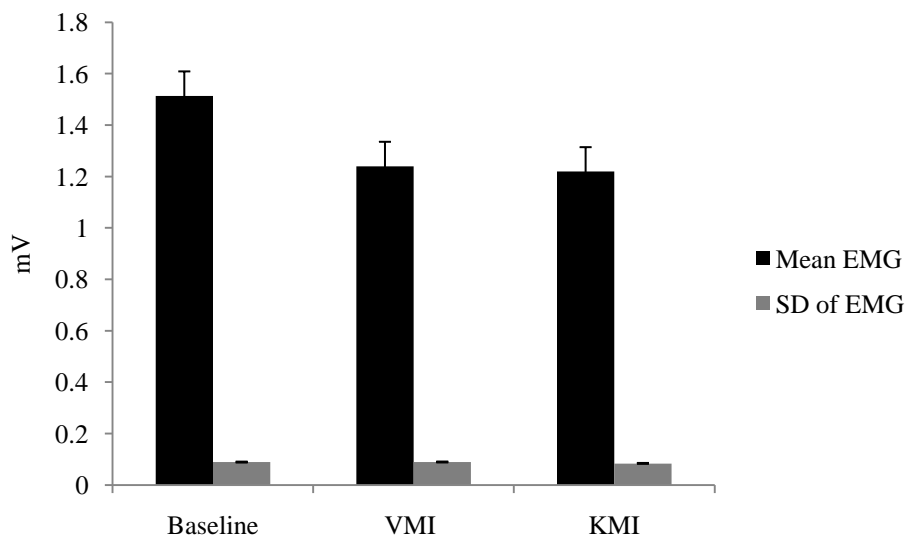


Figure 4:8: Mean EMG levels plotted against SDs of EMG for APB

#### 4.7.9 Mean SDs of thumb path during resting baseline, VMI and KMI.

In this experiment the movement of the thumb had been controlled for using an

electrogoniometer. In order to measure how much movement had taken place in the thumb during the resting baseline and MI treatment, mean SDs of the thumb path were calculated. The mean SDs for the stroke group thumb path were  $1.16^{\circ}$  for the resting baseline,  $0.97^{\circ}$  for VMI and  $0.68^{\circ}$  for KMI. Mean SDs for the healthy group's thumb path were  $2.19^{\circ}$  for the resting baseline,  $0.8^{\circ}$  for VMI and  $0.66^{\circ}$  for KMI. These measurements are shown in figure 4:9.

The SDs of the thumb angle for the stroke group were compared for significant difference using a one way repeated measures ANOVA (Resting baseline x VMI x KMI). It was found that there were no significant differences between any of these conditions,  $F(2, 9) = .017, p = .983$ .

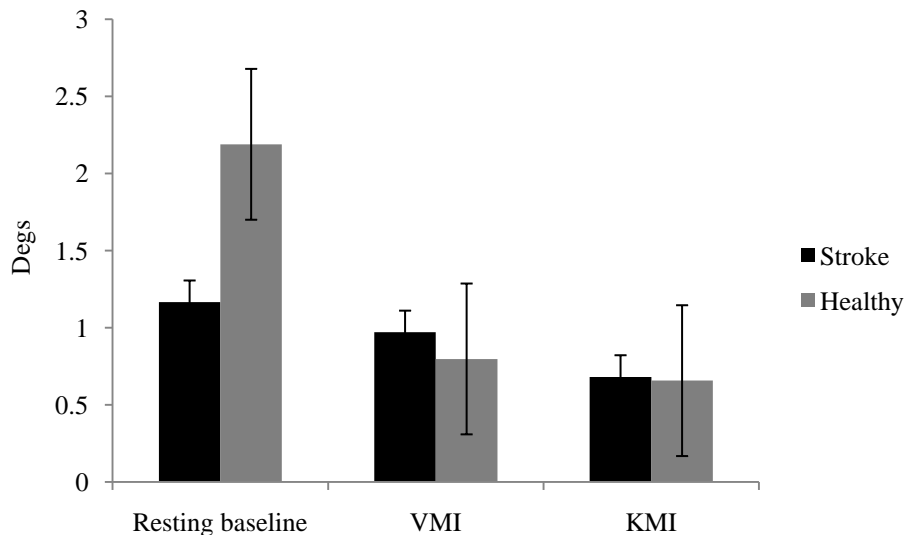


Figure 4:9. SDs of thumb path for the stroke and healthy group



**4.7.10 Correlation between APB and ADM in the stroke and healthy groups during baseline resting period, VMI and KMI.**

Table 4:6

*Correlation Coefficients (r) for APB and ADM for the Stroke Group*

		Relaxation		VMI		KMI	
		APB	ADM	APB	ADM	APB	ADM
Relaxation	APB	-	.686*	.870**	.581	.940**	.624
	ADM	.686*	-	.596	.754*	.648*	.774**
VMI	APB	.870**	.596	-	.748*	.981**	.729*
	ADM	.581	.754*	.748*	-	.715*	.987**
KMI	APB	.940**	.648*	.981**	.715*	-	.720*
	ADM	.624	.774**	.729*	.987**	.720*	-

\* $p=.05$  \*\*  $p=.01$

Table 4:7

*Correlation Coefficients (r) for APB and ADM for the Healthy Group*

		Relaxation		VMI		KMI	
		APB	ADM	APB	ADM	APB	ADM
Relaxation	APB	-	-.157	.850**	.271	.857**	-.066
	ADM	-.157	-	-.126	.588	-.156	.887**
VMI	APB	.850*	-.126	-	.128	.971**	-.001
	ADM	.271	.588	.128	-	.203	.731*
KMI	APB	.857**	-.156	.971**	.203	-	.042
	ADM	-.066	.887**	-.001	.731**	.042	-

\* $p=.05$  \*\*  $p=.01$

Clinical literature has suggested that people with stroke may find it difficult to produce compartmentalized muscle contractions (Gjelsvik, 2008). It was therefore decided to test how far both groups were able to fractionate the EMG activity in the

APB and ADM during the resting baseline and MI conditions. Pearson's product moment correlation coefficient ( $r$ ) was calculated for the EMG activation of the APB and ADM muscles for the stroke and healthy groups during the initial relaxation, VMI and KMI conditions. The key question here was the degree to which correlations could be found between EMG activation in APB and ADM. Table 4:6 shows that for the stroke group there were six occasions on which the EMG activity in APB and ADM showed a positive correlation that was statistically significant. Table 4:7 shows that, for the healthy group, there were no significant correlations between activation in APB and ADM. As table 4:7 shows, the correlations were sometimes very weak and frequently negative. The correlation coefficients were then compared for significant difference using a 2 x 3 (Group [stroke, healthy] x Treatment [initial resting baseline, VMI, KMI]) ANOVA. There was a significant main effect of group,  $F(1,2) = 113.91$ ,  $p = .009$ , but no significant main effect of treatment,  $F(2,2) = 2.34$ ,  $p = .3$ . This indicated that the correlations of APB and ADM EMG were significantly stronger in the stroke group.

#### 4.7.11 Results for abductor digiti minimi (ADM).

Table 4:8

*Mean (SD) Values for EMG (mV) for ADM*

	Stroke	Healthy
Resting baseline	0.962 (0.131)	0.306 (0.063)
MVC 1	0.853 (0.141)	0.614 (0.184)
VMI	0.607 (0.04)	0.299 (0.068)
Post-VMI rest	0.622 (0.214)	0.322 (0.15)
Post-VMI MVC	0.874 (0.159)	0.648 (0.196)
KMI	0.612 (0.047)	0.291 (0.041)
Post KMI rest	0.606 (0.239)	0.289 (0.122)
Post KMI MVC	0.837 (0.157)	0.662 (0.219)

#### 4.7.12 Resting baseline, VMI and KMI conditions.

As for APB, it was of interest to find out if there was any difference in EMG

for the ADM muscle for the resting baseline and MI conditions. Furthermore, it was wished to know if there were any differences between the stroke and healthy groups. The mean levels of EMG activation for ADM during the resting baseline and VMI and KMI conditions were therefore also compared using a 2 x 3 (Group [stroke, healthy] x Treatment [resting baseline, VMI, KMI]) between-within factorial ANOVA. The mean (SD) values of ADM during all conditions can be seen in table 4:8.

There was a significant main effect of group,  $F(1, 18) = 4.697, p = .044$ . A one way ANOVA was therefore conducted on the factor 'group'. This showed that during the resting baseline, the stroke group had a significantly higher level of EMG activation,  $F(1, 18) = 11.719, p = .003$ . However, during the VMI condition the EMG levels of the stroke and healthy group were not significantly different,  $F(1, 18) = 1.950, p = .180$ , nor were they during the KMI condition,  $F(1, 18) = 2.192, p = .156$ . This is shown in figure 4:10.

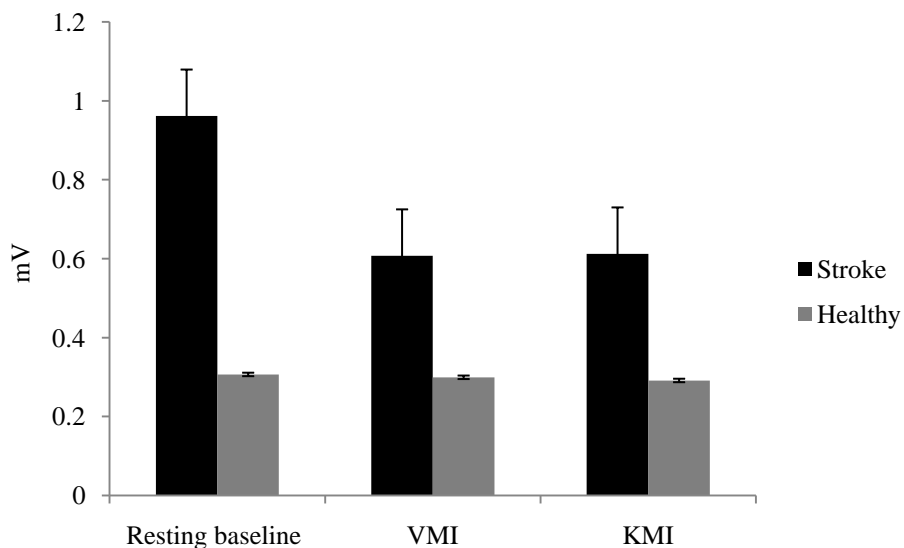


Figure 4:10. Mean EMG for ADM during resting baseline, VMI and KMI

There was, in addition, a significant main effect of treatment,  $F(2, 18) = 5.738, p = .0019$ . A one way ANOVA was therefore conducted on the stroke group alone for the factor 'treatment'. However, there was no significant difference between the treatment conditions,  $F(2, 9) = 1.064, p = .359$ . The same analysis was carried out for the healthy group alone and no significant difference was identified here either,  $F(2, 9)$

= .011,  $p = .989$ .

Further post hoc testing was carried out to compare the EMG levels for the stroke group during the resting baseline and each of the MI conditions separately using a two tailed paired samples  $t$  test. This showed that there was a significant difference for the stroke group between the resting baseline condition (0.962mV) and the VMI (0.607mV) condition,  $t(9) = 2.56$ ,  $p = 0.03$ . In addition, there was a significant difference between the resting baseline condition and the KMI condition (0.612mV),  $t(9) = 2.66$ ,  $p = 0.025$ . In both cases these results showed that there was significantly lower EMG activity in ADM during VMI and KMI, compared with the resting baseline condition. These relationships are also shown in figure 4:10.

However, post hoc testing of the healthy group did not reveal any significant difference between resting baseline (0.306mV) and the VMI condition (0.299mV)  $t(9) = 0.107$ ,  $p = .917$ , nor between resting baseline and the KMI (0.291mV) condition  $t(9) = 0.436$ ,  $p = .673$ .

In addition, there was a significant interaction effect of training and group,  $F(2, 18) = 5.053$ ,  $p = .028$ . As figure 4:10 shows, there was a greater reduction in EMG activation between resting baseline and VMI and resting baseline and KMI for the stroke group than the healthy group.

#### ***4.7.13 Resting baseline compared with post-VMI resting and post-KMI resting for ADM.***

The reasoning here was the same as for APB. It was of interest to know whether there existed a difference between the initial baseline resting period and the EMG readings taken during the resting periods following the two MI treatment conditions. The mean levels of EMG activation for ADM during the resting baseline and during rest following VMI and KMI were compared using a 2 x 3 (Group [stroke, healthy] x Treatment [resting baseline, VMI, KMI]) between-within factorial ANOVA.

The mean (SD) values can be seen in table 4:8. There was a significant main effect of treatment  $F(2, 18) = 3.928$ ,  $p = .048$ . A one way ANOVA was therefore carried out on this factor. The results for the resting baseline were significant,  $F(2, 18) = 11.719$ ,  $p = .003$ , however the results for the post-VMI relaxation condition were not

significant,  $F(2, 12) = 1.566, p = .227$ , nor were those for the post-KMI relaxation condition,  $F(2, 12) = 2.188, p = .156$ . These results are shown in figure 4:11.

In this comparison, there was also a significant main effect of group,  $F(1,18) = 4.528, p = .047$ . A further ANOVA was therefore conducted on the individual groups. The results for the stroke group were not significant,  $F(2,9) = 1.042, p = .367$ , neither were the results for the healthy group,  $F(2, 9) = .035, p = .965$ .

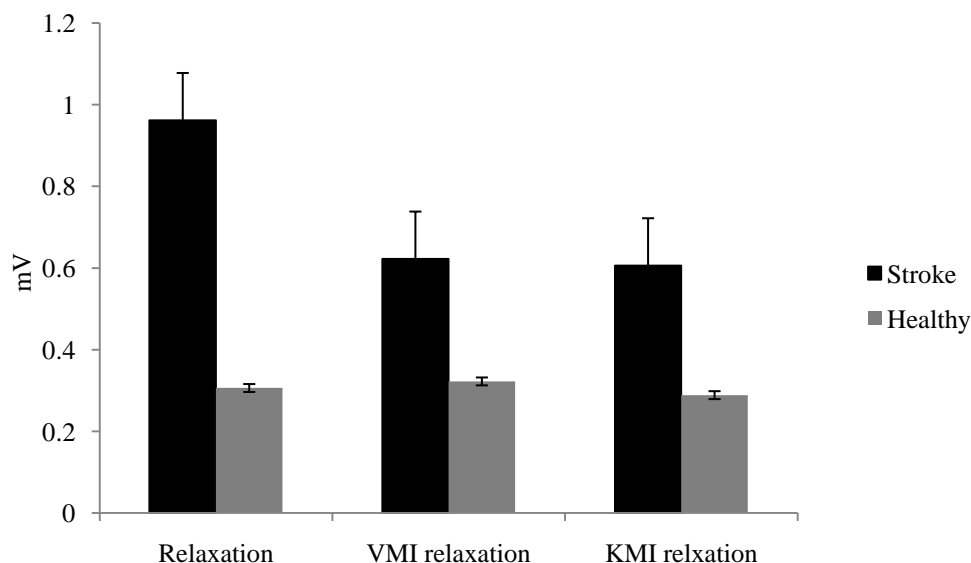


Figure 4:11. Mean EMG for ADM during the resting baseline, post-VMI relaxation and post-KMI relaxation conditions.

However, when the means for the resting baseline condition of the stroke group (0.962mV) were compared with the means for resting following VMI (0.622mV) with a paired samples  $t$  test, the results were significant,  $t(9) = 3.599, p = .003$ . Similarly, the difference between the baseline resting condition and relaxation following KMI (0.612 mV) was found to be statistically significant,  $t(9) = 4.034, p = .003$ . When the same comparison was made for the healthy group it was found that there was no significant difference between the ADM EMG level at resting baseline (0.306mV) and resting following VMI (0.322mv),  $t(9) = 1.068, p = .313$ . Neither was there a significant difference between the resting baseline EMG and the post-KMI

relaxation EMG (0.289 mV),  $t(9) = 1.481, p = .177$ .

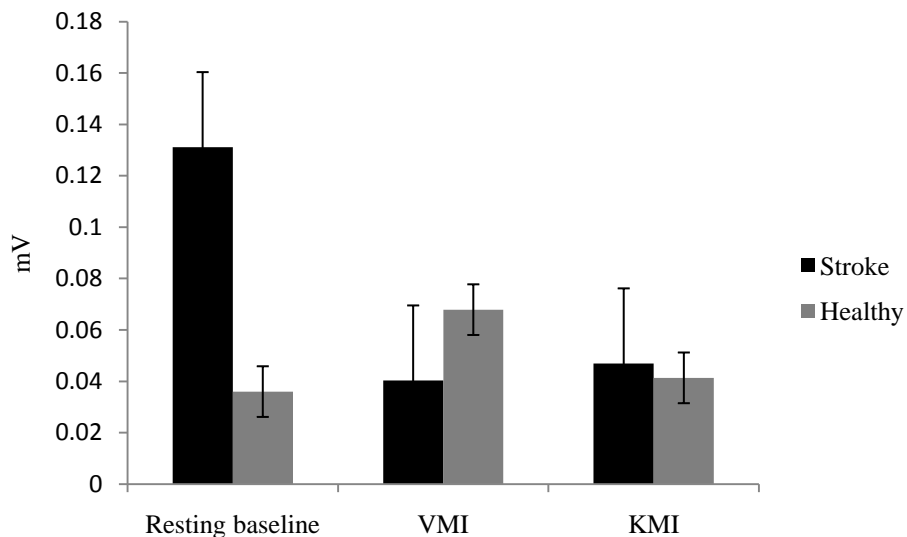
There was also a significant main effect of group,  $F(1,18) = 4.528, p = .047$ . This showed that the stroke group had significantly higher levels of EMG activation across all conditions. In addition, there was a trend towards significance for the interaction of training and group,  $F(2, 18) = 3.846, p = .051$ . Figure 4:11 shows that the MI conditions had a stronger effect on the stroke group.

#### **4.7.14 SDs of ADM during resting baseline, VMI and KMI.**

It was interesting again to pose the question if the reduction in EMG activation during the MI conditions was related in any way to a reduction in the variability of the EMG signal. As before, for APB, the SDs of the mean EMG signal were tested using a 2 x 3 (Group [stroke, healthy] x Treatment [resting baseline, VMI, KMI]) between-within factorial ANOVA.

There was a significant effect of treatment,  $F(2, 18) = 6.046, p = .05$ . A further repeated measures ANOVA was therefore carried out on the stroke group alone for this factor by itself. It was found that for the stroke group the differences between SDs for resting baseline (0.131mV), VMI (0.04mV) and KMI (0.047mV) were statistically significant,  $F(2, 9) = 6.36, p = .028$ . Post hoc testing was carried out using a paired samples two tailed  $t$  test. This showed that the SD during the resting baseline condition was significantly higher than during VMI,  $t(9) = 2.671, p = .037$ , in addition SD during resting baseline was also significantly higher than during the KMI condition,  $t(9) = 2.45, p = .026$ . A repeated measures ANOVA was also conducted on the healthy group for the single factor treatment. Here, it was found that there were no significant differences for any of the treatment conditions,  $F(2,9) = 1.618, p = .233$ .

There was no significant effect of group for the comparison of SDs,  $F(1,18) = 0.618, p = .442$ . It will be noted that the findings for ADM are in contrast with those for APB. Here it was found that there was neither a significant effect of treatment, nor of group. The results are illustrated in figure 4:12.



*Figure 4:12.* SDs of EMG activation during resting baseline, VMI and KMI conditions for ADM

#### **4.8 Discussion**

This experiment aimed to test the hypothesis, based on Jeannerod's (2006) model of motor simulation, that MI would not be linked to higher levels of EMG activation in the APB muscle of healthy individuals during any of the experimental conditions. This was on the basis that MI should, according to the motor simulation analysis, involve a simultaneous suppression of the motor command at the spinal or supra-spinal level. Furthermore, it was hypothesized that the results might differ in a stroke population, and that there may be a higher EMG in APB for this group during MI practice. This was reasoned on the basis that one aspect of the post-stroke syndrome is a reduced ability to inhibit superfluous movement. In addition, a further hypothesis was made that any augmented EMG signal would be greater during KMI, on the basis that KMI may be more effective at promoting cortical excitability than VMI (Stinear et al., 2006). The first part of this discussion examines the separate results from each inferential statistical test. This is followed by a discussion of these results in relation to the experimental hypotheses.

##### **4.8.1 APB during resting baseline, VMI and KMI.**

First, the resting baseline measurement for the APB muscle, when compared

with the VMI and KMI conditions, showed that there was no significant effect of treatment, suggesting that the VMI and KMI conditions had had little effect on the EMG levels. However, a post hoc test was carried out on the EMG levels of the stroke and healthy groups individually to compare levels of EMG activation at resting baseline with VMI and KMI taken in isolation. It was found that in the stroke group the mean EMG activation level during KMI was significantly lower than that for the resting baseline. However, the EMG signal during VMI was not significantly different to the resting baseline EMG. There were, in addition, no significant differences for the healthy group between the resting baseline and KMI, nor was there any significant difference between resting baseline and VMI.

The drop in EMG levels during KMI for the stroke group was unexpected. This surprising finding was of interest as it seemed in complete contradiction to the prediction of raised EMG during KMI for the stroke group. In addition, it would not have been predicted on the basis of the motor simulation hypothesis, where no change in EMG level would have been the expected result.

Further testing revealed that there was a significant main effect for group membership. In particular, this showed that there was a higher level of EMG activation in the stroke group during the initial resting baseline, which was highly significant ( $p = .006$ ). This increased magnitude of EMG was conserved across the MI conditions. However, the significance of these differences when the stroke and healthy groups were compared was lower than for the baseline resting condition.

It seemed, then, that one of the initial hypotheses had been partially supported. This was because there was very little difference for the healthy group APB across any of the three conditions of resting baseline, VMI or KMI. This would suggest, in line with the predictions of the motor simulation hypothesis (Jeannerod, 2006), that MI was not linked to any differences in EMG level for the neurologically intact individuals. In addition, since it was found that EMG activity was consistently higher for the stroke group across all conditions, including the resting baseline, it seemed that the presence of stroke, rather than the use of MI training, was the key factor in driving this raised EMG. This would seem in contradiction to another of the experimental hypotheses, namely that MI might lead to an augmented EMG signal in the stroke



patients. Furthermore, since the EMG values for VMI and KMI in the stroke group were actually quite close, it seemed that the hypothesized advantage for KMI in promoting EMG activation for the stroke group did not, in fact, exist. As stated above, the opposite appeared to be the case, as EMG was significantly lower during KMI when compared with resting baseline.

#### **4.8.2 ADM resting baseline, VMI and KMI.**

In this study EMG readings were also taken of the ADM muscle. The focus of all the MI treatment was always on the APB muscle. It was of interest, therefore, to compare the level of EMG activation during each of the resting and imagery conditions with ADM in order to test the muscle specificity of any effects identified. In this sense, ADM acted as a control muscle.

The EMG signal in ADM was also measured for both the stroke and healthy groups during resting baseline, VMI and KMI. As for APB, there was a significant effect of group. This showed that there was significantly higher EMG activation for the stroke group for ADM. However, post hoc testing showed that there was actually only a significantly higher level for the stroke group during the resting baseline condition and that the significant difference between the groups disappeared once MI was commenced. Furthermore, when resting baseline was compared directly with the VMI condition for the stroke group it was found that EMG levels were significantly lower during VMI. It was also found that EMG levels in the stroke group ADM were significantly lower during KMI when compared with the resting baseline. The same was not true for the healthy group, where the EMG levels in ADM were actually very close across all of the three conditions.

These findings once again seem supportive of the hypothesis that in a healthy population there would be no increase in EMG levels during MI. However, there is no support given to the hypothesis that MI, and KMI in particular, might promote a higher EMG signal in the stroke group. The findings, once again, seem to show that the opposite might be the case, as EMG levels for the stroke group ADM actually dropped significantly during MI when compared with resting baseline, rather than rising. Indeed, for this control muscle, the drop in EMG activation was significant for both KMI and VMI, while it was only significant during KMI for the APB muscle that

was the focus of the MI treatment. It was also found for ADM that the differences in EMG activation between the stroke and healthy groups were only significantly different during the resting baseline condition. However, once MI started, the drop in EMG activation meant that the differences between the stroke and control groups were no longer significant.

#### **4.8.3 APB and ADM resting baseline, post-VMI resting and post-KMI resting.**

As shown above, it had been hypothesized that MI would have no facilitatory effects on the healthy participants, while it might lead to a heightened EMG reading in the stroke group. In this experiment the participants were required to rest the muscles following each MI task, and the new resting EMG levels were recorded, giving post-VMI and post-KMI resting measures. It was of interest, therefore to compare these resting values to see if there was any effect on EMG levels when resting following MI.

For the APB muscle it was first found that the level of EMG activation was greater throughout the initial resting baseline, the resting period following VMI and the resting period following KMI for the stroke group. An initial attempt to identify any differences between these three conditions seemed to suggest there was no difference based on the use of MI prior to resting. However, when each condition was compared directly with the resting baseline a different picture began to emerge. Here, it was found that, for the stroke group, the post-VMI and post-KMI resting EMG levels were both significantly lower than during the resting baseline period. In an interesting finding, it was also found that the EMG levels for APB in the healthy participants were also significantly lower while resting following VMI, when compared with the EMG readings for the resting baseline condition. The post-KMI resting EMG levels were, however, not different to the resting baseline for the healthy group. For the APB muscle, it was found that the resting baseline following VMI was significantly lower than that during the initial resting baseline. The same was true for the EMG levels during the post-KMI resting baseline.

#### **4.8.4 ADM resting baseline, post-VMI resting and post-KMI resting EMG.**

For the ADM muscle it was found that there were no significant differences

between the stroke and healthy groups for relaxation following VMI and relaxation following KMI. This is an interesting contrast with the values recorded for the APB muscle, where, as shown above, there were still significantly higher EMG values during the relaxation periods following MI when compared with the stroke group. However, post hoc testing showed that there were highly significant differences between the resting baseline EMG for the stroke group ADM and the relaxation periods following KMI and VMI training treated in isolation. There was no such effect noted for the healthy group ADM.

These results once again appear to underline the finding that MI made no difference to the level of EMG activation for the neurologically intact population. However, they do appear to show a reduction in EMG levels while resting after MI in the stroke group. Furthermore, it should be noted that both of the post-MI resting conditions had also been followed by an MVC prior to MI treatment. One might have predicted therefore, a raised EMG signal for the stroke group on account of this exertion. This does not, however, appear to have been the case. Another point here is that the comparison between the resting baseline and the resting periods following MI for the ADM showed that the post-MI levels of EMG were lower at a highly significant level, even more so than for the APB. This is interesting because the ADM was not the target muscle in the MI tasks.

#### **4.8.5 APB MVC following resting baseline, following VMI and following KMI.**

Measurements were also taken of the MVC of the thumb prior to MI training and following the two MI treatment conditions. For the APB muscle, it was found that the mean levels of EMG activation were lower during the post-VMI and post-KMI MVCs than during the initial pre-MI MVC. Thus, MI does not seem to have promoted any heightened activation during the MVC conditions.

It is also of interest to note here that there were no significant effects of group during these MVC conditions for APB. This would suggest that the significantly higher levels of EMG for the stroke group that had been observed for the other conditions were not present once MVC had taken place.

#### **4.8.6 SDs of APB and ADM EMG during resting baseline, VMI and KMI.**

It is a commonplace that the EMG signal is particularly noisy (Kamen & Gabriel, 2010; Latash, 2008). It has also been found that in patients with another upper motor neuron disease, multiple sclerosis, there is increased variability in the EMG signal (Dorfman, Howard, & McGill 1989). It was interesting to ask, therefore, if any of the hypothesized changes, or lack of changes, to the EMG level during MI training would have any impact on the variability of the EMG signal. It was felt that a good way to do this was by measuring the SD of the signal. This SD measurement was taken from the RMS values of the EMG signal.

There was, however, no significant difference between the SDs of EMG for the APB muscle between the baseline resting condition, the VMI condition or the KMI condition. In addition, there was no significant effect of group, indicating that there was no difference between the stroke and healthy groups. Indeed, what stood out was how close the SD values were between the two groups, with extremely high probability values for each comparison. This is interesting, as it runs against the trend noted for the EMG readings themselves, where there had been found consistently higher levels of EMG activation for the stroke group across all of the treatment conditions.

There was, however, a slightly different picture for the ADM muscle. Here, the stroke group showed a significant drop in SD during the VMI and KMI conditions, in line with the drop in mean EMG level for these conditions. It may have been the case, therefore, that for ADM the drop in EMG activity noted during the MI conditions was connected to reduced variability in the signal.

#### **4.8.7 SD of thumb path.**

It has been shown that the motor simulation hypothesis (Jeannerod, 2006) sees MI as a purely cognitive phenomenon, in which the activation of a motor image should not result in movement. In addition, it has been shown that some authors have argued that MI that involves peripheral movement is not actually MI at all (Mulder et al., 2004). It was therefore felt important to control for thumb movement in this study during MI of APB. Other authors looking at EMG in MI have simply controlled for peripheral movement through visual inspection of the participant's limb during MI treatment (Harris & Robinson, 1986; Jowdy & Harris, 1990; Lutz, 2003), verbal

prompting of participants not to move (Gandevia et al., 1997; Hale, 1982) or have simply not specified how movement was monitored (Mulder et al., 2004). The present research had the advantage of deploying electrogoniometry, allowing a more reliable and high resolution measurement of thumb movement during MI.

In order to estimate the variability of the thumb movement, the mean standard deviation (SD) of the thumb's angular path was calculated for each group. The mean SD for the stroke group across the resting baseline and the two MI conditions was 0.94°, and for the healthy group 1.21°. Thumb movement was therefore minimal for both groups, and was actually lower for the stroke group. This suggests that the increased EMG signal noted for the stroke group was not linked to any superfluous movement in the thumb.

#### **4.8.8 Correlation of APB and ADM during resting baseline, VMI and KMI.**

In one sense this variable had a slightly oblique relationship to the central research question. What was being examined here was the coupling of EMG activation in the APB and ADM muscles. The main question was if MI had any effect on this coupling. Table 4:6 shows that there were six statistically significant correlations between the EMG activity in ADM and APB for the stroke patients throughout all the conditions. There were no such correlations, however, in the healthy group, as shown in table 4:7. Indeed, the correlations were generally very low indeed and often negative. When these values were compared it was found that the factor of group membership was highly significant, suggesting that the presence of stroke was the most important factor in these strong correlations, whereas the presence or absence of MI did not seem to have any effect. It seems reasonable, therefore, to relate these findings to the upper motor neuron syndrome. In addition, it may be important to take into account the distinctive patterning of the cortical control of the intrinsic hand muscles. These points are considered further on in this discussion.

#### **4.9 General discussion of findings.**

##### ***4.9.1 Raised EMG levels in hemiplegia.***

One of the findings which stands out in this experiment is the consistently raised level of EMG signal for the stroke group in both the APB and the ADM

muscles. This is certainly in harmony with other studies looking at EMG in neurological injury. Kallenberg and Hermens (2010), for example, placed sensors on the affected bicep of eighteen individuals with stroke and also on twenty healthy individuals. They found that there was significantly higher EMG activation in the stroke group both during active and passive movement. Fung and Barbeau (1989), meanwhile, looked at the EMG pattern in the gait of 8 people with spasticity following spinal cord injury compared with 5 healthy controls. These authors found that the EMG activity in muscles used in locomotion continued in a heightened state during a gait phase in which they would normally be dampened in neurologically intact individuals. In addition, Fleuren et al. (2006) found that the rectus femoris of the affected leg in nineteen stroke patients showed higher levels of EMG activity throughout active extension, when compared with the non-affected leg. In addition, these authors found that this pattern continued even as the knee was moving back into flexion, an action in which the muscle would not have been the prime mover. Evidence suggests, therefore, that one consequence of neurological injury may be augmented EMG signalling.

This finding also makes sense in the light of the features of the UMN syndrome, which have been outlined in the introduction to this chapter. As shown above one of these features, spasticity, has been described as the central nervous system driver of muscle hypertonicity (Carr & Shepherd, 1998). It is perhaps useful to recall here that one modern definition of this controversial phenomenon encompasses all sporadic or persistent over-activity in the muscle linked to an upper motor neuron lesion (Johnson, 2004). The finding of raised EMG in the two intrinsic hand muscles measured in the present research seems readily explainable, therefore, in the light of the post-stroke syndrome.

#### ***4.9.2 Lowered EMG in hemiplegia during MI.***

A finding of the present research which seems harder to explain, however, was the drop in EMG activity for the stroke patients upon the commencement of MI. This was in complete contradiction to the experimental hypothesis, which had predicted an increase in EMG during MI for the stroke group. This seems to require further explanation when it is considered that, for the hemiplegic APB muscle that was the

actual target of the MI treatment, the reduction in the EMG signal during KMI alone was sufficient to eradicate the significant difference between the stroke and healthy group on this measurement. As shown above, this would not necessarily have been predicted by the findings of Stinear et al. (2006), who found that only KMI, as opposed to VMI, promoted raised cortical excitability. On this basis it had in fact been initially hypothesized that only KMI would be linked to heightened EMG signal in the stroke group. The opposite, however, seems to have been true for the hemiplegic APB. In addition, for the control ADM muscle, the reduction in EMG activation was enough to make the difference between the stroke and healthy group non-significant during both the VMI and KMI conditions.

Such findings have been reported previously, although sometimes as anomalous outlying cases. Yue and Cole (1992), for example, reported that one member of their group showed a decrease in the EMG levels in a hypothenar muscle following mental training of that muscle. Bakker et al. (1996), meanwhile, noted a drop in EMG activity in the bicep of healthy individuals during imagery based on a stimulus proposition, which might be taken as equivalent to VMI, as compared with a resting baseline EMG. However, to the best of the author's knowledge this has not been a finding in other studies and has not, to date, been systematically examined.

It should also be noted that these results were not found in the healthy group. Here, as had been hypothesized, MI did not seem to be associated with any effect on EMG levels. Indeed, the EMG levels in the healthy group generally showed very little change throughout any of the experimental conditions.

A working hypothesis may be that the effect of MI was to reduce the EMG activation in the stroke group, thus bringing the muscle more into line with the levels of activation that would be found in the non-hemiplegic hand. For the APB muscle, this effect seems to have occurred only during KMI. For the ADM muscle, however, the elimination of the significant difference between the stroke and healthy group took place during both MI conditions. This latter finding is discussed further in the next paragraph.

It should be noted, first of all, that ADM was the control muscle. When the participants were being asked to imagine an action it was of the thumb abducting,

meaning that the target of MI was actually APB. If the tentative hypothesis put forward above, that MI worked by actually reducing and therefore partially normalising the EMG activity, then one might have predicted that the effect would have therefore been noted more consistently in the targeted APB.

Bakker et al. (1996) have drawn on a concept elaborated by Bernstein (1967) to explain such apparently non-specific effects of MI. This author had argued that the motor image would not represent in any way the specific muscles and joints involved in a movement. Rather, Bernstein (1967) argued, what was represented was “a very abstract motor image of space” (p. 49). Bakker et al. (1996) concluded on this basis that the motor image may not contain detailed information about the precise levels of EMG activity in particular muscles. It could be the case in the present research, therefore, that MI had a more general effect at the level of the whole hand, rather than a specific effect targeting only APB.

The findings regarding the reduction in EMG for the stroke group when using MI are reinforced by the post-MI resting conditions. Here, one surprising finding was that, even for the healthy group, there was a significantly lower EMG in APB when resting following EMG when compared with the initial baseline resting condition. While for the stroke group the APB level of activation remained significantly higher than the APB of the healthy group when resting following MI, for the ADM muscle the significant differences disappeared between the healthy and stroke group during resting following both KMI and VMI. This latter finding would lend support to the argument being developed here that MI worked to somehow “normalize” the level of EMG activation in the stroke group. Furthermore, this proposed effect once again appears to have been felt more strongly in the control muscle, which was not the target of MI treatment.

In addition to the above findings it was found that, for the APB muscle, the MI conditions did not have seem to have any facilitatory effect on the EMG levels during MVC. Here, it was found that the level of EMG was lower during MVC following both MI conditions than it had been during MVC following the resting baseline condition. This would seem to once more confirm that MI generally may have had an effect on the EMG activation, though the effect seemed to have been one of



dampening EMG activity.

If it was the case that MI acted to somehow reduce the EMG levels in the hemiplegic muscles, it seemed important to investigate how this might have taken place. This was first of all done by referring to data from some of the other variables measured in this experiment. The first of these to be looked at was the SD of the EMG signal in each condition. This is discussed further below.

#### ***4.9.3 Variability in the EMG signal and MI.***

It has already been suggested that one might have expected a reduction of the variability of the EMG signal, as measured by SD, as the mean EMG level was reduced. Indeed, it could feasibly have been the case that the reduction in mean EMG level was a function of the reduction in the SD of the signal. Based on this it could be hypothesized that the suggested ‘normalization’ effect of MI worked by reducing the variability in the EMG signal. However, this does not seem to have been the case, at least for the APB muscle. Referring to the data for the SDs of EMG in APB it is first of all clear that, despite the reduction in EMG signal which began during the MI conditions, the SDs of the signal showed very little change between the resting baseline and the MI conditions. This is shown in figure 4:8, where the EMG and SD values for each condition for this muscle are plotted alongside each other. Furthermore, while there was a statistically significant correlation between the mean EMG level and the SD of the mean EMG for the APB muscle during the resting baseline condition, the correlation was no longer significant during the MI conditions. As has been shown, this change could only have been connected to the reduction in the EMG signal, as the variability of the signal, as measured by its SD, changed very little.

It is of further interest to note that there were no significant differences based on group for the APB muscle. This is because, as shown in figure 4:7, the SDs of the EMG signal in all conditions were actually very similar for the stroke and healthy group, despite the divergence of the mean EMG levels. In general terms, this suggests that the variability of the signal in the stroke group was no higher than that for the healthy group, which is in contradiction to some of the evidence from other studies of neurological populations mentioned above. Furthermore, it suggests that the proposed

dampening effect of MI did not work by reducing the SD of the EMG signal, but by reducing the EMG level itself.

The pattern of SD was slightly different for the ADM muscle. It was found that the SD was significantly lower during both of the MI conditions when compared with the resting baseline condition. This stands in contrast to the findings for APB and suggests that, for ADM, there may have been a connection between the reduced EMG signal during the MI conditions and a reduction in the variability of the signal.

#### ***4.9.4 Superfluous movement and heightened EMG.***

Another possibility considered was that the heightened EMG activity in the stroke group might be related to a failure to suppress thumb movements in these individuals. It might follow that MI helped reduce such superfluous movement, leading to a reduction in EMG. However, further analysis showed that this did not seem likely.

Thumb movement was controlled for using an electrogoniometer. It was felt that SD of the mean thumb angle was a useful measurement in this respect. This was because a mean value alone would simply have given an impression of the average magnitude of the joint angle. SD, however, indicated the degree to which this value changed over the period of measurement, thus giving a better indication of the movement of the joint. Mean SD was measured for the resting baseline, VMI and KMI conditions.

As shown above, the movement was minimal in both groups and was lower for the stroke group. In addition, there were no significant differences identified for the stroke group for thumb SD between any of the three experimental conditions. It was felt, therefore, that the presence of superfluous thumb movements was an unlikely explanation for the raised EMG levels in the hemiplegic APB. As a consequence it was therefore unlikely that its subsequent reduction was a result of MI somehow reducing that movement.

#### ***4.9.5 Possible explanations for the raised EMG signal in the stroke group.***

Rothwell (1987) has suggested that such increases in EMG signal are likely to have a neural origin in stroke patients. This author argued that the descending signal to the lower motor neuron is weaker and shows higher levels of variability following

stroke. As a result, he suggested, the output of the motor unit is correspondingly attenuated and more variable. This may then lead to a failure to produce a coordinated contraction of the motor unit. The overall consequence for the patient may be an inability to exert maximal force from the affected muscle. In order to produce a given amount of force there would need to be a central increase in corticofugal drive and it will be recalled that one feature of the post-stroke syndrome does indeed appear to be increased cortical excitability (Nudo, 1999). At the periphery, an increase in force production would be achieved for the patient by more motor units being recruited. Since the EMG signal is actually based on the summed action potentials of the motor units (Kamen & Gabriel, 2010), then the higher recruitment of motor units could well result in the raised EMG signal found in this experiment. Furthermore, the intrinsic hand muscles are estimated to have only about 100 motor units each (Kamen & Gabriel, 2010; Rothwell, 1987). This means that they rapidly reach the maximum force capacity possible via increased motor unit recruitment (Rothwell, 1987). From this point force increments can only take place by raising the firing rate of the previously recruited motor units, thus further increasing the EMG signal (Kamen & Gabriel, 2010; Rothwell, 1987).

A further factor which may need to be taken into account here is that the normal parameters used by the motor control system may become disrupted following stroke. It has been found, for instance, that the recruitment of motor neurons normally follows a size principle, with smaller motor neurons being recruited first and larger ones later (Henneman, 1985). As Kamen and Gabriel (2010) point out, this simplifies the very challenging job that the motor control system has in scaling motor unit recruitment to the force requirements of a given task. However, this process may be disrupted following stroke. On this point authors have offered contrasting views: Gjelsvik (2008) states that such disruption is an issue for the hemiplegic patient, although Rothwell (1985) has argued that the recruitment pattern is typically unaffected in UMN lesions. This may, therefore, remain an open question. However, Henneman (1985, p. 107) has shown that, despite the recruitment principle holding at a systemic level, at the level of individual motor neurons the process is marked by a degree of probability which reflects “dynamic, state dependent processes”. This may

be linked, for example, to the distance traversed by the 1a fibre to the spinal motor neuron or to the narrowness of the fibre diameter (Henneman, 1985). Since a monosynaptically innervated distal hand muscle is some distance away from the spinal cord and also has a narrow fibre diameter, such variability is likely to be a factor even in the healthy muscle. Rothwell (1985) presents evidence, in addition, that the abductor pollicis (he does not specify whether the short or long muscle) is one of the muscles in humans which shows task dependent changes in recruitment order. It would seem likely, therefore, that there could be disordered motor neuron recruitment following stroke and that this could lead to problems grading force.

In order to understand the way in which these neurophysiological factors would impact on the EMG levels noted in the stroke patients in the present study, it may be useful to consider the movement history of people with stroke. The person may, for example, begin to use the upper limbs more for stabilization of posture during locomotion (Gjelsvik, 2008). This could result in the need for higher levels of force in the hand. As shown above, this may lead to a higher EMG signal either through the recruitment of more motor units, or because of increased firing in the already enlisted motor units. Furthermore, disrupted neural transmission may well lead to problems with the orderly recruitment of motor neurons, reducing the degree of control over force production, and perhaps also leading to a raised EMG. In addition, there may be a shift away from the predominance of slow twitch, type one fibres in the intrinsic hand muscles (Gjelsvik, 2008; Rothwell, 1987). Gracies (2005), for example, has argued that there may be a general shift towards type two fast twitch fibres in affected body parts following stroke. This might also contribute to the intensifying of the EMG.

Further factors that may be connected to the UMN syndrome, and which may lead to increased EMG activation, are the changes taking place in the actual mechanical properties of the muscle due to changes in the muscle's architecture (Dietz, 2003). Gracies (2005) has pointed out that the maintenance of the joint in a shortened resting position for extended periods following stroke can lead to a number of structural changes in the muscle. These may include atrophy of and a consequent reduction in the number of sarcomeres. The extant sarcomeres may then be

reorganized into new overlapping and less contractile formations. There may also be increases in the amount of connective tissue and raised collagen in the muscle body (Gracies, 2005). Such changes lead to decreased extensibility of the muscle and therefore require more effort from the patient to move it, leading to higher levels of firing or recruitment of motor units and raised EMG.

Higher EMG levels were found consistently throughout all conditions for the stroke patients when compared with the non-stroke group. However, the highest levels of EMG were actually found during the resting baseline, prior to MVC being executed or MI treatment. Here, it is important to take into account the motor experience of the patients following their stroke, as discussed above. They may therefore have begun the study with a pre-existing history of raised EMG in the muscle.

#### ***4.9.6 Possible mechanisms by which MI could reduce the EMG signal.***

It would seem that the reduction in the EMG signal during MI was likely to have been a result of reduced recruitment and/or firing intensity of the motor units rather than any overall change in motor unit firing variability, at least in the APB. In addition, it has been shown that it was unlikely that MI worked by reducing superfluous thumb movements. An explanation needs to be sought, therefore, for the ways in which MI may have impacted upon the motor unit itself.

The starting point for the motor unit is the firing of the spinal or brainstem motor neuron, which propagates an action potential that in turn causes a muscular fibre action potential (Kamen & Gabriel, 2010; Rowland, 2000). As shown above, it is the combined activity of these action potentials that produce the motor unit action potentials, which are summed and can be recorded as the electromyogram at the surface (Kamen & Gabriel, 2010). On this basis it would seem reasonable to suppose that the effect of MI was somehow felt at the spinal level, resulting in reduced motor unit recruitment and a lower EMG signal.

Despite the fact that such spinal or brainstem activity may not seem readily accessible to cognitive processes such as MI, evidence does suggest that it may, in fact, be modulated voluntarily, a phenomenon which has been described as “cognitive penetrability” (Jeannerod, 1988, p. 3). Bonnet, Decety, Jeannerod, and Requin (1996), for example, conducted a study in which twenty healthy individuals were asked to

imagine pressing down with one foot, while EMG readings were taken bilaterally from the soleus muscles. It was found that the increase in tendon reflex activity was significantly larger in the leg which was being imaged and also in imaginary conditions involving greater force production. In addition, Gandevia and Rothwell (1987) were able to show, in an experiment on four healthy individuals using cortical electrical stimulation, that the participants were able to learn to voluntarily increase or decrease EMG activity in either ADM or APB on command. This was done in the absence of any movement or background EMG signal in the muscles, and took a maximum of one hour's training. In addition, it was found that this could not be done for the extrinsic hand muscles. Gandevia and Rothwell (1987) surmised that the changes noted in levels of muscular activation may well have been associated with alterations in spinal reflex excitability.

A number of studies have presented data suggesting that even in cases of neurological dysfunction cognition may impact upon processes which are routinely not under conscious control. Neilson and McCaughey (1982), for example, found that individuals with cerebral palsy were able, with practice, to lower the EMG levels in their affected muscles and consciously moderate the sensitivity of the stretch reflex. Latash et al. (2002), meanwhile, found that people with Down syndrome could reduce the level of co-contraction of antagonist and agonist muscles in a finger force task. These individuals learned to negatively co-vary the force production in each finger in order to produce a constant force output (Latash et al., 2002).

Furthermore, from the perspective of neurophysiology, it is now generally accepted that higher control centres can influence the reflex response and that the reflex response is also partially task dependent (Pearson & Gordon, 2000). Voluntary movement, for example, generally involves alpha and gamma motor neuron co-activation (Rothwell, 1987). The gamma motor neurons, however, are divided into active and static types, and the precise balance of their activation levels is known to be a function of the task being performed (Pearson & Gordon, 2000). Indeed, as discussed in chapter two, Rodrigues et al. (2010) have posited that KMI may operate to produce increase postural sway via selective activation of the gamma motor neuron.

Evidence that spinal reflex responses can be controlled cortically also comes

from work on the so-called “long-latency stretch reflex”, or M2 reflex (Rothwell, 1987, p. 130). This occurs following muscle stretch. An initial short EMG burst, the M1 reflex, is followed almost straight away by a longer second burst, which is the M2 reflex (Pearson & Gordon, 2000). A number of authors have shown that this reflex can be mediated cortically and controlled cognitively (Capaday, Forget, Fraser, & Lamarre, 1991; Rothwell, Traub, & Marsden, 1980). This is, then, further evidence that a cognitive phenomenon such as MI could “penetrate” spinal reflex action.

#### ***4.9.7 Correlation coefficients of APB and ADM EMG activity.***

It has been suggested that the MI training in the present study may have been effective at the spinal level by reducing recruitment of or firing rate in the motor units. This argument might be supported with reference to one of the other variables measured in the experiment. This was the correlation coefficient for the levels of EMG activation in the APB and ADM muscles. It will be recalled that six statistically significant correlations were found between the muscles in the stroke group. The healthy group, meanwhile, was distinguished by an absence of significant correlation and actually very weak and sometimes negative  $r$  values for the two muscles. In addition, the MI training was found to have no effect on the level of correlation. The presence of these strong correlations may be an example of the phenomenon of enslavement, already outlined in the introduction to this thesis, and discussed further below.

Zatsiorsky et al. (2000) argued that enslavement is likely to be of central origin. Li (2007) developed this idea, relating the phenomenon to the cortical representation of the hand. The distal muscles display high levels of divergence, so that one neuron can be implicated in the control of spatially dispersed muscle groups, and convergence, meaning that several neurons that may be distant from each other cortically can activate the same muscle group (Kandel, 2000; Lang & Schieber, 2004; Latash, 2007; Porter & Lemon, 1993). Li (2007) therefore suggested that a movement image for a single finger could potentially also activate representations for adjacent fingers, causing the enslavement effect. This would arguably be more marked in stroke, where there may be a blurring of the cortical map of a body part (Gracies, 2005).

The evidence presented above may explain the high levels of co-activation noted in the hemiplegic APB and ADM in the present study, and also its absence for the healthy participants. It has been argued in this discussion that MI may have been effective at the spinal level, dampening down motor unit activity. These results for the APB-ADM correlations might also suggest that MI did not have any effect on a process which may have had a cortical origin.

It has been argued throughout this discussion that the results for the healthy group would seem to provide support for the hypothesis of motor simulation, in so far as no change in EMG signal was noted during MI. However, it might be the case that the findings of reduced EMG signalling for the stroke group could also be explained in relation to this hypothesis. It will be recalled that Jeannerod (2001; 2006) proposed that an action representation must be accompanied by a simultaneous signal to inhibit the motor command. It could be the case therefore, that the reduced EMG signal was the result of such an inhibitory impulse. A possible spinal mechanism for this is considered below.

#### ***4.9.8 A possible mechanism for inhibition of the EMG signal during MI.***

The Renshaw cell is a glycinergic interneuron that is triggered by a collateral offshoot from the motor neuron, and is also controlled from the supraspinal level (Gracies, 2005a). Its action is inhibitory on the motor neuron and is described as “recurrent inhibition” (Alvarez & Fyffe, 2007; Gracies, 2005a, p. 563; Schwartz & Westbrook, 2000). Bonnet et al. (1996) have suggested that the Renshaw system provides a feasible mechanism by which inhibition could take place. They argue that when the motor image is propagated, then the Renshaw cell may be involved in suppressing any motor output. This then may provide a way in which MI could influence the spinal level of motor control and inhibit the EMG signal. Furthermore, this would be in keeping with the predictions made as part of Jeannerod’s (2006) motor simulation hypothesis.

In conclusion, it may be the case that the reduction in EMG during MI may well have been an example of the kind of ‘cognitive penetrability’ described for spinal reflexes in the above paragraphs. MI may have led to a reduced recruitment of motor units via an inhibitory mechanism such as that outlined above. This could then have



brought the EMG back to a more normal level. It will be recalled, for example, that the difference between EMG activation for the ADM muscle in the stroke and healthy group ceased to be statistically significant once MI commenced. Any effect of MI, however, did not seem to be modality specific, as there was very little difference between the VMI and KMI conditions. As suggested, this change in EMG may have resulted from a more general process such as increased attention being focused by the patients on their hands. Furthermore, this must have happened unconsciously, as reducing levels of activity in the muscle was not specified in the instructions given to the participants. It may indicate, therefore, that MI was somehow linked to non-conscious planning processes. This would certainly be in line with the work of authors who have developed the hypothesis of motor simulation and associate MI with a pre-execution stage of movement (Decety & Jeannerod, 1989; Decety & Jeannerod, 1996; Jeannerod, 2001; Jeannerod & Decety, 1995; Jeannerod, 2006). Overall, however, the results discussed might therefore be seen as offering support to Jeannerod's (2006) motor simulation hypothesis.

#### **4.10 Limitations of experiment three.**

##### **4.10.1 Limitations of sampling and recruitment processes.**

The stroke group was recruited from patients who had already participated in experiment two, the same limitations apply which have been discussed for that study. The snowballing approach used to recruit healthy participants also has the disadvantage of being unrepresentative of the wider population. As Patrick et al. (1998, p. 296) have noted, this recruitment technique depends upon individuals knowing each other and leads to the underrepresentation of "socially isolated" sectors. Streeton et al. (2004, p. 41) have pointed out that this approach can lead to the rapid development of "referral chains" which can actually mask the exclusion of more poorly represented groups.

Patrick et al. (1998) aver that a non-probabilistic sampling technique such as snowballing should involve the simultaneous collection of information relating to demographic factors such as educational and employment status, ethnic origin or marital status in order to check how representative the sample is. Streeton et al. (2004) echo this, commenting that the researcher should monitor the individuals recruited,

being sure to actively encourage less well represented groups to participate.

These strategies, which may have ensured a more representative sample, were not, however, used while recruiting participants to the current study. One mitigating factor here is that the focus of this research was on basic neurophysiological processes. One might predict these would vary little between neurologically intact adult humans within a particular age range. From this perspective, a more representative sample may have made little difference to the overall results of the experiment.

#### **4.10.2 Possibility of covert MI practice between experiments two and three.**

Motor imagery ability is a cognitive skill and therefore can improve with practice, or decay with lack of practice (Hall, 2002). Furthermore, the benefits of mental practice on performance decline as the time since mental practice increases (Driskell, 1994). It may therefore follow that those who continue practising MI can improve both in the ability to utilize the technique, and also in the performance of the motor skills that they are imaging.

It will be recalled that, following experiment two, individuals were informed that they might be contacted to take part in a further experiment. However, no specific instructions were given for them to desist from MI training in the intervening period. This may have therefore entered experiment three as a confounding variable, as some individuals may have continued with MI and become more adept, giving them an advantage over others prior to entering the study. It is felt, however, that this is unlikely for a number of reasons, explained in the following paragraph.

First, in experiment two, the participants were not given any instructions about how to perform MI, but were merely asked to visualise or imagine the sensation of the movement. In this sense the participants were only being asked to perform everyday cognitive tasks, albeit with more attentiveness than normal. This is in contrast, for example, to the study of Liu et al. (2004), in which participants were clearly trained how to perform and apply MI in the process of motor learning. It seems unlikely, therefore, following experiment two, that individuals would have had sufficient information regarding the use of MI to be able to continue with their own personal

practice. Second, even if they had continued to practise, the task in experiment three was slightly different to that practised in experiment two. It will be recalled in experiment two that the participants were asked to imagine or visualise the fingers and knuckles contracting, without a specific goal. In experiment three, they were required to picture or imagine the sensation of their thumb abducting against a block of wood. In addition, the earlier experiment was focused on the learning of a motor task, which was explained clearly to the individual prior to MI. However, in the third experiment, there was no explicit learning condition, the participants needed simply to watch a video, rest, listen to MI scripts and produce MVCs. In this sense they were not comparable. It is not clear, therefore, that continuing to mentally practise the task from experiment two would necessarily have a priming effect on the task used in experiment three. Furthermore, all participants were naïve as to the experimental hypotheses. It was therefore not possible for them to be guided by knowledge of any predicted outcomes. Finally, it was some months since the individuals had taken part in the earlier study, with a person who had taken part three months previously being the most recent. Since the previous study had involved a one-off session of MI training it is not felt that this could have realistically produced any bias in the outcomes of the present study. Nonetheless, this aspect should have been controlled with more care, given that the same individuals were to take part in a further experiment. This could have been done through explicit instructions to the potential participants not to use in MI in the meantime.

#### **4.10.3 The characteristics of the imagery task.**

The nature of the imagery task itself may have militated against the presence of EMG activation. Roosink and Zijdewind (2010), for example, have highlighted the importance of the participants' active engagement during imagery. These authors compared passive observation of a task and active observation, that is, observation with an instruction to imitate the task later on, with KMI and VMI. They found that only active observation led to increased EMG. In addition, only observation of a complex, as opposed to a simple, movement produced such an effect (Roosink and Zijderwind, 2010). According to these definitions, all of the imagery tasks in the present study would have been passive, as they did not involve any further instructions

beyond imaging the sensation or visualizing the action. In addition, they were all of a simple thumb abduction task. It may be the case, therefore, that an imagery task which combined instructions to act, and a training task of a more complex movement, may produce a different EMG response to that found in the current study.

#### **4.10.4 Experiment site.**

A further factor to take into consideration regarding the nature of the imagined task in the present experiment is the comparatively weak activation which would be involved in actual execution of the task. Bonnet et al. (1997), for example, found that only imagery of a strong contraction of a large prime mover muscle led to a raised EMG signal. The abduction of the thumb imagined in the present study, therefore, may have simply been too weak a movement to provoke an EMG response. It may therefore be important to consider the nature of the imagined task in future work.

A further objection may be that testing did not take place in a laboratory, but in the participants' homes. Here, it should be stated that all stimulus materials were presented in an identical way to each participant. All instructions, for example, were pre-recorded, as was the video which showed the thumb abduction task. In addition, the tasks were all presented using headphones, so that any background noise was minimized. The participants also closed their eyes throughout all of the conditions except for the initial viewing of the video. Furthermore, the participants were invited to sit comfortably at a table, the same instructions as would have been provided in a laboratory. It should be pointed out that another recent study, that of Ietswaart et al. (2011), also reported the testing of patients in their own homes. It is not felt overall, therefore, that the location of testing would have made any difference to the outcomes.

#### **4.11 Conclusion**

No evidence of raised EMG was found for neurologically intact individuals when using MI. It was found, in addition, that individuals with stroke had higher levels of EMG in intrinsic hand muscles at resting baseline. However, these levels then dropped once they started using EMG. It was argued, therefore, that the effect of MI may have been to bring the EMG activation more in line with that found in normal movement. It was noted, in addition, that the effects of MI were not confined to the target hand muscle and that none of the effects of MI seemed to be specific to the

sensory modality of the imagery used. An argument was therefore developed that MI may not work by targeting specific muscles, but may have represented a more general process of directed attention towards the affected hand by the patients. Further analysis showed that the reduced EMG was not linked to any change in variability of the EMG signal, at least for the APB muscle. It was proposed therefore that the explanatory mechanism for the reduction in EMG activity was likely to be a reduction in motor unit recruitment at the spinal or brainstem level. It would follow from this that these spinal level processes were cognitively penetrable when using MI.

**Appendix 4:1.**

***Script for resting baseline and relaxation condition for stroke and healthy group.***

“Relax, put your hands on your knees and keep your eyes shut. Relax your muscles. Breathe normally. Forget any worries. Ignore any sounds inside or outside the room. Just concentrate on your breathing”.

This was repeated three times.

***Script for MVC for the stroke group.***

“Close your eyes. Concentrate on your breathing, breathe normally. Ignore any sound inside or outside this room, just concentrate on your breathing. Ignore any worrying thoughts.”

“With your eyes closed, and with your hand resting on the table, open the thumb of the hand which has been weakened by the stroke and press it against the block of wood as hard as you can, then relax again”

These last instructions were repeated four times.

***Script for MVC for the healthy group.***

“Close your eyes. Concentrate on your breathing, breathe normally. Ignore any sound inside or outside this room, just concentrate on your breathing. Ignore any worrying thoughts.”

“With your eyes closed, and with your hand resting on the table, open the thumb of your non-dominant hand and press it against the block of wood as hard as you can, then relax again.”

These last instructions were repeated four times.

***Script for VMI task.***

“With your eyes closed, remember the video of the thumb moving which you watched earlier. Imagine you are watching the video again. Remember what you saw. Remember watching the thumb pressing the block of wood. Just imagine that you are watching the video again. Then relax.”

These instructions were repeated four times.

***Script for KMI task for the stroke group.***

“With your eyes closed, imagine the thumb of the hand weakened by the

stroke. Don't move the thumb, but concentrate on the sensation of the muscles and joints and imagine the thumb once again pressing the block of wood. Don't move the thumb, just imagine the movement. Think of the pressure of the wood against your thumb. Then relax."

These instructions were repeated four times

***Script for KMI task for the healthy group.***

"With your eyes close, focus on the thumb of your non-dominant hand. Don't move the thumb, but concentrate on the sensation of the muscles and joints and imagine the thumb once again pressing the block of wood. Don't move the thumb, just imagine the movement. Think of the pressure of the wood against your thumb. Then relax."

This was repeated four times.

## Chapter Five

### **Novel Contribution and Clinical Relevance of the Thesis, General Discussion of Limitations and Proposals for Future Work.**

#### **5.1 The Novel Contributions of the Thesis**

Experiment one presented in this thesis provided an original insight into the use of MI in the treatment of the hemiplegic hand. It was found that MI using a kinaesthetic sensory modality was associated with a faster overall mean movement time when performing a grasping action with the hemiplegic hand. This was interesting, as the script used for the KMI task was aimed at the activation of a specific muscle group, and did not refer to the speed of hand movements. It may be the case, therefore, that the effect of KMI was not necessarily manifest at the level of activation of a particular muscle. Rather, the effects may be to stimulate attention at the level of “early-selection”, which involves attending to a stimulus prior to careful analysis of its explicit content (Gazzaniga et al., 1998, p. 211).

The novel contribution of experiment two was to compare the effects of VMI and KMI on the activation of muscles in the hemiplegic hand. To the best of the author’s knowledge, this has not been done before. An original finding was that KMI and VMI may be useful for different aspects of motor retraining following stroke. The finding that KMI was linked to a faster overall movement time was, for example, replicated. However, it was also found that VMI was more effective in promoting improved joint interaction.

Experiments one and two were also original in that they involved optoelectronic motion capture and kinematic analysis of rotational movements in distal joints in the hemiplegic hand. To the best of the author’s knowledge, this has not been attempted before. In addition, experiment two also made use of a novel approach to the problem of determining the start and end points of such movements for kinematic analysis.

Experiment three presented evidence showing that the use of KMI and VMI was linked to a reduction in EMG activity in the hemiplegic hand muscles. Earlier discussions of the relationship between MI and peripheral muscular activity have focused on whether MI increases EMG activity or has no effect. The finding of



experiment three therefore represents an original contribution. In addition, it was found that the sensory modality of the imagery used made little difference to the level of reduction in electrical activity. This is therefore further evidence that MI may work at the early-selection stage of attention, as described above for experiment one.

## **5.2 How the Work Might Benefit Rehabilitation Practice**

In general terms, all three experiments have provided evidence that a one-off session of MI training may produce changes in the hemiplegic hand. This is a finding with applicability to the acute clinical setting. It has been shown, for example, that therapists have very limited time to spend with their patients at this stage (Turton & Pomeroy, 2002), and evidence for the viability of such a short intervention is therefore welcome.

More specifically, the finding in experiments one and two that mental training with KMI can lead to faster overall movement time in the hemiplegic hand has clinical relevance. It is known, for instance, that one consequence of stroke can be impairment of non-conscious postural control mechanisms (Gjelsvik, 2008). The individual may therefore have more recourse to responses to postural perturbation by reacting with protective extension of the upper limb (Gjelsvik, 2008). If MI training can help improve the speed of upper limb movements, then this could reduce the person's fear of falling and improve the confidence of the person when mobilising.

It was argued above that MI as used in these experiments might work via early-selection attention. This is clinically relevant as evidence shows that persons with stroke may develop a habit of not using the more affected upper limb (Yekutieli & Guttman, 1993). This can lead to a reduction of sensory feedback from the limb, further passivity, and ultimately a reduction in the size of the cortical representation of the limb (Nudo, 2007). If MI training can lead to attention being focused on the hand, then this may help overcome the problems stemming from such "learned non-use" (Carr & Shepherd, 1998, p. 14).

A key finding in experiment two was that MI using a visual sensory modality may promote improved interaction between distal joints affected by hemiplegia. The importance of this is highlighted by the work of Levin (1996). This author found that, while individuals with upper limb hemiplegia and neurologically intact individuals

were able to reach targets in all areas of a workspace, the hemiplegic participants showed marked impairments in the coordination of elbow and shoulder movements. Furthermore, she argued that impairments were likely to be a result of difficulties in the integration of altered proprioceptive feedback with attention mechanisms (Levin, 1996). This author concluded by calling on clinicians to focus on the improvement of interjoint coordination as part of improving function in the hemiplegic upper limb (Levin, 1996). The results of experiment two suggest that VMI may be a suitable way to achieve this, at least for distal joints while grasping, and are therefore clinically useful.

Experiment three showed that the resting EMG levels in the intrinsic hand muscles of individuals with stroke were significantly higher than those in neurologically intact participants. This may be related to the phenomenon of cortical hyperexcitability which has been noted following stroke (Cicinelli et al., 2007; Nudo, 1999). The source and function of this hyperexcitability remains a matter of debate (Cicinelli et al., 2007; Nudo, 1999). However, it may be potentially damaging (Turton & Pomeroy, 2002). The results of experiment three seem to show a reduction in activity during MI, at least at the spinal level. It is feasible, then, that MI might also have a role in reducing potentially destructive cortical hyperexcitability.

Another pathological phenomenon following stroke is spasticity, linked to the presence of increased neural drive resulting in muscle hypertonicity (Gjelsvik, 2008; Johnson, 2004). A key clinical feature of spasticity is an abnormally high EMG signal, when compared with the same muscle in healthy controls (Stokic, 2010). The finding that MI may lead to a reduction in EMG in a hemiplegic muscle may therefore point to a role for MI in the treatment of spasticity.

A key finding in experiment three was that the use of MI might actually reduce the EMG signal in a hemiplegic hand muscle. It was speculated, on the basis of the work of Bonnet et al. (1996) that this might occur as a result of engagement of inhibitory mechanisms via the Renshaw cell system. If this is the case, then MI might also be deployed in the sensory re-training of the hand following stroke. This is because sensory acuity requires “lateral inhibition” in order to distinguish stimuli (Gardner & Kandel, 2000, p. 462). This means that the neurons representing the zone

between two converging inputs is inhibited, allowing discrimination of the two inputs. If MI does play a role in inhibition, then, it may also be of use in the recovery of stereognosis, which is considered to be important in the general treatment of the post-stroke hand (Gjelsvik, 2008).

### **5.3 Limitations and Proposals for Future Work**

#### **5.3.1 The need for better experimental controls.**

Issues arose in experiment two regarding the type of motor training task used. In addition, it was also argued that the site of the patients' lesions may have been a source of bias. Furthermore, the age of the participants in the samples may also have been a limitation. These three issues are discussed in more detail below.

It was found in experiment two that VMI, rather than KMI, actually seemed more effective in promoting the pattern of joint interaction in line with the constraints of the lumbrical action. As part of the discussion of this finding a number of authorities were referenced which have suggested that KMI and VMI might be suited to the learning of different types of motor task. In particular, evidence was put forward from Féry (2003), White and Hardy (1995) and Hardy and Callow (1999) to show that MI using a visual sensory modality may be more appropriate for tasks involving closed skills and which emphasize the exact reproduction of the form of an action. Conversely, as suggested by Féry (2003), KMI may be more suitable when learning to reproduce a specified movement duration. Given the evidence provided in experiment two, and that provided by these earlier researchers, it may be the case that the training task needs to be more clearly defined in future research. For example, it would need to be specified whether the task was an open or closed skill.

It was also argued that the participants in experiment two were more able to use VMI on the basis that their visual cortices remained intact; their ability to use KMI would by implication be impaired as a result of damage to cortical motor regions. If this were the case, then future research should control more carefully for lesion site. VMI and KMI ability could be tested, for example, on patients presenting only with visual field loss, perhaps following a stroke affecting the optic nerve behind the optic chiasm or the occipital cortex (Mew & Winnall, 2010). Alternatively, a sample made up of patients suffering only from a motor impairment could be tested using KMI and

VMI. The results of such an experiment would help clarify whether lesion site affected the ability to use MI in different sensory modalities. One limitation of the present study was that clear lesion site data were not available for all patients. Future work would need to involve the careful collection of radiological data in cooperation with neurologists and radiologists. This would then allow carefully controlled patient selection.

A further issue involves the range of times since stroke used in these experiments and other studies. Evidence has shown that there may be a period following the initial event during which experience based neural plasticity and motor learning are likely to occur, although the optimal time period remains an open question (see, for example, Nudo, 1999; Woldag & Hummelsheim, 2002). It may be helpful, therefore, to stratify a patient sample on this basis. For example, the responses of patients using MI at the acute stage could be compared with patients who were several months post-event. It would then be possible to gain a clearer picture of the efficacy of MI training at different time points.

### **5.3.2 The use of the Modified Ashworth Scale.**

The Modified Ashworth Scale was used in the experiments as a measure of muscle stiffness. As has been shown, muscle stiffness can result from altered patterns of descending neural control (Carr & Shepherd, 1998), and also from peripheral changes in the neuromuscular apparatus (Gracies, 2005). The Modified Ashworth Scale has, however, been criticized on the basis that it does not take account of these differing sources of hypertonicity (Carr & Shepherd, 1998). The scale has also recently been shown to have low levels of inter-rater reliability and correlate poorly with muscle reflex activity as measured by EMG (Fleuren et al., 2009). Indeed, one reviewer has recently commented sardonically on the “perpetual investigations on the limitations of (the) Ashworth scale” (Stokic, 2010, p. 1790). A more reliable and valid way to assess muscle stiffness may therefore be needed in future work. This might be of particular importance, given that patients with higher levels of hypertonicity may find an intervention such as mental practice using MI less beneficial than patients who have greater freedom of movement in the affected limb. It should also be borne in mind that some studies using the MI approach have been of very high functioning

patients. This was the case, for example, in the research of González et al. (2005), where the patients appear to have made a full motor recovery. It is, however, important to include patients with higher levels of impairment in a study of MI and a more reliable and valid test of hypertonicity would be a step towards greater control over the levels of impairment represented in a sample. A way forward may be to combine a clinical scale, such as the tonus component of the Motor Assessment Scale, with a less subjective technique, such as measuring EMG.

### **5.3.3 Controlling for compliance with MI.**

One radical critique of MI research suggests that individuals are likely not to be using internal imagery at all, but merely accessing implicit knowledge about the way objects behave in the real world (Pylyshyn, 2002). It might be argued, for example, that human beings clearly do have implicit knowledge about the path, velocity and acceleration of moving objects. It would not be possible, for example, to safely cross a busy road without such tacit knowledge. As a consequence it could be maintained that patients may simply be accessing such stored covert knowledge when supposedly using MI, a point made by several authorities in the field (Sharma et al., 2006; Decety & Jeannerod, 1996; Kosslyn et al., 2006).

An issue which arose in this thesis was that what was taken as MI may, in fact, have been identical with early-selection attention, as described by Gazzaniga et al., 1998. To explore this further it would be necessary to find a reliable method to determine the degree to which a participant is actually deploying MI. Two techniques which have been developed to accomplish this task are discussed below.

Sharma, Jones, Carpenter, and Baron (2008) have demonstrated one feasible method to determine compliance through the use of a test based on mental chronometry. Participants are asked to indicate the actual and imagined tempo at which they can no longer perform a movement sequence. If the two times match then it is assumed that they were, in fact, using MI.

Another method for controlling for compliance with MI was put forward by Guillot et al. (2007) and Guillot et al. (2009). Here, changes in hand skin resistance were taken as a measure of autonomic nervous system (ANS) activity. This was done by measuring ohmic perturbation duration (OPD), which is a drop in palmar skin

resistance thought to correlate with mental activities such as movement planning (Guillot et al., 2009). It is believed that the onset of the MI task coincides with an increase in sweating and a decrease in skin resistance and the researchers in both these cases posited that this could be taken as an objective measure of the duration of the MI practice. Guillot et al. (2009), for example, compared the timing of the OPD during MI of a finger sequencing activity with the time taken to actually execute the movement. This then provided the authors with evidence as to whether the participants were using MI or not.

#### **5.3.4 Controlling for high levels of movement variability.**

A major problem that arose throughout the research was the issue of variability in the movements of the hemiplegic hand. It has been suggested that few authors have paid sufficient attention to this problem in measuring movements in hemiplegia, with the exception of Levin (1996) and Van Vliet and Sheridan (2007). It was found, in addition, that MI did not appear to have any effect at the level of motor variability. For example, the number of MUs during the approach to grasp the object was unaffected by the use of MI. Most interestingly, perhaps, neither were the SDs of the APB EMG in experiment three affected, despite the overall reduction of EMG activity during MI for that muscle. It is believed that it will be essential to address this issue if research into the use of MI in stroke is to meaningfully progress.

One way forward here may be to take this variability into account at the research design stage. As has been shown, for example, the use of repeated measures allows, to an extent, the experiment's subjects to act as their own control group. This means that the issue of inter-subject variability becomes less important. In addition, statistical methods such as repeated measures ANOVA or a dependent samples *t* test, take into account the reduced variability consequent upon testing the same sample two or more times (Vincent, 1998). It may be desirable, therefore, to maximise the repeated measures aspect of any research design.

Disadvantages of the repeated measures design may, however, include practice effects as the participants become used to the training task being used in the study (Boniface, 1995). They may therefore simply improve on a reaching and grasping task with repetition, not necessarily because of the experimental treatment being used. In

experiment two this issue was addressed by comparing the initial and final movement times using a dependent samples *t* test. Such a post hoc test might help control for such practice effects in a repeated measures design.

A further limitation of the repeated measures approach is that if more than one condition is being tested on the same sample, then one condition might affect the other and this interaction might enter the study as a confounding variable (Boniface, 1995). However, as Boniface (1995) suggests, these issues, which may affect the validity of the study, can be controlled for. This could be done, for example, by altering the order of presentation of the conditions. In experiment three in the present research, for instance, the VMI and KMI conditions were presented in a counterbalanced order. This meant that the possible effects of one condition upon the other were at least partially controlled for.

One final problem, specific to the stroke population, may be the level of increased fatigue upon repetition of a reaching and grasping task. This could also lead to increased muscle and joint stiffness, making the task more difficult to execute. Ways would have to be found, therefore to minimize these factors in any repeated measures design, perhaps by spacing the practice sessions out appropriately.

### **5.3.5 Developing models of the hemiplegic hand.**

One methodological aspect of experiments one and two which was felt to be innovative in terms of MI research in stroke, was the use of time normalisation, splining and ensemble-averaging as a data reduction technique. This allowed the construction of a single curve that was representative of a typical grasp for an individual participant. This provided a way of analysing the kinematics of the joints across a whole movement, allowing, for example, the phase changes of the grasping movement to be identified. The technique could be developed in future research and one possible application is discussed below.

Several times in this thesis the lack of appropriate models of the hemiplegic hand has been discussed. Kleissen, Burke, Harlaar, and Zilvold (1998) have, in addition, pointed to the paucity of models which might be applied to movements in hemiplegia generally. Such a model should allow the key features of a class of movements to be identified and therefore deviations away from these patterns to be

analysed. This would also allow predictions to be made as to the kind of improvements the researcher would need to look for as proof of success for a MI training strategy.

A model may, however, present an oversimplified picture, which fails to capture the complexity of the actual movements. Both Schmidt (1975) and Latash (2008) have highlighted this pitfall. Latash (2008, p. 323) has commented that “a formal model is justified only when it is built on a deep understanding of the system of interest and when it is specific to this system”. It was noted in the present research that, to the best of the authors’ knowledge, the only model which has specifically addressed the hemiplegic hand is that of Meulenbroek et al. (2001). However, this model was not based upon close analysis of patients’ hand movements, but on a computer simulation. This does not seem to fit with the demands of Latash (2008) and Schmidt (1975) that any model should be based on careful observation of data derived from actual human movement and should also permit the generation of testable hypotheses.

The use of time normalisation, splining and ensemble averaging could provide a way forward in the development of a model of the hemiplegic hand. For example, by using these techniques to construct data sets for categories of hemiplegic hand movements while performing, for instance, different types of grip or targeted reaches captured from a large number of patients, the researcher could construct ensemble-averaged curves representative of the movements of a particular kind of patient performing a particular task. For example, the PIP joints of a number of patients of a particular age with a specific lesion site performing a handwriting activity could be captured and ensemble-averaged. The same measurements could then be taken of age-matched healthy controls performing the same activity and ensemble averages of these movements could be constructed. These two sets of values might be plotted in a scatter plot and also compared using Pearson’s correlation coefficient. An improved performance following MI training might then be marked by a strengthening of the correlation coefficient and a reduction in the magnitude of the residuals on either side of the best fit line on the scatter plot. This would also allow the development of testable hypotheses, as the researcher would have a clearer idea of precisely what



would constitute an improved performance for a stroke patient on a given dependent variable.

A related question concerns the biomechanical model of the hand used in experiment one and developed in experiment two. Other models used in research on the effects of MI on the kinematics of the hemiplegic upper limb have tended to neglect the joints of the fingers. The model used by Hewett et al. (2007), for example, used a single marker on one finger tip and a single marker on an MCP joint. Lin et al. (2007) had markers on the thumb and index finger tips only. These biomechanical models would not be adequate to capture all the degrees of freedom in the hand and would also not allow detailed analysis of rotational angular motion. Experiments one and two presented in this thesis did attempt this and the model developed there may offer a useful starting point for future research.

### **5.3.6 Measurement problems.**

The problem of measuring the precise start and end points of movements in the hemiplegic hand has already been discussed in chapters one and two. The experiment presented in chapter two offered a solution to this problem based on the velocity profiles of the movement which has already been discussed in detail above. It was also suggested that an approach which combined physiological measures, such as EMG, might be useful here. However, the results of experiment three also highlight potential problems with the use of EMG in such a way. It will be recalled that the EMG signal in the APB of the hemiplegic patients was only slightly higher during the initial maximum voluntary contraction than during the resting baseline and that the difference between the two conditions was not statistically significant. The level of the EMG signal may therefore not provide the clearest picture of the initiation point of a movement in adult hemiplegia. Another solution might be the use of multiple raters examining the data and estimating the start and end points. This could be done visually, for example, by inspecting the moving images of the reflective markers on the computer monitor. The identified start and end points of the movements could then be compared with a measure such as Cronbach's alpha, to determine the degree of inter-rater reliability. This, of course, would be a time consuming approach. However, it remains the case that future research will need to find a reliable solution to this

perennial problem in human movement studies: how to measure the start and end points of multi-joint movements.

### **5.3.7 Controlling for mainstream therapy.**

Another issue which arose during the review of literature was the fact that many studies of the use of MI in the clinical setting have failed to control for the normal mainstream therapy which the patients were receiving. As has already been stated, as long as this is not clearly defined, then there is a possibility that this therapy could enter the experiment as a confounding, uncontrolled variable. While having the advantage of reflecting the reality of everyday clinical practice, the validity of the findings may be open to question. In experiments one and two, presented in the present research, the therapy intervention for the hemiplegic hand was very carefully defined and each patient was treated according to an identical therapy protocol. Future research should strengthen this aspect of the study design. Of course, this might be difficult. As has been shown, there is a variety of approaches used in the clinical setting, and therapists often work in an ad hoc manner (Kuipers et al., 2006). Furthermore, the use of a stereotyped treatment protocol clashes with principles of neurological rehabilitation, which stresses that the treatment should be tailored to the needs of the individual patient (Raine et al., 2009). Nonetheless, a valid test of the effects of MI alongside mainstream treatment will need to find a way of controlling for this.

### **5.4 The Future of MI Research in Stroke.**

Looking to the immediate future of MI research in stroke, it is felt that some recent work is likely to lead to a degree of pessimism regarding the use of the technique in the treatment of upper limb hemiplegia. The study by Ietswaart et al. (2011), for example, used what, to the best of the author's knowledge, is the most extensive sample to date to test the technique in upper limb treatment and found the results to be completely negative. However, the results of the three experiments presented in this thesis suggest that some rethinking is called for. First, MI might have very general effects that may in fact be similar to early-selection attention. Second, the effects noted here for KMI and VMI suggest that different types of MI might be useful depending on the nature of the task which is being trained. Third, clinical outcome

measures should be combined with higher resolution electronic measurement techniques if the benefits of the technique for the rehabilitation of the hemiplegic hand are to be meaningfully explored. If these points are taken into account, then there may still be cause for cautious optimism.

## References

- Abend, W., Bizzi, E. & Morasso, P. (1982). Human arm trajectory formation. *Brain*, *105*, 331-348.
- Allami, N., Paulignan, Y., Brovelli, A., Boussaoud, D. (2008). Visuo-motor learning with combination of different rates of motor imagery and physical practice. *Experimental Brain Research*, *184*, 105-113.
- Amemiya, K., Ishizu, T., Ayabe, T and Kojima, S. (2010). Effects of motor imagery on intermanual transfer: A near-infrared spectroscopy and behavioural study. *Brain Research*, *1343*, 93-103.
- América Economía (2011). *Los mejores hospitales de América Latina*. Retrieved from: <http://rankings.americaeconomia.com/2011/clinicas/>
- Alarcón, F., Zijlmans, J.C.M., Dueñas, G., & Cevallos, N. (2004). Post-stroke movement disorders: report of 56 patients. *Journal of Neurology, Neurosurgery and Psychiatry*, *75*, 1568–1574. doi: 10.1136/jnnp.2003.011874
- Alvarez, F. J. & Fyffe, R.E.W. (2007). The continuing case for the Renshaw cell. *Journal of Physiology*, *58*, 31–45.
- Arksey, H. & O'Malley, L. (2005). Scoping studies: towards a methodological framework. *International Journal of Social Research Methodology*, *8*, 19-32.
- Avanzino, L. Giannini, A., Tacchino, A, Pelosin, Ruggeri, P. & Bove, M. (2009). Motor imagery influences the execution of repetitive finger opposition movements. *Neuroscience Letters*, *466*, 11–15.
- Barclay-Goddard, R.E., Stevenson, T.J., Poluha, W., & Thalman, L. (2001). Mental practice for treating upper extremity deficits in individuals with hemiparesis after stroke (review). *The Cochrane Library*, issue 8.
- Baddeley, A. (2003). Working memory: looking back and looking forward. *Nature Reviews Neuroscience*, *4*, 829 -839.
- Bakker, F.C., Boschker, M.S.J., & Chung, T. (1996). Changes in muscular activity while imagining weight lifting using stimulus or response propositions. *Journal of Sport and Exercise Psychology*, *18*, 313-324.
- Bastian, H.C. (1887). The muscular sense; its nature and cortical localisation. *Brain*, *10*, 1-89. doi:10.1093/brain/10.1.1

- Battaglia, F., Quartarone, A., Ghilardi, M.F., Dattola, R., Bagnato, S., Rizzo, V., Morgante, L., & Girlanda, P. (2006). Unilateral cerebellar stroke disrupts movement preparation and motor imagery. *Clinical Neurophysiology*, *117*, 1009–1016.
- Beebe, J.A. & Lang, C.E. (2009). Active range of motion predicts upper extremity function 3 months after stroke. *Stroke*, *40*, 1772-1779.
- Bernstein, N. (1967). *The co-ordination and regulation of movements*. Oxford: Pergamon Press.
- Bertrand, A.M., Mercier, C., Shun, P.L.W., Bourbonnais, D., & Desrosiers, J. (2004). Effects of weakness on symmetrical bilateral force exertion in subjects with hemiparesis grip. *Journal of Neurophysiology*, *91*, 1579-1585.  
doi:10.1152/jn.00597.2003
- Binkofski, F., Dohle, C., Posse, S., Stephan, K.M., Hefter, H., Seitz, R.J., & Freund, H.J. (1998). Human anterior intraparietal area subserves prehension: a combined lesion and functional MRI activation study. *Neurology*, *50*, 1253-1259.
- Bizzi, E., Kalil, R.E., & Tagliasco, V. (1971). Eye-head coordination in monkeys. Evidence for centrally patterned organization. *Science*, *173*, 452-4.
- Blakemore, S-J., Wolpert, D., & Frith, C. Why can't you tickle yourself? *NeuroReport*, *11*, 11-16.
- Blaxter, L., Hughes, C. and Tight, M. (2010). *How to research (fourth edition)*. Milton Keynes: Open University.
- Bobath, B. (1989). *Adult hemiplegia: evaluation and treatment*. Oxford: Heinemann.
- Bohannon, R.W. & Smith, M.B. (1987). Interrater reliability of a modified Ashworth scale of muscle spasticity. *Physical Therapy*, *67*, 206-207.
- Boniface, D.R. (1995). *Experiment design and statistical methods*. London: Chapman & Hall.
- Bonnet, M., Decety, J., Jeannerod, M., & Requin, J. (1997), Mental simulation of an action modulates the excitability of spinal reflex pathways in man. *Cognitive Brain Research*, *5*, 221–228.
- Bovend'Eerd, T.J., Dawes, H., Sackley, C., Izadi, H., & Wade, D.T., (2010). An

- integrated motor imagery program to improve functional task performance in neurorehabilitation: a single-blind randomized controlled trial. *Archives of Physical Medicine and Rehabilitation*, 91, 939-46.
- Braun, S.M., Beurskins, A.J., Borm, P.J., Schack, T., & Wade, D.T. (2006) The effects of mental practice in stroke rehabilitation: A systematic review. *Archives of Physical Medicine and Rehabilitation*, 87,842-852.
- Brodal, A. (1973). Self-observations and neuroanatomical considerations after a stroke. *Brain*, 96, 675-694.
- Butler, A.J. & Page, S.J. (2006). Mental practice with motor imagery: evidence for motor recovery and cortical reorganization after stroke. *Archives of Physical Medicine and Rehabilitation*, 87, S2-11.
- Callow, N. & Hardy, L. (2004). The relationship between the use of kinaesthetic imagery and different visual imagery perspectives. *Journal of Sports Sciences*, 22, 167-177. doi: 10.1080/02640410310001641449
- Capaday, C. , Forget, R., Fraser, R.,& Lamarre, Y. (1991). Evidence for a contribution of the motor cortex to the long-latency stretch reflex of the human thumb. *Journal of Physiology*, 440, 243–255.
- Carr, J.H. & Shepherd, R.B. (1998). *Neurological rehabilitation – optimizing motor performance*. Oxford: Butterworth Heinemann.
- Carr, J.H. & Shepherd, R.B. (1987). *A motor relearning programme for stroke*. Oxford: Butterworth Heinemann.
- Carr, J.H., Shepherd, R.B., Nordholm, L. and Lynne, D. (1985). Investigation of a new motor assessment scale for stroke patients. *Physical Therapy*, 65, 175-180.
- Castiello, U. (2005). The neuroscience of grasping. *Nature Reviews Neuroscience*, 6, 726 -736.
- Chau, T., Young, S. & Redekop, S (2005). Managing variability in the summary and comparison of gait data. *Journal of NeuroEngineering and Rehabilitation*, 2, doi:10.1186/1743-0003-2-22
- Champion, J. Barber, C. & Lynch-Ellerington (2009). Recovery of upper limb function. In S. Raine, L. Meadows, M. Lynch-Ellerington (Eds.), *Bobath*

*concept: theory and practice in neurological rehabilitation.* Oxford: Blackwell.

- Cheung, V.C.K., Piron, L., Agostini, M., Silvoni, S., Turolla, A., & Bizzi, E. (2009). Stability of muscle synergies for voluntary actions after cortical stroke in humans. *Proceedings of the National Academy of Science*, *106*, 19563-19568. doi: 10.1073/pnas.0910114106
- Cicinelli, P., Marconi, B., Zaccagnini, M., Pasqualetti, P., Filippi, M.M., & Rossini, P.M. (2006). Imagery-induced cortical excitability changes in stroke: A transcranial magnetic stimulation study. *Cerebral Cortex*, *16*, 247-253.
- Cincotta, M., Tozzi, F., Zaccara, G., Borgerheresi, A., Cosottini, M., & Cantello, R. (1999). Motor imagery in a locked-in patient: evidence from transcranial magnetic stimulation. *Italian Journal of Neurological Science*, *20*, 37-41.
- Cirstea, M.C. & Levin, M.F. (2000). Compensatory strategies for reaching in stroke. *Brain*, *123*, 940 -953.
- Cirstea, M.C., Mitnitski, A.B., & Feldman, A.G. (2003). Interjoint coordination dynamics during reaching in stroke. *Experimental Brain Research*, *151*, 289–300. doi 10.1007/s00221-003-1438-0.
- Collins, D. (2001). The PETTLEP approach to motor imagery: a functional equivalence model for sport psychologists. *Journal of Applied Sport Psychology*, *13*, 60-83.
- Courtine, G., Papaxanthis, C., Gentili, R. & Pozzo, T. (2004). Gait-dependent motor memory facilitation in covert movement execution. *Cognitive Brain Research*, *22*, 67-75.
- Crosbie, J.H., McDonough, S.M., Gilmore, D.H., & Wiggam, M.I. (2004). The adjunctive role of mental practice in the rehabilitation of the upper limb after hemiplegic stroke: a pilot study. *Clinical Rehabilitation*, *18*, 60–68.
- Crossman, E.R.F.W. & Goodeve, P.J. (1963). Feedback control of hand movement and Fitts' Law. Paper presented at the meeting of the Experimental Psychology Society, Oxford, July 1963. Published in *Quarterly Journal of Experimental Psychology*, *35A*, 251 – 278.
- Davis, P.M. (1994). *Steps to follow – a guide to the treatment of adult hemiplegia.*

Berlin: Springer-Verlag.

- Decety, J. (1996). Do imagined and executed actions share the same neural substrate? *Cognitive Brain Research*, 3, 87-93.
- Decety, J. & Jeannerod, M. (1996). Mentally stimulated movements in virtual reality: does Fitts' Law hold in mental imagery? *Behavioral Brain Research*, 72, 127-134.
- Decety, J., Jeannerod, M., & Prablanc, C. (1989). The timing of mentally represented actions. *Behavioural Brain Research*, 34, 35-42.
- Decety, J., Jeannerod, M., Germain M., & Pastene, J. (1991). Vegetative response during imagined movement is proportional to mental effort. *Behavioural Brain Research*, 42, 1-5.
- de Morton, N.A. (2009). The PEDro scale is a valid measure of the methodological quality of clinical trials: a demographic study. *Australian Journal of Physiotherapy*, 55, 129-133.
- Department of Health (2005). *Research governance framework for health and social care*. London: Department of Health.
- Dickstein, R. & Deutsch, J.E. (2007). Motor imagery in physical therapist practice. *Physical Therapy*, 87, 942-953.
- Dickstein, R., Gazit-Grunwald, M., Plax, M., Dunsky, A., & Marcovitz, E. (2005). EMG activity in selected target muscles during imagery rising on tiptoes in healthy adults and post stroke hemiparetic patients. *Journal of Motor Behaviour*, 37, 475-483.
- Dietz, V. (2003) Spastic movement disorder: what is the impact of research on clinical practice? *Journal of Neurology, Neurosurgery and Psychiatry*, 74, 820-821.
- Di Pellegrino, L., Fadiga, L., Fogassi, V., Gallese, V., Rizzolatti, G. (1992). Understanding motor events: a neurophysiological study. *Experimental Brain Research*, 91, 176- 180.
- Dijkerman, H.C., Ietswaart, M., Johnston, M., & MacWalter, R.S. (2004). Does motor imagery training improve hand function in chronic stroke patients? A pilot study. *Clinical Rehabilitation*, 18, 538-549.
- Dobkin, B. (2004). Strategies for stroke rehabilitation. *Lancet Neurology*, 3, 528-536.



- Donovan, J.J. & Radosovich, D.J. (1999). A meta-analytic review of the distribution of practice effect: now you see it, now you don't. *Journal of Applied Psychology, 54*, 795-805.
- Dorfman, L.J., Howard, J.E., & McGill, K.C. (1989). Motor unit firing rates and firing rate variability in the detection of neuromuscular disorders. *Electroencephalography and Clinical Neurophysiology, 73*, 215-224.
- Driskell, J.E., Copper, C., & Moran A. (1994). Does mental practice enhance performance? *Journal of Applied Psychology, 79*, 481-492.
- Dronkers, N., Pinker, S., & Damasio, A. (2000). Language and the aphasias. In E.R., Kandel, J.H. Schwartz & T.M Jessell (Eds.), *Principles of neural science* (pp.1170-1187). New York: McGraw-Hill.
- European Medicines Agency (2002). *Guideline for good clinical practice*. London: European Medicines Agency.
- Ferbert, A. & Gerwig, M.(1993). Tremor due to stroke. *Movement Disorders, 8*, 179-182.
- Féry, Y-A., (2003). Differentiating visual and kinesthetic imagery in mental practice. *Canadian Journal of Experimental Psychology, 57*, 1-10.
- Fitts, P. (1954). The information capacity of the human motor system in controlling the amplitude of movement. *Journal of Experimental Psychology, 47*, 381-391.
- Fitts, P.M. & Peterson, J.R. (1964). Information capacity of discrete motor responses. *Journal of Experimental Psychology, 67*, 103-112.
- Flash, T. & Hogan, N. (1985). The coordination of arm movements: an experimentally confirmed mathematical model. *The Journal of Neuroscience, 7*, 1688-1703.
- Fleming, M.K., Stinear, C.M., & Byblow, W.D. (2010). Bilateral parietal cortex function during motor imagery. *Experimental Brain Research, 201*, 499–508. doi 10.1007/s00221-009-2062-4.
- Fleuren, J.F.M., Nederhand, M.J., & Hermens, H.J. (2006). Influence of posture and muscle length on stretch reflex activity in post stroke patients with spasticity. *Archives of Physical Medicine and Rehabilitation, 87*, 981-988.
- Fleuren, J.F.M., Voerman, G.E., Erren-Wolters, C.V., Snoek, G.J., Rietman, J.S.,

- Hermens, H.J., & Nene, A.V. (2010). Stop using the Ashworth scale for the assessment of spasticity. *Journal of Neurology, Neurosurgery and Psychiatry*, *81*, 46-52.
- Fourkas, A., Avenanti, A. Urgesi, C., & Aglioti, S.M. (2006). Corticospinal facilitation during first and third person imagery. *Experimental Brain Research*, *168*, 143-151. doi: 10.1007/s00221-005-0076-0
- Freitas, S.M.S.F, & Scholz, J.P. (2009). Does hand dominance affect the use of motor abundance when reaching to uncertain targets? *Human Movement Science*, *28*,169–190.
- Frey, S.H., Vinton, D., Norlund, R. & Grafton, S.T. (2005) Cortical topography of human anterior intraparietal cortex active during visually guided grasping. *Cognitive Brain Research*, *23*, 397-405.
- Fung, J. & Barbeau, H. (1989). A dynamic EMG profile index to quantify muscular activation disorder in spastic paretic gait. *Electroencephalography and Clinical Neurophysiology*, *73*, 233-244.
- Gandevia, S.C. (1999). Mind, muscles and motoneurons. *Journal of Science and Medicine in Sport*, *2*,167-180.
- Gandevia, S.C. & McCloskey, D.I. (1977). Changes in motor commands, as shown by changes in perceived heaviness, during partial curarization and peripheral anaesthesia in man. *Journal of Physiology*, *272*, 673-689.
- Gandevia, S.C., Wilson, L.R., Inglis, J.Y., & Burke, D. (1997). Mental rehearsal of motor tasks recruits a-motoneurons but fails to recruit human fusimotor neurones selectively. *Journal of Physiology*, *505*, 259-266.
- Gandevia, S.C. & Rothwell, J.C. (1987). Knowledge of motor commands and recruitment of motoneurons. *Brain*, *110*, 1117-1130.
- Hambleton R.K. & Bollwark J. (1991) Adapting tests for use in different cultures: technical issues and methods. *Bulletin of the International Test Commission*, *18*, 3-32.
- Hart, C. (1999). *Doing a literature review: releasing the social science research imagination*. London: Sage.
- Ganis, G., Keenan, J.P. Kosslyn, S.M., & Pascual-Leone, A. (2000). Transcranial

- magnetic stimulation of primary motor cortex affects mental rotation. *Cerebral Cortex*, 10, 175-80.
- Gardner, E.P., Martin, J.H., & Jessell, T.M. (2000). The bodily senses. In E.R., Kandel, J.H. Schwartz & T.M Jessell (Eds.), *Principles of neural science* (pp.430-450). New York: McGraw-Hill.
- Gazzaniga, M.S., Ivry, I.B. & Mangun, G.R. (1998). *Cognitive neuroscience - the biology of the mind*. New York: Norton.
- Gjelsvik, B.E.B. (2008). *The Bobath concept in adult neurology*. Stuttgart: Thieme.
- González, B., Rodríguez, M., Ramirez, C., & Sabaté, M. (2005). Disturbance of motor imagery after cerebellar stroke. *Behavioral Neuroscience*, 119, 622–626.
- Gracies, J.M. (2005). Pathophysiology of spastic paresis. I. Paresis and soft tissue changes, *MuscleNerve*, 31, 535-551.
- Gracies, J.M. (2005a). Pathophysiology of spastic paresis. II. Emergence of muscle overactivity. *Muscle Nerve*, 31, 552-571.
- Guillot, A., Collet, C., & Dittmar, A. (2004). Relationship between visual and kinesthetic imagery, field dependence-independence, and complex motor skills. *Journal of Psychophysiology*, 18, 190–198.
- Guillot A. Lebon, F., & Collet. C. (2010). Electromyographic activity during motor imagery. In A. Guillot & C. Collet (Eds.), *The neurophysiological foundations of mental and motor imagery* (pp.83-91). Oxford: Oxford University Press.
- Guillot, A., Louis, M., & Collet, C. (2010). Neurophysiological substrates of motor imagery ability. In A. Guillot & C. Collet (Eds.), *The neurophysiological foundations of mental and motor imagery* (pp.109–123). Oxford: Oxford University Press.
- Guillot, A., Collet, C., Nguyen, V.A., & Malouin, F. (2009). Brain activity during visual versus kinesthetic imagery: an fMRI study. *Human Brain Mapping*, 30, 2157–2172.
- Guillot, A., Debarnot, U., Louis, M., Hoyek, N., & Collet, C. (2010). Motor imagery and motor performance: evidence from the sport science literature. In A. Guillot & C. Collet (Eds.), *The neurophysiological foundations of mental and*

- motor imagery* (pp.215-226). Oxford: Oxford University Press.
- Guillot, A., Lebon, F., Rouffet, D., Champely, S., Doyon, J., & Collet, C. (2007). Muscular responses during motor imagery as a function of muscle contraction types. *International Journal of Psychophysiology*, *66*, 18–27.
- Guyatt, G.H., Sackett, D.L. & Cook, D.J. (1993). Users' guides to the medical literature: II. how to use an article about therapy or prevention: A. are the results of the study valid? *The Journal of the American Medical Association*, *270*, 2598-2601.
- Hale, B.D. (1982). The effects of internal and external imagery on muscular and ocular concomitants. *Journal of Sport Psychology*, *4*, 379-387.
- Hansen, S., Elliot, D., & Khan, M.A. (2007). Comparing derived and acquired acceleration profiles: 3-D optical electronic data analyses. *Behavior Research Methods*, *39*, 748-754.
- Hardy, L. & Callow, N. (1999). Efficacy of external and internal visual imagery perspectives for the enhancement of performance of tasks in which form is important. *Journal of Sport and Exercise Psychology*, *21*, 95-112.
- Harris, D.V. & Robinson, W.J. (1986). The effects of skill level on EMG activity during internal and external imagery. *Journal of Sport Psychology*, *8*, 105-111.
- Harris, C.M. & Wolpert, D.M. (1998). Signal-dependent noise determines motor planning. *Nature*, *394*, 780-784. doi:10.1038/29528
- Hashimoto, R. & Rothwell, J.C. (1999). Dynamic changes in corticospinal excitability during motor imagery. *Experimental Brain Research*, *125*, 75-81.
- Haynes, J-D, & Rees, G. (2006). Decoding mental states from brain activity in humans. *Nature Reviews Neuroscience*, *7*, 523-534.
- Hemmen, B. & Seelen, H. (2007). Effects of movement imagery and electromyography-triggered feedback on arm-hand function in stroke patients in the subacute phase. *Clinical Rehabilitation*, *21*, 587-594.
- Henneman, E. (1985). The size principle: a deterministic output emerges from a set of probabilistic connections. *Journal of Experimental Biology*, *115*, 105-112.
- Hermens, H.J., Freriks, B., Disselhorst-Klug, C., & Rau, G. (2000). Development of recommendations for SEMG sensors and sensor placement procedures.

- Journal of Electromyography and Kinesiology*, 10, 361–374.
- Hewett, T.E., Ford, K.R., Levine, P., & Page, S.J. (2007). Reaching kinematics to measure motor changes after mental practice in stroke. *Topics in Stroke Rehabilitation*, 14, 23–29.
- Iacoboni, M. (2005). Neural mechanisms of imitation. *Current Opinion in Neurobiology*, 15, 632-637.
- Iacoboni, M. & Dapretto, M. (2006). The mirror neuron system and the consequences of its dysfunction. *Nature Reviews Neuroscience*, 7, 12, 942-951.
- Ietswaart, M., Johnston, M., Dijkerman, H.C., Scott, C.L., Joice, S., Hamilton, S., & McWalter, R.S. (2006). Recovery of hand function through mental practice. A study protocol. *BMC Neurology*, 6, 39.
- Ietswaart, M., Johnston, M., Dijkerman, H.C., Joice, S., Scott, C.L., MacWalter, R.S., & Hamilton, S.J.C. (2011). Mental practice with motor imagery in stroke recovery: randomized controlled trial of efficacy. *Brain*, 134, 1373–1386. doi:10.1093/brain/awr 077
- Intercollegiate Stroke Working Party (2010). *National sentinel stroke audit organisational audit 2010*. London: Royal College of Physicians.
- Iwamuro, B.T., Cruz, E.G., Connelly, L.I., Fischer, H.C., & Kamper, D.G. (2008). Effect of a gravity-compensating orthosis on reaching after stroke: evaluation of the therapy assistant WREX. *Archives of Physical Medicine and Rehabilitation*, 89, 2121-8.
- Jackson, J.H. (1899). On the comparative study of the diseases of the nervous system. *The British Medical Journal*, 1494, 355-382.
- Jackson, P., Lafleur, M.F., Malouin, F., Richards, R., & Doyon, J. (2001). Potential role of mental practice using motor imagery in neurologic rehabilitation. *Archives of Physical Medicine and Rehabilitation*, 82, 1133-41.
- Jacobson, E. (1930). Electrical measurements of neuromuscular states during mental activities. V. Variation of specific muscles contracting during imagination. *American Journal of Physiology*, 96, 115-121.
- Jacobson, E. (1930a). Electrical measurements of neuromuscular states during mental activities. VI. A note on mental activities concerning an amputated upper

- limb. *American Journal of Physiology*, 96, 122-125.
- James, W. (1905). *The principles of psychology*. New York: Henry Holt and Company.
- Jeannerod, M. (1986). The formation of finger grip during prehension. A cortically mediated visuomotor pattern. *Behavioural Brain Research*, 19, 99-116.
- Jeannerod, M. (2006). *Motor cognition: what actions tell the self*. Oxford: Oxford University Press.
- Jeannerod, M. (1988). *The neural and behavioural organization of goal-directed movements*. Oxford: Oxford University Press.
- Jeannerod, M. (1997). *The cognitive neuroscience of action*. Oxford: Blackwell.
- Jeannerod, M. (2001). Neural simulation of action: a unifying mechanism for motor cognition. *NeuroImage*, 14, 103–109. doi:10.1006/nimg.2001.0832
- Jeannerod, M. & Decety, J. (1995). Mental motor imagery: a window into the representational stages of action. *Current Opinion in Neurobiology*, 5, 727-732.
- Jeka, J. (1997) Light touch contact as a balance aid. *Physical Therapy*, 77, 476-487.
- Johansson, B.B. (2000). Brain plasticity and stroke rehabilitation. *Stroke*, 31, 223- 230.
- Johnson, G.R. (2004). *Spasticity – perceptions, definitions and measurement*, 9th Annual Conference of the International FES Society.
- Jowdy, D.P. & Harris, D.V. (1990). Muscular responses during mental imagery as a function of motor skill level. *Journal of Sport and Exercise Psychology*, 12, 191-201.
- Kallenberg, L.A.C. & Hermens, H.J. (2010). Motor unit properties of biceps brachii during dynamic contractions in chronic stroke patients. *Muscle and Nerve*, 43,112–119.doi: 10.1002/mus.21803
- Kamen, G. & Gabriel, D.A. (2010). *The Essentials of Electromyography*. Champaign, IL: Human Kinetics.
- Kandel, E.R. (2000). Cellular mechanisms of learning and the biological basis of individuality. In E.R. Kandel, J.H. Schwartz, & T.M Jessell (Eds.), *Principles of neural science* (pp.1247-1279). New York: McGraw-Hill.
- Kandel, E.R., Jessell, T.M., & Sanes, J.R. (2000). Sensory experience and the fine

- tuning of synaptic connections. In E.R., Kandel, J.H. Schwartz., & T.M. Jessell (Eds.), *Principles of neural science* (pp.1113-1130). New York: McGraw-Hill.
- Kandel, E.R. and Siegelbaum, S.A. (2000). Transmitter release. In E.R., Kandel, J.H. Schwartz & T.M. Jessell (Eds.), *Principles of neural science* (pp.252-279). New York: McGraw-Hill.
- Keele, S.W. (1968). Movement control in skilled motor performance. *Psychological Bulletin*, 70, 6.
- Kerr, A., Durward, B., & Kerr, K.M. (2004). Defining phases for the sit-to-walk movement. *Clinical Biomechanics*, 19, 385–390.
- Kim, J.S. (1992). Delayed onset hand tremor caused by cerebral infarction. *Stroke*, 23, 292-294.
- Kim, J.S. & Lee, M.C. (1994). Writing tremor after discrete cortical infarction. *Stroke*, 25, 2280-2282.
- Kim, J-S., Oh, D-W., Kim, S-Y., & Choi, J-D. (2011). Visual and kinesthetic locomotor imagery training integrated with auditory step rhythm for walking. *Clinical Rehabilitation*, 25, 134–145.
- Klein, I., Dubois, J., Mangin, J-F., Kherif, F., Flandin, G., Poline, J-B., Denis, M., Kosslyn, S.M., & Le Bihan, D. (2004). Retinotopic organization of visual mental images as revealed by functional magnetic resonance imaging. *Cognitive Brain Research*, 22, 26–31.
- Kleissen, R.F.M., Buurke, J.A., Harlaar, J., & Zilvold, G. (1998) Electromyography in the biomechanical analysis of human movement and its clinical application. *Gait and Posture*, 8, 143–158.
- Koesler, I.B.M., Dafotakis, M., Ameli, M., Fink, G.R., Nowak, D.A. (2009). Electrical somatosensory stimulation improves movement kinematics of the affected hand following stroke. *Journal of Neurology, Neurosurgery and Psychiatry*, 80, 614–619. doi:10.1136/jnnp.2008.161117
- Kontaxis, A., Cutti, A.B., Johnson, G.R., & Veeger, H.E.J. (2009). A framework for the definition of standardized protocols for measuring upper-extremity kinematics. *Clinical Biomechanics*, 24, 246–253.

- Kosslyn, S.M., Ganis, G., & Thompson, W.L. (2010). Multimodal images in the brain. In A. Guillot & C. Collet (Eds.), *The neurophysiological foundations of mental and motor imagery* (pp.3–16). Oxford: Oxford University Press.
- Kosslyn, S.M., Pascual-Leone, A., Felician, O., Camposano, S., Keenan, J.P., Thompson, W.L., Ganis, G. Sukel, K.E., & Alpert, N.M. (1999). The role of area 17 in visual imagery: convergent evidence from PET and rTMS. *Science*, *284*, 167-170.
- Kosslyn, S.M., Thompson, W.L., & Ganis, G. (2006). *The case for mental imagery*. Oxford: Oxford University Press
- Kuipers, K., McKenna, K., & Carls, G. (2006). Factors influencing occupational therapists' clinical decision making for clients with upper limb performance dysfunction following brain injury. *British Journal of Occupational Therapy*, *69*, 106-114.
- Kwakkel, G., Kollen, B.J., Van der Grond, J., & Prevo , A.J. (2003). Probability of regaining dexterity in the flaccid upper limb: Impact of severity of paresis and time since onset in acute stroke. *Stroke*, *34*, 2181-6.
- Lancaster, G., Dodd, S., & Williamson, P.R. (2002). Design and analysis of pilot studies: recommendations for good practice. *Journal of evaluation in clinical practice*, *10*, 307–312.
- Lang, C.E. & Schieber, M.H. (2004). Reduced muscle selectivity during individuated finger movements in humans after damage to the motor cortex of corticospinal tract. *Journal of Neurophysiology*, *91*, 1722-1733.
- Lang, P. (1979). A bioinformational theory of emotional imagery. *Psychophysiology*, *16*, 495-512.
- Langhorne, P. Coupar, F., & Pollock, A. (2009). Motor recovery after stroke: a systematic review. *Lancet Neurology*, *8*, 741–54.
- Latash, M.L. (2008). *Synergy*. Oxford: Oxford University Press.
- Latash, M.L. Kang, N., & Patterson, D. (2002). Finger coordination in persons with Down syndrome: atypical patterns of coordination and the effects of practice. *Experimental Brain Research*, *146*, 345–355. doi: 10.1007/s00221-002-1189
- Lawrence, D.G. & G.J.M. (1968). The functional organization of the motor system in



- the monkey: I. the effects of bilateral pyramidal lesions. *Brain*, 91, 1-14.
- Lawrence, D.G. & Kuypers, G.J.M. (1968a). The functional organization of the motor system in the monkey: II. the effects of lesions of the descending brainstem. *Brain*, 91, 15-36.
- Levin, M. (1996). Interjoint coordination during pointing movements is disrupted in spastic hemiparesis. *Brain*, 119, 281-293.
- Li, S. (2007). Movement-specific enhancement of corticospinal excitability at subthreshold levels during motor imagery. *Experimental Brain Research*, 179, 517–524.
- Li, S., Latash, M.L., & Zatsiorsky, V.M. (2004) Effects of motor imagery on finger force responses to transcranial magnetic stimulation, *Cognitive Brain Research*, 20, 273– 280.
- Lin, K-C., Wu, C-Y., Wei, T-H., Gung, C., Lee C-Y., & Liu, J-S. (2007). Effects of modified constraint-induced movement therapy on reach-to-grasp movements and functional performance after chronic stroke: a randomized controlled study, *Clinical Rehabilitation*, 21, 1075-1086. doi: 10.1177/0269215507079843
- Liu, K.P., Chan, C.C., Lee, T.M., & Hui-Chan, C.W. (2004). Mental imagery for promoting relearning for people after stroke: a randomized controlled trial. *Archives of Physical Medicine and Rehabilitation*, 85, 1403-8.
- Llinas, R.R. (2003). *I of the vortex*. Massachusetts: The MIT Press
- Long, C. (1968). Intrinsic-extrinsic muscle control of the fingers: electromyographic studies. *The Journal of Bone and Joint Surgery*, 50, 973-984.
- Lutz, R.F. (2003). Covert muscle excitation is outflow from the central generation of motor imagery. *Behavioural Brain Research*, 140, 149 – 163.
- Malouin, F., Belleville, S., C.L. Richards, Desrosiers, J., & Doyon, J. (2004). Working memory and mental practice outcomes after stroke. *Archives of Physical Medicine and Rehabilitation*, 85, 177-83.
- Malouin, F., Richards, C.L., Doyon, J., Desrosiers, J., Belleville, S. (2004b). Training mobility tasks after stroke with combined mental and physical practice: a feasibility study. *Neurorehabilitation and Neural Repair*, 18, 66-75.

- Marshall, P.A. (2007). *Ethical challenges in study design and informed consent in resource poor settings*. Geneva: World Health Organization.
- The Mathworks Inc. (2007). *Matlab and simulink: student version*. Natick, MA: USA.
- Medical Research Council (1998). *MRC guidelines for good clinical practice in clinical trials*. London: Medical Research Council.
- Menegoni, F., Galli, M., & Fermi, E. (2008). *The analysis of upper limb movements with SMART*. Milan: BTS Bioengineering.
- Meulenbroek, R.G.J., Rosenbaum, D.A., & Vaughan, J. (2001). Planning reaching and grasping movements: simulating reduced movement capabilities in spastic hemiparesis. *Motor Control*, 2, 136-150.
- Mew, M. & Winnall, S. (2010). Management of visual and sensory impairments. In Edmans, J. (Ed.). *Occupational therapy and stroke* (pp 117-143). Chichester: Wiley Blackwell.
- Ministerio de Salud (1993). *Resolucion número 8430 de 1993*. Bogotá, D.C.: Ministerio de Salud.
- Ministerio de la Protección Social (2008). *Resolucion 002378*. Bogotá, D.C.: Ministerio de la Protección Social.
- Moseley, A.M., Herbert, R.D., Sherrington, C., & Maher, C.G. (2002). Evidence for physiotherapy practice: a survey of the physiotherapy database (PEDro). *Australian Journal of Physiotherapy*, 48, 43-49.
- Mulder, T. (2007) Motor imagery and action observation: cognitive tools for rehabilitation. *Journal of Neural Transmission*, 114, 1265-1278.
- Mulder, T., de Vries, S., & Zijlstra, S. (2005). Observation, imagination and execution of an effortful movement: more evidence for a central explanation of motor imagery. *Experimental Brain Research*, 163, 344-351.
- Mulder, T., Zijlstra, S., Zijlstra, W., & Hochstenbach, J. (2004). The role of motor imagery in learning a totally novel movement. *Experimental Brain Research*, 154, 211-217.
- Nagasaki, H. (1989). Asymmetric velocity and acceleration profiles of human arm movements. *Experimental Brain Research*, 74, 319-326.

- National Audit Office (2007). *Joining forces to deliver improved stroke care*. London: The Stationery Office.
- Neilson, P.D. & McCaughey, J. (1982) Self-regulation of spasm and spasticity in cerebral palsy. *Journal of Neurology, Neurosurgery, and Psychiatry*, *45*, 320-330.
- Neuper, C., Scherer, R., Reiner, M., & Pfurtscheller, G. (2005). Imagery of motor actions: differential effects of kinesthetic and visual–motor mode of imagery in single-trial EEG. *Cognitive Brain Research*, *25*, 668 – 677.
- Nilsen, D.M., Gillen, G., & Gordon, A.M. (2010). Use of mental practice to improve upper- limb recovery after stroke: a systematic review. *American Journal of Occupational Therapy*, *64*, 695–708. doi: 10.5014/ajot.2010.09034
- Nowak, D.A. (2008). The impact of stroke on the performance of grasping: Usefulness of kinetic and kinematic motion analysis. *Neuroscience and Biobehavioral Reviews*, *32*, 1439–1450.
- Nudo, R. (1999). Recovery after damage to motor cortical areas. *Current Opinion in Neurobiology*, *9*, 740-747.
- O'Brien, J. & Bracewell, R.M. (2010). A brief historical review of motor control theory. *Advances in Clinical Neuroscience and Rehabilitation*, *10*, 22-23.
- Page, S.J., Levine P., & Leonard, A. (2005). Effects of mental practice on affected limb use and function in chronic stroke. *Archives of Physical Medicine and Rehabilitation*, *86*, 399-402.
- Page, S.J., Levine, P., & Khoury, J.C. (2009). Modified constraint-induced therapy combined with mental practice: thinking through better outcome measures. *Stroke*, *40*, 551-554. doi: 10.1161/STROKEAHA.108.528760
- Page, S.J., Levine P., & Leonard, A. (2007) Mental practice in chronic stroke - results of a randomized, placebo-controlled trial. *Stroke*, *38*, 1293-1297.
- Page, S.J., Levine, P., Sisto, S.A., & Johnston, M.V. (2001). A randomized efficacy and feasibility study of imagery in acute stroke. *Clinical Rehabilitation*, *15*, 233–240.
- Palastanga, N., Field, D., & Soames, R. (2006). *Anatomy and human movement – structure and function*. London: Butterworth Heinemann.

- Papaxanthis, C., Pozzo, T., Skoura, X., & Schieppati, M. (2002). Does order and timing in performance of imagined and actual movements affect the motor imagery process? The duration of walking and writing task. *Behavioural Brain Research, 134*, 209-215.
- Papaxanthis, C., Schieppati, M., Gentili, R., & Pozzo, T. (2002). Imagined and actual arm movements have similar durations when performed under different conditions of direction and mass. *Experimental Brain Research, 143*, 447-45.
- Patrick, J.H., Pruchno, R.A., & Rose, M.S. (1998). Recruiting research participants: a comparison of the costs and effectiveness of five recruitment strategies. *The Gerontologist, 38*, 295-302.
- Payne, R.A. & Donaghy, M. (2010). *Payne's handbook of relaxation techniques*. London: Churchill Livingstone.
- Pearson, K. & Gordon, J. (2000). Spinal reflexes. In E.R., Kandel, J.H. Schwartz, & T.M Jessell (Eds.), *Principles of neural science* (pp.713-736). New York: McGraw-Hill.
- Porter, R. & Lemon, R. (1993). *Corticospinal function and voluntary movement*. Oxford: Clarendon Press.
- Porro, C.A., Francescato, M.P., Cettolo, V., Diamond, M.E., Baraldi, P., Zuiani, C, Bazzocchi, M., & di Prampero, P.E. (1996). Primary motor and sensory cortex activation during motor performance and motor imagery: a functional magnetic resonance imaging study. *The Journal of Neuroscience, 16*, 7688-7698.
- Pylyshyn, Z.W. (2002). Mental imagery: in search of a theory. *Behavioral and Brain Sciences, 25*, 157-238.
- Radulescu, P., Adam, J.J., Fischer, M.H., & Pratt, J. (2009). Fitts's Law violation and motor imagery: are imagined movements truthful or lawful? *Experimental Brain Research, 201*, 607-611.
- Raine, S. (2009). The Bobath concept: developments and current theoretical underpinning. In S. Raine, L. Meadows, & M. Lynch-Ellerington (Eds.), *Bobath concept - theory and clinical practice in neurological rehabilitation*. (pp. 1-22). Oxford: Wiley Blackwell.

- Raghavan, P. (2007). The nature of hand motor impairment after stroke and its treatment. *Current Treatment Options in Cardiovascular Medicine*, 9, 221–228.
- Riccio, I., Iolascon, G., Barillali, M.R., Gimigliano, R., & Gimigliano, F. (2010). Mental practice is effective in upper limb recovery after stroke: a randomized single-blind cross-over study. *European Journal of Physical and Rehabilitation Medicine*, 46, 19-25.
- Robertson, D.G.E. & Caldwell, G.E. (2004). Planar kinematics. In D.G.E Robertson, J. Hamill, G. Caldwell, G. Kamen, & S. Whittlesey. (Eds.), *Research methods in biomechanics* (pp 9-34). Champaign, IL: Human Kinetics.
- Rosenbaum, D.A., Meulenbroek, R.J., Vaughan, J., & Jansen, C. (2001). Posture based motion planning: applications to grasping. *Psychological Review*, 108, 709-734.
- Roosink, M. & Zijdwind, I. (2010). Corticospinal excitability during observation and imagery of simple and complex hand tasks: Implications for motor rehabilitation. *Behavioural Brain Research*, 213, 35–41.
- Rothwell, J.C. (1987). *Control of human voluntary movement*. Maryland: Aspen.
- Rothwell, J.C., Traub, M.M., & Marsden C.D. (1980). Influence of voluntary intent on the human long-latency stretch reflex. *Nature*, 286, 496–498.
- Rowland, L. (2000). Diseases of chemical transmission at the nerve-muscle synapse: myasthenia gravis. In E.R., Kandel, J.H. Schwartz, & T.M Jessell (Eds.), *Principles of neural science* (pp.298-308). New York: McGraw-Hill.
- Roy, A., Forrester, L.W. and Macko, R.F. (2011). Short-term ankle motor performance with ankle robotics training in chronic hemiparetic stroke. *Journal of rehabilitation research and development*, 48, 417-430.
- Rosenkrantz, K. & Rothwell, J.C. (2004). The effect of sensory input and attention on the sensorimotor organization of the human motor cortex. *Journal of Physiology*, 561, 307-320.
- Sabaté, M., González, B., & Rodríguez, M. (2007). Adapting movement planning to motor impairments: the motor-scanning system. *Neuropsychologia*, 45, 378–386.

- Saghaei, M. (2004). *Random allocation software*. Iran: University of Isfahan.
- Schmidt, R.A., Zelaznik, H., Hawkins, B., Frank, J.S., & Quinn, J.T. (1979). Motor-output variability a theory for the accuracy of rapid motor acts. *Psychological Review*, *86*, 415-451.
- Schmidt, R. A. (1975). A schema theory of discrete motor skill learning. *Psychological Review*, *82*, 225-260.
- Schmidt, R.A. & Lee, T.D. (2005). *Motor control and learning: a behavioral emphasis*. Champaign: Human Kinetics.
- Schuster, C., Hilfiker, R., Amft, O., Scheidhauer, A., Andrews, B., Butler, J., Kischka, U., & Ettlin, T. (2011). Best practice for motor imagery: a systematic literature review on motor imagery training elements in five different disciplines. *BMC Medicine*, *9*. doi:10.1186/1741-7015-9-75
- Schwartz, J.H. & Westbrook, G.L. (2000). The cytology of neurons. In E.R., Kandel, J.H. Schwartz, & T.M Jessell (Eds.), *Principles of neural science* (pp.67-87). New York: McGraw-Hill.
- Schwartzman, R.J. (1978). A behavioral analysis of complete unilateral section of the pyramidal tract at the medullary level in macaca mulatta. *Annals of Neurology*, *4*, 234-244.
- Sharma, N. Baron, J-C., & Rowe, J.B. (2009a). Motor imagery after stroke: relating outcome to motor network connectivity. *Annals of Neurology*, *66*, 604-616. doi: 10.1002/ana.21810
- Sharma, N., Jones, P.S., Carpenter, T.A., & Baron, J-C. (2008). Mapping the involvement of BA 4a and 4p during Motor Imagery. *NeuroImage*, *41*, 92–99.
- Sharma, N., Pomeroy, V.M., & Baron, J.C. (2006). Motor imagery – A backdoor to the motor system after stroke? *Stroke*, *37*, 1941-1952.
- Sharma, N., Simmons, L.H., Jones, S., Day, D.J., Carpenter, T.A., Pomeroy, V.M., Warburton, E.A., & Baron, J-C. (2009). Motor imagery after subcortical stroke: a functional magnetic resonance imaging study. *Stroke*, *40*, 1315-1324. doi:10.1161/STROKEAHA.108.525766
- Sherrington, C.S. (1947). *The integrative action of the nervous system*. Cambridge:

Cambridge University Press.

- Simon, A.M., Kelly, B.M., & Ferris, D.P. (2009). Sense of effort determines lower limb force production during dynamic movement in individuals with post stroke hemiparesis. *Neurorehabilitation and Neural Repair*, 23,811-818.
- Sirigu, A. & Duhamel, JR. (2001). Motor and visual imagery as two complementary but neurally dissociable mental processes. *Journal of Cognitive Neuroscience*, 13, 910–919.
- Sirigu, A. Duhamel, J-R., Cohen, L. Pillon, B., Dubois, B., & Agid, Y. (1996). The mental representation of hand movements after parietal cortex damage. *Science*, 273, 1564-1568. doi: 10.1126/science.273.5281.1564
- Slifkin, A.B. & Grilli, S.M. (2006). Aiming for the future: prospective action difficulty, prescribed difficulty, and Fitts' law. *Experimental Brain Research*, 174, 746-753.
- Stinear, C., Fleming, M.K., Barber, P.A., & Byblow, W.B. (2007) Lateralization of motor imagery following stroke. *Clinical Neurophysiology*, 118, 1794–1801.
- Stinear, C., Byblow, W. D., Steyvers, A.E., Levin, O., & Swinnen, S. (2006). Kinesthetic, but not visual, motor imagery modulates corticomotor excitability. *Experimental Brain Research*, 168,157–164.
- Stokic, D.S. (2010). Spasticity 30 years later: The truth is still in the eye of the beholder. *Clinical Neurophysiology*, 121, 1789–1791.
- Streeton, R., Cooke, M., & Campbell, J. (2004). Researching the researchers: using a snowballing technique. *Nurse Researcher*, 12, 35-46.
- Smania, N., Paolucci, S., Tinazzi, M. Borghero, A. Manganotti, P., Fiaschi, A. Moretto, G., Bovi, P., & Gambarin, M. (2007). Active finger extension: a simple movement predicting recovery of arm function in patients with acute stroke. *Stroke*, 38, 1088-1090.
- Stroke Association. (2006). *Stroke buried in key Welsh NHS policy*. Retrieved from <http://www.stroke.org.uk/mediacentre/pressreleases/strokeburiedin/html>.
- Stuart, D.S. (2005). Integration of posture and movement: contributions of Sherrington, Hess, and Bernstein. *Human Movement Science*, 24, 621-643.

- Szameitat, A.J., Shen, S., & Sterr, A. (2007). Motor imagery of complex everyday movements. An fMRI study. *Neuroimage*, *34*, 702-713.
- Thies, S.B., Tresadern, P.A., Kenney, L.P., Smith, J., Howard, D., Goulermas, J.Y., Smith, C., & Rigby, J. (2009). Movement variability in stroke patients and controls performing two upper limb functional tasks: a new assessment methodology. *Journal of NeuroEngineering and Rehabilitation*, *6*, doi:10.1186/1743-0003-6-2.
- Turton, A. & Pomeroy, V. (2002). When should upper limb function be trained after stroke? Evidence for and against early intervention. *NeuroRehabilitation*, *17*, 215–224.
- Radulescu, P.V., Adam, J.J., Fischer, M.H., & Pratt, J. (2009). Fitts's Law violation and motor imagery: are imagined movements truthful or lawful? *Experimental Brain Research*, *201*, 607-611. doi: 10.1007/s00221-009-2072-2.
- Ramnani, N. (2006) The primate cortico-cerebellar system: anatomy and function. *Nature Reviews Neuroscience*, *7*, 511-522.
- Rizzolatti, G. & Fabbri-Destro, M. (2007). Mirror neurons: from discovery to autism. *Experimental Brain Research*, *200*, 223-237.
- Rodrigues, E.C., Lemos, T., Gouvea, B., Volchan, E., Imbiriba, L.A., & Vargas, C.D. (2010). Kinesthetic motor imagery modulates body sway. *Neuroscience*, *169*, 743–750.
- Ros-Morente, A., Vilagr a-Ruiz, R.,Rodriguez-Hansen, G., Wigman, J.H. & Barrantes-Vidal, N. (2011). Proceso de adaptaci n al castellano de la escala de evaluaci n comunitaria de experiencias ps quicas (CAPE), *Actas Espa oladas De Psiquiatr a*, *39*, 95-105.
- Rosenbaum, D., Meulenbroek, R.J., Vaughan, J., & Jansen, C. (2001). Posture-based motion planning: applications to grasping. *Psychological Review*, *108*, 709-734.
- Sireci, S.G., Yang, Y., Harter, J. & Ehrlich, E.J. (2006). Evaluating guidelines for test adaptations : a methodological analysis of translation quality. *Journal of Cross-Cultural Psychology*, *37*, 557-567.
- Van Deursen, R.W.M. & Everett, T. (2005). Biomechanics of human movement. In



- M. Trew & T. Everett (Eds.), *Human movement: an introductory text* (pp37-66). London: Churchill Livingstone.
- Van Vliet, P.M. & Sheridan, M.R. (2007). Coordination between reaching and grasping in patients with hemiparesis and healthy subjects. *Archives of Physical Medicine and Rehabilitation*, 88, 1325-31.
- Vincent, W.J. (1999). *Statistics in kinesiology*. Champaign, IL: Human Kinetics.
- Voisin, J.I.A., Mercier, C., Jackson, P.L., Richards, C.L., & Malouin, F. (2011). Is somatosensory excitability more affected by the perspective or modality content of motor imagery? *Neuroscience Letters*, 493, 33–37.
- Von Holst, E. (1954). Relations between the central nervous system and the peripheral organs. *British Journal of Animal Behaviour*, 2, 89-94.
- Wagner, J.M., Rhodes, J.A., & Patten, C. (2008). Reproducibility and minimal detectable change of three-dimensional kinematic analysis of reaching tasks in people with hemiparesis after stroke. *Physical Therapy*, 88, 652– 663.
- Walter, C.B. (1984). Temporal quantification of electromyography with reference to motor control research. *Human Movement Science*, 3, 155-162.
- White, A. & Hardy, L. (1995). Use of different imagery perspectives on the learning and performance of different motor skills. *British Journal of Psychology*, 86, 191-216.
- Wiener, N. (1948). *Cybernetics or control and communication in the animal and the machine*. Cambridge, MS: The MIT Press.
- Wing, A.M. & Fraser, C. (1983). The contribution of the thumb to reaching movements. *The Quarterly Journal of Experimental Psychology A*, 35, 297-309.
- Woldag, H. & Hummelsheim, H. (2002). Evidence-based physiotherapeutic concepts for improving arm and hand function – a review. *Journal of Neurology*, 249, 518-528.
- Wolpert, D.M., Ghahraman, Z., & Flanagan, J.R. (2001). Perspectives and problems in motor learning. *Trends in Cognitive Sciences*, 5, 487-494.
- Woodworth, R.S. (1899). The accuracy of voluntary movements. *Psychological Review Monograph Supplement* 3, 1-119.

- World Medical Association General Assembly. (2008). *World Medical Association declaration of Helsinki: ethical principles for medical research involving human subjects*. Retrieved from <http://www.wma.net/en/30publications/10policies/b3/17c.pdf>
- Wurtz, R.H. & Kandel, E.R. (2000) Central visual pathways. In E.R., Kandel, J.H. Schwartz, & T.M Jessell (Eds.), *Principles of neural science* (pp.523-547). New York: McGraw-Hill.
- Yekutieli, M. & Guttman, E. (1993). A controlled trial of the retraining of the sensory function of the hand in stroke patients. *Journal of Neurology, Neurosurgery, and Psychiatry*, 56, 241-244.
- Yue, G. & Cole, K.J. (1992). Strength increases from the motor program: comparison of training with maximal voluntary and imagined muscle contractions. *Journal of Neurophysiology*, 67, 1114-1123.
- Zatsiorsky, V.M. (1998). *Kinematics of human motion*. Champaign, IL: Human Kinetics.
- Zatsiorsky, V.M., Li, Z-M., & Latash, M.L. (2000). Enslaving effects in multi-finger force production. *Experimental Brain Research*, 131, 187-195.
- Zimmermann-Schlatter, A., Schuster, C., Puhan, M., Siekierka, E., & Steurer, J. (2008). Efficacy of motor imagery in post-stroke rehabilitation: a systematic review. *Journal of NeuroEngineering and Rehabilitation*, 5, 8. doi:10.1186/1743-0003-5-8.