Is there a difference in cortical representation between dominant and non-dominant arm muscles of elite badminton players?

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IS THERE A DIFFERENCE IN CORTICAL REPRESENTATION BETWEEN DOMINANT AND NON-DOMINANT ARM MUSCLES OF ELITE BADMINTON PLAYERS?

BY

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Bachelor of Applied Science (Sports Science)

A thesis submitted in partial fulfilment of the requirements for the award of

Bachelor of Applied Science (Sport Science) with Honours

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USE OF THESIS

The Use of Thesis statement is not included in this version of the thesis.
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Thank you to my parents for still looking after me, and for bringing me into this world to contribute my piece to science.
DECLARATION

“I certify that this thesis does not incorporate without acknowledgment any material previously submitted for a degree or diploma in any institution of higher education; and that to the best of my knowledge and belief it does not contain any material previously published or written by another person except where due reference is made in the text”.
ABSTRACT

Training for sport involves the development of skill and coordination. The physiological changes associated with skill acquisition are complex and at present poorly understood. One of the areas in the central nervous system thought to be involved with skill acquisition is the cerebral motor cortex where localised areas are responsible for controlling specific muscle groups. Learning or improving a motor skill may require reorganisation of the cortical areas controlling relevant muscles to accommodate the new skill. To test this idea we studied a group of elite badminton players that were highly skilled in their dominant playing arm. Transcranial magnetic stimulation was used to stimulate the motor cortex, and surface electrodes recorded the evoked muscle response. A forearm wrist flexor muscle was examined in this study and a comparison was made between the representation of this muscle on the motor cortex, with that of the contralateral untrained muscle. The experiments were repeated in a control group of normal subjects to assess if any interhemispheric differences occur under normal conditions. In order to quantify the results, topographic maps were produced illustrating the area of representation of each muscle on the cortex, and the centre of the map. The maps showed the representation taken from the amplitude of the evoked response, and the silent period following this response. Comparison of the maps revealed no significant differences between the trained and untrained muscles, in the size of the representation, or the excitability of the area. The location of the maps was slightly posterior for the athlete group, particularly in the dominant hemisphere, which also showed a non-significant more lateral placement compared to the control group. Skill differences between the dominant and non-dominant arm in badminton players is not reflected in the representation of the muscles on the motor cortex.
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CHAPTER ONE

Introduction

Sport-related scientific research, endeavours to improve athletic performance through an enhanced understanding of the functioning of the human body. One of the areas of interest to sports scientists is the acquisition of motor skills. Skill can be defined as the precision and efficiency with which one executes a motor task. Knowledge of skill acquisition can assist in the planning of training programs to optimise the development of important sport skills. This could lead to faster development of new skills and maximise coordination and skill in elite athletes. The wider community may also benefit as more effective teaching strategies are implemented to improve motor learning in people with coordination problems.

The physiological changes associated with skill acquisition are poorly understood and probably involve numerous anatomical structures within the central nervous system (CNS). One area of the CNS thought to be involved with skill acquisition is the primary motor cortex, where localised areas are responsible for controlling specific muscle groups. The role of the motor cortex in the sequence of events controlling human voluntary movement, and also its anatomical position, make the motor cortex both convenient and appropriate to examine in this study.

The purpose of this study was to compare the corticomotor excitability and cortical representation of muscles which are highly trained for skilled movements, with untrained contralateral muscles. Comparison of the two will indicate if prolonged changes occur in the motor cortex with skill development. It was hypothesised in this study that the skill development in training for a sport would cause a change in the size
of the representation of the target muscle in the motor cortex, or a change in the excitability of that area.

The localised areas on the motor cortex controlling muscles of the upper extremity are the most accessible and popular for central stimulation and mapping experiments. These muscles also have the lowest thresholds for activation (Brouwer & Ashby, 1990), and hence more comfortable stimulus intensities can be used. Common muscles to be studied are in the hand, arm and shoulder and few experiments have examined the forearm muscles. For this reason and the fact that these muscles are used in many sporting skills this study has examined a wrist flexor muscle. In elite badminton players, the dominant arm is highly skilled, particularly in specific wrist movements. The wrist flexor muscles are used in a number of badminton shots (Lo & Stark, 1991; Broer & Houtz, 1967) however, the frequency of activation of these muscles during a game or training is not specified. By examining the muscles of sportspersons that are skilled unilaterally, it is possible to use the non-dominant side to assess changes that may occur as a result of activity and/or skill development.

Original investigation of the function of the motor cortex used direct electrical stimulation of the exposed cortex (Penfield & Boldrey, 1937, cited in Wassermann, McShane, Hallet, Cohen, 1992). Currently, stimulation of the cortex can be achieved using electrical stimulation with electrode placement either sub-dural or on the skull, or by transcranial magnetic stimulation (TMS). TMS uses a large pulsed magnetic field to induce currents within the nervous tissue. TMS can be used to examine the cortical motor outputs in humans, by stimulating over the scalp of the subject and recording muscular activity from the contralateral or ipsilateral target muscle. More specifically,
this method can be used to examine cortical excitability, and to map the representational area of specific muscles on the motor cortex.

Contrary to earlier ideas, recent research has indicated that neural remodelling can occur in the mature nervous system (Aglioti, Bonazzi, Cortese, 1994). There is clear evidence that the cortical representation of muscle groups can change in certain pathological conditions, but few studies have examined the potential for change to occur under normal conditions. Pascual-Leone, Grafman and Hallet (1994) studied the representation of muscles before and after learning a simple motor task in normal subjects. The findings showed evidence for significant differences in the representation of the target muscles with training. However, information on motor cortex plasticity as a result of athletic training for highly skilled movements is absent from the literature. This is the first study to investigate the nature of the training response of athletes by looking at the control level of voluntary motor activity, and assessing interhemispheric differences in cortical motor outputs as they project to trained and non-trained muscles.
CHAPTER TWO

Literature Review

_Lateral preference_

In performing both simple and sophisticated unilateral motor tasks, most individuals express a clear lateral preference to the side of the body performing the task. This occurs in every day activities and in sporting situations where the most proficient hand or foot is used to execute the task. There has been a tendency to view skill, strength, and preference as relatively interchangeable indicators of the dominant hand; however, evidence suggests that they are separable aspects of behaviour perhaps mediated by different mechanisms (Porac & Coren, 1981). The concept of lateral preference suggests an element of choice. For example, the preferred hand is the hand chosen when only one hand can be used for a given activity. One would assume then that the chosen member of paired limbs would be the better or more proficient of the two but this is not always true. Strength and skill, for example can be affected by environmental factors and may be independent of preference.

Much of the research in lateral preference and sensorimotor performance has been in the interest of isolating variables that might help athletes obtain maximum levels of proficiency. Porac and Coren (1981) studied athletes from a wide variety of sports (including badminton) and varying levels of proficiency to determine how sidedness and preference affected sports performance. They suggest that in the same way that we are right or left handed, we are also right or left footed, eyed, and eared. They also suggested that proficiency in badminton is very much related to hand eye coordination. If
the preferred eye is on the same side of the body as the preferred hand, the individual has a larger useful visual field in the sector of the environment where most of the activity is occurring. If visual input is monitoring both the projectile to be hit and the racquet that will strike it, ipsilateral hand-eye positioning means that both the target and the racquet will enter the visual field of the preferred eye earlier than would be the case if monitoring was done by a contralateral preferred eye. In this case the useable visible field would be obstructed by the bridge of the nose. With the earlier appearance of the hand to the ipsilateral preferred eye, there is more time to make minor adjustments to the ongoing swing and hence improve overall accuracy. It appears then that the use of both the preferred hand and preferred eye contribute independent advantages that act to increase speed of performance. They concluded that right or left sidedness alone does not seem to affect sports proficiency. The combination of lateral preferences, and ambisidedness versus consistent preference is more predictive of ability.

The apparent physical symmetry of the human body has created a great deal of interest for research about the mechanisms that give rise to the functional asymmetries seen in the common displays of handedness and other lateral preferences. Many theories have been developed attempting to explain why one hand is dominant and also how it became dominant. Some early attempts were made to correlate handedness with anatomical and physiological asymmetries, others link it to genetic factors explaining that handedness is determined before birth (Porac & Coren, 1981). Other theories suggest that the reason most people are right handed may be due to left handed individuals having learned to use their right hand skilfully out of necessity, because many tasks in today's society require the skilful use of the right hand. No definite conclusions have
been drawn from these approaches and contemporary researchers have shifted their attention to neural control systems.

**Hemispheric asymmetry, cerebral dominance and handedness**

Hemispheric asymmetries in the brain have been the focus of more recent research explaining lateral preferences. Structural differences include a heavier, larger or denser left hemisphere (Bastian, 1869; Boyd, 1861, cited in Porac & Coren, 1981), a greater complexity of and a greater number of convolutions in the left hemisphere (Bateman, 1869; Cunningham, 1902, cited in Porac & Coren, 1981), and substantial differences in the fissure lengths (Porac & Coren, 1981), specifically the Sylvian fissure (LeMay & Culebras, 1972; Rubens et al., 1976, cited in Koff, Naeser, Pieniadz, Foundas, Levine, 1986). The observation of such asymmetries has previously been linked to lateral preference behaviour, however, sufficient evidence has not been produced to support these claims. Porac and Coren (1981) state that hand preference is not predictable directly from, nor strongly correlated with, anatomically definable asymmetries in the brain.

Cerebral dominance refers to one side of the brain being more important for certain functions than the other side (Galaburda, LeMay, Kemper, Geschwind, 1978). Each hemisphere is dominant for particular functions: for example the left for language and certain spatial functions; the right for other spatial functions, attention, and at least some aspects of emotion (Geschwind, 1983). One of the behaviours in which dominance effects are prominent is handedness. With regard to motor function, the cerebral hemisphere controlling the preferred hand is referred to as the dominant hemisphere for handedness. With the majority of the population being right handed (Porac and Coren,
the dominant hemisphere for handedness is generally the left. Haaland and Harrington (1989, cited in Kim, Ashe, Hendrich, Ellermann, Merkle, Ugurbil, Georgopoulos, 1993) reported that motor function deficits resulting from a left hemispheric lesion are more pronounced than those resulting from a right hemispheric lesion. These results indicate that dominance may occur for handedness, however the research does not explain whether the dominance is due to hemispheric asymmetries or some other factor. Galaburda et al. (1978) suggests that structural differences between the hemispheres may underlie cerebral dominance. Koff et al. (1986) concluded that if structural differences are substrates for handedness, it appears more likely that they are related to at least some aspects of language dominance than to cerebral dominance for handedness.

The fact that one hemisphere may be dominant for handedness is particularly relevant for this study as interhemispheric comparisons may reveal interhemispheric differences in the representation of the wrist flexors for both groups of subjects. If this is the case it may be due to hemispheric asymmetries. Wilson, Thickbroom, Mastaglia (1993a) investigated the representations of two intrinsic hand muscles of normal subjects using TMS mapping for both the dominant and non-dominant hands. The result of this study showed no significant difference between the two sides indicating that handedness alone, is not sufficient to show a difference in cortical representation for muscles. It is also unknown if other hand muscles or more proximal muscles, show the same results. Excitability differences in the motor cortex and handedness has also been investigated (Macdonell, Shapiro, Chiappa, Helmers, Cros, Day, Shahani, Phil, 1991). The results showed lower cortical thresholds for the activation of spinal motor neurons for the dominant hand muscle (abductor digitii minimi).
Motor cortex and voluntary movement

Current concepts regarding the motor areas of the cerebral cortex, derived from studies of cortical lesions and metabolic activity of the cerebral cortex during motor tasks, delineate three major motor areas in humans (Fox, Bowers, Foss, 1988). These are the primary motor area (area 4 of Brodmann’s chart, precentral gyrus), the premotor area (Brodmann’s area 6, gyrus anterior to the pre-central gyrus), and the supplementary motor area (medial frontal cortex anterior to the premotor area). The primary motor cortex is essential to voluntary action, and destructive focal lesions in this area cause paralysis of voluntary movement.

Measurements of regional cerebral blood flow (CBF) and metabolism have demonstrated that the primary motor cortex is activated only when voluntary movements are executed (Mazziotta & Phelps, 1984). The pyramidal tract, made up of long axons of the pyramidal cells, is the route used to send impulses from the motor cortex to the lower motor neurons of the spinal cord which terminate in skeletal muscle. Most of the pyramidal tract fibres cross over before entering the spinal cord so that the right motor cortex controls the muscles of the left side of the body and vice versa. The pre-motor cortex is especially concerned with the acquisition of specialised motor skills. Lesions involving the premotor region generally cause a transient deficit in acquired motor skills (Freund & Hummelsheim, 1985; Fox, Bowers, Foss, 1988). The pre-motor area may respond to direct electrical stimulation with isolated movements of individual contralateral muscles, but significantly higher stimulation is required than for the primary motor cortex (Ransom & Clark 1959). The supplementary motor area is also activated...
during voluntary motor activity, and lesions in this area may result in a reduction in spontaneous motor activity that may be accompanied by a reduction in speech production. Roland (1984) found that an increase in metabolic activity in this area during the planning and execution of voluntary movement occurs during routine voluntary movements (such as walking) and non-routine voluntary movements (such as performing a new skill or activity). Stimulation of the supplementary motor area produces synergic and bilateral movements, such as raising of the contralateral arm with turning of the head and eyes toward the arm.

*Stimulation of the motor cortex*

Direct stimulation of the motor cortex will evoke a muscular contraction and the muscle activated will depend on the point of stimulation. Early studies have used electrical stimulation applied directly onto the cerebral cortex (of patients undergoing craniotomy) to investigate which area of the cortex controls the different muscle groups (Penfield and Boldrey, 1937, cited in Wassermann, McShane, Hallet, Cohen, 1992). Several attempts to stimulate the brain through the intact skull were made in the 1950's using trains of electrical stimuli delivered through electrodes over the motor strip and most were unsuccessful (Rothwell, Day, Thompson, Marsden, 1990). In 1980 Merton and Morton devised a method of electrical stimulation through the intact scalp that achieved an evoked muscular response. They claimed that previous experiments attempting to stimulate through the intact scalp had problems with the electrical resistance given by the skull and scalp, and experiments have been stopped by pain or otherwise failed. By using brief but very high voltage shocks they found that this reduced the effective resistance and it was possible to stimulate the cortex without undue
discomfort. Monopolar and bipolar electrical stimulation has been used in transcranial stimulation studies, with the bipolar stimulation either anodal or cathodal. Stimulation through electrodes on the scalp reduce the localisation of the stimulus on the cortex, and more localised stimuli are given with electrodes on the brain surface however the application of this technique is limited.

Magnetic stimulation was introduced initially for peripheral nerve stimulation in 1985 and was soon adopted for brain stimulation. Unlike the electrical method of stimulation, magnetic stimulation is relatively painless, since the currents induced on the scalp are small. With TMS the the rapid discharge of current through a coil held over the scalp generates a time varying magnetic field which in turn induces current flow in the underlying brain (Barker, Jalinous, Freeston, 1985). Current flow induced in the cortex is sufficient to activate pyramidal tract neurones (PTNs) trans-synaptically (Day, Dressler, Martens de Noordhout, Marsden, Nakashima, Rothwell, Thompson, 1989) and, under some circumstances directly (Berardelli, Inghilleri, Cruccu, Manfredi, 1990), and can lead to multiple descending volleys in PTNs projecting to the motor neuron. When the membrane excitability of the motor neuron reaches threshold, a measurable response in the surface EMG may be recorded, the motor evoked potential (MEP).

Muscular facilitation

Many stimulation experiments examining the human motor cortex have stimulated the cortex of relaxed individuals, and the threshold levels to evoke MEPs were thought to be related directly to the stimulus intensity. Current ideas are that the degree of muscular response and threshold depend on both the stimulus intensity (Valls-Sole, Tolosa, Pujol, 1992), and the state of relaxation/contraction of the target muscle (Rothwell, Thompson,
Day, Dick, Kachi, Cowan, Marsden, 1987). A tonic voluntary contraction of the target muscle during stimulation, decreases the threshold (Mazzocchio, Rothwell, Day, Thompson, 1994), and decreases the onset latency of the MEP (Hess, Mills, Murray, 1987; Day, Thompson, Dick, Nakashima, Marsden, 1987).

*Evoked potential mapping of the motor cortex*

By means of the early stimulation experiments, it has been possible to outline most of the motor projection from the motor cortex. Various body parts have been represented or mapped onto the motor cortex, and the representation of each body part, is proportionate in size to the skill with which the part is used in fine voluntary movement. The accuracy of the early stimulation experiments can be questioned due to the observation of gross body movements following stimulation of the cortex, and the body part representations are only an estimation. Following the development of increasingly focal stimulation techniques, TMS has been applied in exploratory studies of the organisation of the human corticomotor representation. Current techniques used for mapping require recording of the muscular response following the stimulation of the motor cortex, and allow the representation of individual muscles to be mapped.

*MEP maps*

By stimulating specific scalp sites and measuring the MEP responses it is possible to map the representation of individual muscles on the cortex. Wilson et al. (1993a) mapped the representation of two intrinsic hand muscles on the motor cortex in normal subjects. The study used TMS to stimulate the cortex, and surface electrodes placed over abductor pollicis brevis (APB) and abductor digiti minimi (ADM) recorded the muscular response.
The results showed the mapped representations of APB and ADM were large and overlapping but that there was a statistically significant separation of the two areas, the APB being more laterally placed than the ADM area. The two representations showed no significant interhemispheric differences which suggests that handedness may not affect the representation of hand muscles on the motor cortex. The results also indicated that this method could produce reliable and reproducible maps of the muscular representations on the motor cortex. Wassermann, McShane, Hallet and Cohen (1991) examined four upper extremity muscles bilaterally in normal subjects. TMS was used to map the representations, and the maps were described in terms of number of excitable scalp positions, amplitude of MEPs, scalp positions for evoking the largest MEP amplitudes, and threshold for producing MEPs. Distal muscles were found to have larger representations with higher amplitude MEPs and lower thresholds. Both the biceps and deltoid on the left side had larger representations and higher MEP amplitudes than on the right contrary to the interhemispheric comparisons for more distal muscles as found by Wilson et al. (1993a).

_Silent period maps_

Transcranial magnetic stimulation of the motor cortex has both excitatory and inhibitory effects on muscles (Rothwell, Thompson, Day, Boyd, Marsden, 1991). Stimulation during voluntary contraction produces an MEP followed by a silent period (SP) in the EMG (Cantello, Gianelli, Civardi, Mutani, 1991) before a return of the voluntary EMG signal. Other studies have characterised the TMS SP in hand muscles (Wassermann, Pascual-Leone, Valls-Sole, Toro, Cohen, Hallet, 1993; Wilson, Thickbroom, Mastaglia, 1993b), and more proximal muscles of the arm, and also following stimulation of the
ipsilateral hemisphere (Wassermann, Pascual-Leone, Hallet, 1994). Wilson et al. (1993b) mapped the SP on the motor cortex by measuring its duration following stimulation at multiple scalp sites, and compared the SP representation with that of the MEP maps for the same muscle (APB). The results from this study showed the silent period representation to surround and encompass the MEP area.

*Plasticity of the motor cortex*

The representation of muscles in the motor cortex have been found to be capable of change in people who have had amputations (Cohen, Bandinelli, Findley, Hallet, 1991). The study examined the representation of muscles proximal to the stump (biceps and deltoid) and contralateral muscles by MEPs in response to TMS. Stimulation evoked larger MEPs, recruited a larger percentage of the motor neuron pool, and elicited MEPs at lower intensities of stimulation in muscles ipsilateral to the stump than in contralateral muscles. Also, muscles ipsilateral to the stump were found to be activated from a larger area than those contralateral to the stump. The findings where supported in experiments analysing the motor cortex of subjects following lower limb amputation in man (Fuhr, Cohen, Dang, Findley, Haghighi, Oro, Hallet, 1992). Similar studies examining subjects with spinal cord injuries have also been reported (Levy, Amassian, Traad, Cadwell, 1990; Topka, Cohen, Cole, Hallet, 1991). These studies identified a pattern of motor system reorganisation that results in enlarged muscle representation areas and larger motor evoked potentials for muscles immediately proximal to the lesion. Furthermore, findings are supported in animal models where motor outputs are reorganised after peripheral nerve lesions (Merzenich, Kaas, Wall, Sur, Nelson, Felleman, 1983; Huerta, Wall, Kaas,
1986; Kalaska & Pomeranz, 1979), removal of body parts (Kelahan, Ray, Carson, Massey, Doetsch, 1981; Merzenich, Nelson, Stryker, Cynder, Shoppmann, Zook, 1984; Pons, Garraghty, Ommaya, Kaas, Taub, Mishkin, 1991; Calford, Tweedale, 1990) and reversible limb deafferentation by local anaesthesia (Metzler & Marks, 1979). Such capability of the motor cortex to modulate outflow to specific muscle groups introduces the possibility that these mechanisms may play a role in adaptation to environmental needs, such as the learning of precision tasks, enhanced performance, special training conditions and exercise.

The nervous system may undergo changes according to the patterns of use, thereby providing a substrate for the acquisition of new skills. Proficiency in a motor task may require a change in the cortical motor outputs to accommodate new skills (Brasil-Neto, Cohen, Pascual-Leone, Jabir, Wall, Hallet, 1992). Brasil-Neto et al. (1992) suggest that human motor outputs can experience both short and long term changes. The short term changes are referred to as "modulation" and the long term changes as "reorganisation". Modulation of muscular representation on the motor cortex has been demonstrated immediately after learning a motor task (Pascual-Leone, Grafman, Hallet, 1994). Normal subjects were required to learn sequential finger movements, and TMS was used to map the cortical motor outputs to the right first dorsal interosseus, abductor digiti minimi, forearm finger flexors, and abductor pollicus brevis muscles. Results from this study showed that modulation of the cortex occurs rapidly during the learning phase of the task by the increase of skill development, and soon after this phase there is a rapid return of the cortical motor outputs to their baseline topography. This is also evident in studies examining regional cerebral blood flow and acquisition of motor skills (Seitz, Roland, Bohm, 1990; Grafton, Mazziotta, Presty, 1992).
Weill et al. (1992, cited in Pascual-Leone, Grafman, Hallet, 1994) suggest that as a motor sequence is learned, the contribution of the motor cortex is attenuated and other brain structures assume more active roles in the execution of the task. Although the relationship between changes of motor thresholds in relation to motor learning have not been described in the literature, an increased excitability of the area of representation following ischaemic nerve block (Brasil-Neto, Valls-Sole, Pascual-Leone, Cammarota, Amassian, Cracco, Maccabee, Cracco, Hallet, Cohen, 1993), and amputation (Cohen et al., 1991) has been reported to be associated with motor reorganisation.

Brasil-Neto et al. (1992) examined rapid reversible modulation of human motor outputs after transient deafferentation of the forearm in normal subjects using TMS. To study the timing of these changes they recorded MEPs in the arm muscles before, during, and after anaesthetic block of the forearm and hand. The amplitudes of MEPs from biceps, which was the muscle immediately proximal to the block, gradually increased with anaesthesia and then returned to preanesthesia levels shortly after the anaesthesia was ended. MEPs from the contralateral arm were unaffected. They suggest that similar fast changes in motor output organisation may be associated with learning skilled motor tasks in humans. The rapid time course of this modulation is most compatible with unmasking of previously existing connections, perhaps as a result of decreased inhibition or increased synaptic efficacy in existing neural circuits (Asanuma & Keller, 1991). The acquisition of new skills appears to cause short-term changes to the motor cortex and could lead to structural changes in the intracortical and subcortical networks as the skill becomes more learned and automated (Pons et al., 1991; Ramachandran, Rogers-Ramachandran, Stewart, 1992).
Similar findings have come from studies of plasticity of the sensorimotor cortex representation of the reading finger in Braille readers. Pascual-Leone and Torres (1993a) recorded somatosensory evoked potentials (SEPs) in one group of subjects and controls, and TMS was used in another group of subjects. TMS was used to interfere with the functioning of the sensory cortex, and from this they determined the size of the sensorimotor cortical representation of the tip of the index finger. The results of the reading finger were then compared to those of the contralateral non-reading finger. The findings from this experiment suggest that reading Braille is associated with the expansion of the sensorimotor cortical representation of the reading finger. The motor cortical outputs of the FDI and ADM muscles of Braille readers has also been examined bilaterally (Pascual-Leone, Cammarota, Wassermann, Brasil-Neto, Cohen, Hallet, 1993b). In the control group, the interhemispheric representations were not significantly different for both muscles. In the Braille group, the reading hand had a significantly larger representation for the FDI compared to the contralateral muscle. The representation of ADM however, was significantly smaller for the dominant hand. They suggest that the cortical representation of the reading finger in the Braille readers is enlarged at the expense of the representation of other fingers. They also explain that these differences were not due to differences in motor thresholds. The subjects in the Braille studies were proficient adult Braille readers that had learned Braille in childhood. The fact the these changes are present in Braille readers suggest that long term changes to the motor cortex representation may occur as a result of practising a skill on a regular basis over a long period of time. Elite athletes may show the similar long term changes from years of regular training for skilled movements.
Summary

Throughout life specific molecular, biochemical, electrophysiological and structural changes take place in central nervous system neurons and neuronal networks in response to activity and behaviour (Cotman and Nieto-Sampedro, 1982; Farley and Alkon, 1985). These plastic changes are part of the structural and physiological processes for recovery of function after injury, learning and memory (Marshall, 1984; Lederhendler and Alkon, 1986; Kaas, 1991). Plasticity in the human motor system has been studied using transcranial magnetic stimulation of individuals following amputation (Cohen et al., 1991; Furh et al., 1992) and spinal cord injuries (Topka et al., 1991), and a pattern of motor system reorganisation that results in enlarged muscle representation areas of the motor cortex and larger motor evoked potentials for muscles immediately proximal to the lesion has been demonstrated. Such capability of the motor cortex to modulate outflow to specific muscle groups introduces the possibility that these mechanisms may play a role in adaptation to learning of precision tasks, enhanced performance, special training conditions and exercise.
CHAPTER THREE

Methods

Subjects

Studies were performed on eight elite badminton players and nine control subjects with no known neurological disorders. The badminton group (current and former state representatives) included six males and two females, aged 21-30 years (mean, 25.4 yrs) of which all subjects were right handed as assessed by a standard handedness questionnaire (Bryden, 1976). The control group included eight males and one female, aged 19-31 years (mean, 24.4 yrs) of which eight were right handed and one was left handed. In each subject the corticomotor representation of the flexor carpi ulnaris (FCU) muscles of both arms was investigated. All subjects attended one testing session only, during which, both FCU muscles were examined (dominant arm first). The subjects were tested at the Australian Neuromuscular Research Institute laboratories and each session lasted approximately two hours. Each subject was informed of the procedures involved in participation, and signed a consent form prior to participation in the study (Appendix A). The project had approval from the Human Rights Committee of the University of Western Australia.

Stimulation

A Magstim 200 magnetic stimulator with a 50mm diameter figure eight coil was used. The stimulator coil was held in position against the scalp, with the centre of the figure eight coil (as previously located by X-ray) over the site to be stimulated. The
orientation of the coil can affect the recorded results due a change in the direction of current flow within the cortex. To maintain consistency, the coil was held in the same orientation for all sites stimulated (tangential to the skull with the handle posterior).

**Stimulus sites**

To locate the stimulus sites, a flexible, translucent, rubber cap was fitted over the scalp of the subject with pre-marked sites at spacings of one centimetre (Fig. 1). The cap was held in place by two velcro straps and positioned using anatomical landmarks to locate the centre of the cap on the vertex of the scalp. Measurements between the nasion and inion, and the left and right preauricular crease were used to locate the vertex at the mid-point and intersection of the nasion-inion line, and the inter-aural line. Stimulus sites were located using a latitude/longitude based coordinate system. Latitude was defined as the distance over the scalp from the nasion-inion line, and longitude as the distance from the inter-aural line.

**Recording of muscular response**

Surface electromyograph (EMG) electrodes were placed over the flexor carpi ulnaris muscle of each arm. The active electrode was placed over the motor point of the muscle, with the inactive electrode 10mm distal. The earth electrode was placed over the lateral epicondyle of the humerus. The amplified signal was high pass filtered at 10 Hz and low pass at 2 kHz, and the digitised data was collected at a rate of 200 Hz for 500 ms after TMS.
Facilitation

A muscle in a slightly contracted state will be activated by TMS at a lower stimulus intensity than a relaxed muscle (Mazzocchio et al., 1994). By using a lower stimulus intensity and slightly contracting the target muscle, neighbouring muscles are less likely to be activated during the experiment. It was important that surrounding muscles were activated as little as possible, as this may cause interference in the EMG recording. A method of facilitation of the FCU was adopted in this study in an attempt to isolate this muscle (as illustrated in Figure 2). Subjects were carefully instructed on the manoeuvre involved in facilitation of the FCU, and were given ample time to familiarise themselves with the technique. The arm being examined was rested on a pillow in a supinated position with the wrist adducted to 50% of its range of motion, and flexed to 50% of its range of motion. During facilitation, the muscle was contracted isometrically in this position using the opposite hand as a resistance. To keep consistency for dominant/non-dominant muscles and between subjects, it was necessary to quantify the contraction level for facilitation of FCU. Each subject performed an isometric maximal voluntary contraction (in the above facilitation position) against a manual restraint for five seconds. The root mean square ( rms) of the EMG interference patterns during the five second contraction was used as a measure of the maximal voluntary EMG activity. During stimulation, subjects were required to maintain facilitation of the FCU muscle at 10% of the maximal voluntary contraction (MVC) for that muscle. A computer (286-PC) displayed the level of contraction as feedback to the subject to maintain that level. The display showed a bar graph illustrating the current level of contraction, which was updated approximately every 500 ms. The computer allowed stimulation to occur only if
the subject maintained the contraction within the range of 10% +/- 3% of the maximal contraction for 1.5 sec.

**Thresholds**

To familiarise each subject with the sensation of magnetic stimulation, four stimuli were given. The site of stimulation was in the region expected to control FCU (as determined by previous experiments) at a longitude of 0cm and a latitude of 4cm from the vertex. The stimulation intensity was 30% of the stimulator output (output range of the Magstim 200 being 0-100%) which in most cases was below the subject’s threshold and caused no muscular response. This was repeated at increasing intensities until each stimuli produced distinct motor evoked potentials (MEPs) with an amplitude clearly above that of the background level of EMG. This usually occurred at approximately 70-75% of the stimulator output. Four stimuli were recorded, at three separate sites moving laterally along the interaural line, to determine which site produced the largest response at the same intensity. The site with the largest response was closest to the centre of the area controlling FCU. This procedure was done to locate the optimum site for determination of the thresholds responses.

To determine the threshold level of stimulation required to produce MEPs in each subject, the optimal site (as described above) was stimulated at 30% intensity and repeated at increments of 5% until an intensity was reached where individual waveforms had ceased to become larger, or 100% intensity stimulation had been achieved. This stage was often accompanied by MEPs decreasing in amplitude and becoming more polyphasic, possibly due to contribution from neighbouring muscles to the EMG. At each intensity, four stimuli were given 5sec apart, and the EMG level was recorded. Threshold
for the mapping procedure was defined as the intensity at which at least two out of four stimuli evoked a MEP discernible above background EMG. Figure 3 illustrates recorded MEP responses during the thresholding procedure. The left hand axis numbers each 500msec EMG trace and the base axis gives an indication of the time scale for which each event occurs. Each set of four stimuli represents a 5% increment in intensity starting with epochs 1-4 at 30%. With each increase in intensity the MEP becomes larger and always begins at approximately 20msec following a stimulus. Associated with the increase in MEP amplitude is the length of the silent period in the following EMG. The peak-to-peak amplitude of the MEPs was averaged and recorded for each intensity. Threshold data for MEP amplitude at each stimulus intensity was plotted to produce threshold curves. A line of best fit was then calculated to the data, and a precise value for the threshold level was determined from the intensity at which the line of best fit intersected a line representing two standard deviations above the mean background EMG level. The calculated threshold was used for interhemispheric and intergroup threshold comparisons.

**Mapping of the Motor Cortex**

The methodology for mapping of the motor cortex has been previously described (Wilson et al., 1993a) and this study has followed the same procedure for stimulating the cortex, compiling the map and interpreting the data.

During the mapping process, the stimulator intensity was set at the lowest threshold obtained over each hemisphere separately, plus 20% of stimulator output to ensure that the intensity was high enough to evoke a response, and to maintain a consistent level above threshold for each hemisphere. The difference in mapping
intensities between hemispheres was within 10% in all but one subject. The first site stimulated for mapping was the same site used to examine thresholds, being close to the estimated centre of the motor area for FCU. Mapping this muscle on the motor cortex required the stimulation of all sites around the estimated centre of the map. At each stimulus site (1cm equidistant in latitude; 2cm in longitude) moving away from the estimated centre, the MEP response became smaller, until a site was reached where stimulation caused no measurable MEP response discernible above background EMG. All sites around the estimated centre were stimulated until no measurable response was recorded after stimulation and this signified the border of the representation for the target muscle. This generally required the stimulation of 25-30 sites. Four muscle responses to stimulation of each scalp position were recorded and the average of the four responses was assigned to represent the scalp position stimulated.

Map compilation and interpretation

For each hemisphere, MEP waveforms from each site were reviewed off line by the experimenter and those not containing artefact were averaged. The peak-to-peak amplitude of the averaged MEP waveforms at each scalp site were assigned to that site as an index of the contribution of the underlying cortex to the control of FCU. The latitude and longitude of stimulus sites over the scalp (in centimetres) was converted to positions on an idealised sphere of half circumference given by the subjects inter-aural distance. The sites were then defined in degrees by their latitude and longitude compensating for differences in head size between subjects. From the MEP amplitude measured at discrete sites over the hemisphere, the expected MEP amplitude for intermediate sites on the hemisphere was estimated. The results are presented in map 23.
form, where a square matrix is used to represent the scalp viewed from above the vertex. The map shows a two dimensional representation of FCU on the motor cortex in contralateral cerebral hemispheres (Fig. 4). The map indicates the optimal stimulus site (in centimetres latitude and longitude) and contours according to the muscle EMG response, decreasing towards the edge of the map until no response is measured. The shaded contours are scaled in the key at the base of the figure, and represent from zero (clear) to one hundred percent (black) of the maximum amplitude that is measured or estimated for that representation. The optimal site of each representation which occurs at the calculated point of maximum amplitude is marked on the map with a white cross. The area of the map is calculated (in centimetres squared) from and above the 50% and 75% contours, and this study has used the 50% area in all subjects. To map the silent period, the individual waveforms were again reviewed and the SP duration in milliseconds (ms) was cursored. The duration was recorded beginning from the onset of the MEP, and ending at the return of the pre-MEP EMG activity (Fig. 5). The mean SP duration from the four waveforms at each site was used for SP map calculation. Data was presented in the same format as the MEP maps.

**Statistical analysis**

Data was statically analyzed using non-parametric tests. Interhemispheric differences in MEP and SP map area, threshold, maximum amplitude for MEP's, optimal stimulus location (latitude and longitude), SP duration, and MVC EMG amplitude, were tested using the Wilcoxin signed rank test. The Mann-Whitney U-test was used for comparisons between control and athlete groups. The level of significance for all tests was set at $P<0.02$. 
Figure 1. The photograph illustrates the cap used to locate stimulus sites over the scalp and the figure eight coil used for stimulation.

Figure 2. The method of facilitation adopted in this study to isolate FCU with wrist flexion and adduction. An isometric contraction using the opposing hand as resistance.
Figure 3. The recorded responses during the threshold procedure with four individual waveforms at each stimulus intensity. The diagram illustrates the increasing MEP amplitude with increased stimulus intensity and an associated increase in SP duration.
Figure 4. This represents the head viewed from above the vertex. The area of representation for FCU is mapped on the contralateral cerebral hemisphere. The optimal stimulus site is indicated by the white cross in each map. The contours represent percentages of the maximum amplitude for the map, decreasing towards the edge of the map as the MEP response decreases.
Figure 5. This illustrates an MEP followed by a silent period in the EMG. The SP duration was recorded beginning from the onset of the MEP, and ending at the return of the pre-EMG activity.
CHAPTER FOUR

Results

All results are tabulated in Appendix B. For the control group, the MEP and SP maps, and threshold curves, are presented in Appendix C for each individual. The same information for the athlete group is presented in Appendix D. All group means are given in the text ± the standard error of the mean (SEM).

*Maximum voluntary EMG*

The mean rms EMG level obtained from FCU muscles during maximal voluntary wrist flexion is displayed in Figure 6. The control group had a greater mean rms EMG in the dominant arm (0.371 mV ± 0.047) compared to the non-dominant arm (0.302 mV ± 0.049). The difference of 0.069 mV is not significant. A difference of similar magnitude is present between the dominant and non-dominant arm in the athlete group, however the non-dominant arm shows the larger EMG (0.363 ± 0.059 mV) compared to the contralateral arm (0.276 ± 0.035 mV). As with the control group, the difference in EMG (0.087 mV) between arms in the athlete group is small. Interhemispheric and intergroup comparisons show a small and statistically non-significant difference in maximum voluntary EMG between the two sides.

*Threshold curves*

Figure 7 shows a typical FCU threshold curve illustrating the line of best fit to the averaged data at each stimulus intensity at 5% increments. The S-shaped curve is
characteristic of most of the threshold graphs in this study. The initial part of the curve represents sub-threshold stimulus intensities. As the stimulus intensity increased to threshold level a measurable response (MEP) was measured with an amplitude approximately equal to the amplitude two standard deviations above the mean EMG. After this point, increases in stimulus intensity cause more dramatic increases in MEP amplitude and hence the steeper gradient of the curve. The rapid rise in amplitude with increased stimulus intensity ceases approximately 20% above the threshold level. At and beyond this point, increases in stimulus intensity produce more polyphasic responses with no further increases peak-to-peak MEP amplitude, resulting in a plateau of the threshold curve. There were no noticeable differences in the slope of the threshold curves between hemispheres or groups of subjects.

The mean threshold level (Fig. 8) was within five percent for each hemisphere in both subject groups, and was not significantly different both between hemispheres of each group, and between groups. For the control group, the mean threshold of the dominant hemisphere was 49.00 ± 3.88%, and that of the non-dominant hemisphere was 50.11 ± 3.96%, a difference of 1.1% which was not significant. The threshold range for the control group was 35-78%, and in the athlete group was 36-77%.

**Maximum MEP amplitude**

The mean maximum peak-to-peak MEP amplitude as calculated from the fit of the MEP maps is represented in Figure 9. The mean amplitude for the control group dominant hemisphere was 2.20 ± 0.56mV, and for the non-dominant hemisphere was 0.92 ± 0.16mV. In the athlete group the dominant side was 1.40 ± 0.14mV compared to the non-dominant side mean 1.23 ± 0.29mV. For both groups, the interhemispheric
difference was small and statistically not significant. The control group on average had a
greater MEP amplitude in the dominant hemisphere than the athlete group (by 0.79mV),
and in the non-dominant hemisphere the athlete group had larger amplitudes but the
difference was smaller (0.31mV) and the intergroup differences were also not statistically
significant.

**SP duration**

The mean maximum duration of the EMG silent period is displayed in Figure 10
and was fairly consistent between hemispheres for both subject groups, being within
12ms of all the muscles studied. The mean maximum SP duration for the control group
dominant side was 164.8± 8.83ms, with the non-dominant side 8.4msec less at 154.9 ±
7.29ms. The athlete group showed an interhemispheric difference of 10.2msec with the
non-dominant hemisphere mean (166.6 ± 15.83ms) greater than the dominant (156.4 ±
9.49ms). There was no statistically significant difference in the inter-group comparisons,
or in the interhemispheric comparisons for each group.

**Typical MEP and SP maps**

The MEP map (Fig. 11) illustrates the representation of both FCU muscles on the
contralateral cerebral hemispheres of one subject from the control group. The map
represents areas where the largest MEP amplitudes were recorded (as indicated by the
darker contours) and the optimal stimulation site (as indicated by the cross in the centre).
The perimeter of the lightest contour of each FCU representation shows that as a whole,
they were similar in size, shape, and location. Small differences in these parameters were
common in interhemispheric comparisons between all subjects in this study, however
they were not significantly different. The location of the optimal stimulus sites are typical of the control group MEP maps with the dominant hemisphere site positioned slightly anterior.

The silent period was mapped from the same waveforms used to map MEP amplitude, and Figure 12 illustrates the SP map of the same control subject which also has data values close to the mean of the control group. The contours represent the SP duration with the darker contours corresponding to an increase in length of SP. Comparisons between the two representations show a size difference which was commonly observed in the other control subjects, although there is no trend to suggest a consistently larger representations in one hemisphere. The optimal location was quite even between hemispheres in both latitude and longitude in Figure 12, however other control subjects showed larger variations. Comparison of the MEP and SP map representation (compare Fig. 11&12) shows that silent period maps are clearly larger than the MEP maps. This was true for both hemispheres and consistent in all control subjects. The location of the representation in each hemisphere was similar between the SP and MEP maps although the shape was slightly different in all contours.

Figure 13 illustrates the MEP maps of one subject selected from the athlete group which demonstrates the typical shape, size and optimal stimulus sites from the group. The area of representation was quite similar for both maps in Figure 13. The difference in shape was small and the variation was consistent with the other athlete MEP maps. The silent period map of the same athlete is presented in Figure 14 and was typical of the athlete group. This map shows a more posterior optimal site on in the dominant hemisphere compared to its contralateral side. The difference in shape of the two representations was not uncommon to the variation shown in other interhemispheric
comparisons in the athlete group. The map shows the dominant hemisphere to have a larger area than the non-dominant side as was the case for most of the athlete group. The area of the SP maps for each hemisphere between the athlete and control was not significantly different. The comparison between the MEP map area and SP map area in each hemisphere of the athlete shows a substantially larger SP map than MEP map on both sides, and this occurred in all subjects in the athlete and control groups.

Area of MEP maps

The mean area of the maps calculated by the MEP amplitude is shown in Figure 15. Interhemispherically there is little difference in both groups. The control group showed a dominant hemisphere mean area of 12.71 ± 0.94cm² and non-dominant area of 12.29 ± 1.17cm². In the athlete group the mean for the dominant side was 11.59 ± 1.96cm², and 10.68 ± 0.74cm² in the non-dominant side. The interhemispheric difference for both groups was not statistically significant. The mean area of the athlete group maps were smaller than the controls for both hemispheres although the difference was also not significant.

Area of SP maps

Both the control and athlete groups showed larger SP maps areas on the dominant hemisphere compared to the non-dominant hemisphere (Fig. 16), the interhemispheric differences of the mean being 1.71cm² and 3.3cm² (P=0.05), for the control and athlete groups respectively. Intra-hemispheric comparison between the MEP
map area and the SP map area was significantly different in the control and the athlete group.

Optimal latitude of MEP and SP maps

The mean latitude of the optimal sites from the MEP maps is illustrated in Figure 17 for both groups. The Y-axis represents the nasion-inion line and the X-axis represents the distance from the vertex. The negative numbers on the X-axis represent centimetres of latitude, on the hemisphere for the centres of areas controlling the dominant FCU muscle. The control group showed an interhemispheric difference in mean latitude of 0.06cm with the non-dominant hemisphere mean (5.16 ± 0.25cm) being more lateral to the dominant (5.1 ± 0.15cm). The interhemispheric difference in the athlete group is 0.23cm with the dominant hemisphere being more lateral (5.24 ± 0.2cm) to the non-dominant (5.01 ± 0.25cm). The graph shows the very little and non-significant difference in optimal latitude, both between hemispheres in each group, and between groups.

Figure 18 shows the mean optimal latitudes of the silent period maps for both groups. The interhemispheric difference in the control group is small (0.04cm) with the dominant side marginally lateral (4.98 ± 0.23cm) to the non-dominant (4.94 ± 0.23cm). In the athlete group the optimal latitude in the non-dominant hemisphere was 4.84 ± 0.19cm from the vertex, and in the dominant hemisphere was 5.58 ± 0.31cm, the latter being more lateral by 0.74cm. The control and athlete group show similar mean latitudes (difference= 0.1cm) in the non-dominant hemisphere. In the dominant hemisphere the group difference was larger (0.6cm) with the athlete group at 5.58cm, and the control group at 4.98cm from the vertex. Comparison between the optimal latitude from the MEP maps and the optimal latitude from the SP maps shows a difference in each
hemisphere for both groups. In the control group the mean latitude from the MEP maps were further from the vertex than the SP maps (dominant side difference = 0.12 cm, non-dominant side difference = 0.22 cm). The athlete group shows the SP mean to be further from the vertex than the MEP mean on the dominant side (by 0.22 cm), and closer to the vertex on the non-dominant side (by 0.17 cm). Intergroup comparisons show little difference in optimal latitude in both MEP and SP maps, with the exception of the dominant hemisphere in the SP maps, where the mean for the athlete group is comparatively quite lateral to the control group.

Optimal longitude of MEP and SP maps

Figure 19 shows the mean optimal longitude of the optimal sites taken from the MEP maps in both groups. The X-axis represents the interaural line and the Y-axis shows the distance from the interaural line. The negative direction on the Y-axis indicates that the mean longitude is posterior to the interaural line. In the control group the longitude mean in the dominant side was 0.59 ± 0.27 cm which was anterior (difference = 0.49 cm) to the non-dominant side at 0.17 ± 0.21 cm, and both were anterior to the interaural line. In the athlete group the non-dominant side mean longitude (0.11 ± 0.3 cm) was anterior to the dominant side (-0.4 ± 0.21 cm). The non-dominant side was anterior to the interaural line, and the dominant side was posterior (difference = 0.51 cm). The graph shows that the mean for both hemispheres in each group is anterior of the interaural line except for the dominant side in the athlete group which is distinctively posterior. In both the dominant and non-dominant hemisphere the mean longitude for the control group was anterior to the athlete group. Although the mean values vary between groups particularly in the dominant side, the range is quite large as indicated by the
standard error bars. Intergroup and interhemispheric differences in longitude were not statistically significant.

Figure 20 shows the mean optimal longitude taken from the silent period maps and the representation is in the same format as figure 19. The interhemispheric difference in the control group is 0.42cm with the dominant side mean (0.21 ± 0.37cm) anterior to the non-dominant side (-0.21 ± 0.4cm). In the athlete group the difference between the two sides was 0.83cm, with the dominant side (0.8 ± 0.21cm) posterior to the non-dominant side (0.03 ± 0.29cm). Comparisons between each group show the dominant hemisphere to have a relatively large difference in mean longitude (1.1cm) with the control group being more anterior. In the non-dominant side, the difference between groups was smaller (0.51cm) and the control mean is posterior to the athlete mean.

Comparisons between the optimal longitude of the MEP maps, and that of the SP maps for each hemisphere show a general shift posterior in both groups in the SP maps. The mean longitude for the control group dominant hemisphere was 0.59 ± 0.27cm for the MEP maps, and 0.21 ± 0.4cm for the SP maps (difference=0.38cm), and in the non-dominant hemisphere was 0.17 ± 0.21cm and -0.21 ± 0.37cm respectively (difference=0.38). In the athlete group the longitude in the dominant side was -0.4 ± 0.21cm for the MEP maps, and -0.8 ± 0.21cm for the SP maps (difference=0.4). In the non-dominant hemisphere for this group the mean MEP longitude was 0.11 ± 0.3cm and the SP longitude was 0.03 ± 0.29cm (difference=0.08cm). The two graphs highlight the posterior placement of the optimal longitude in the dominant hemisphere of the athlete group, and the posterior shift in map centres in the SP maps, however the differences are not statistically significant.
Optimal sites for MEP and SP maps

Figure 21 shows the individual optimal latitude and longitude sites taken from the MEP maps plotted in relation to one another on a two dimensional graph. The origin of the graph axes represent the vertex with the X-axis (optimal latitude) being the interaural line and the Y-axis (optimal longitude) being the nasion-inion line. From the graph it is evident that both groups exhibit similar optimal latitudes particularly in the dominant hemisphere. The range of latitudes do not show any trends that may suggest hemispheric differences between each group. A larger variation in optimum longitude of the MEP maps is clearly evident in each hemisphere for both groups. In the dominant hemisphere, the athlete group longitudes show a posterior distribution (six out of eight subjects with map centres behind the interaural line). In contrast, the dominant hemisphere of the control group had a more anterior distribution of map position, with seven out of nine subjects having MEP maps centred in front of the interaural line.

Figure 22 displays the optimal sites from the SP maps on each hemisphere of both groups. The control group showed a similar range in latitudes (approximately 2cm) to that seen in the MEP maps in both hemispheres. In comparison, the athletes tended to have a larger range of latitude (approximately 3cm). Like the MEP latitudes, the longitudes of the SP map centres tended to show a greater dispersion in the dominant hemisphere of both groups studied, with the control group having 4cm dispersion of mean results. The latter finding was largely due to a single subject having a SP map centred 2cm behind the interaural line.
Figure 6. The mean voluntary EMG level obtained from FCU muscles during maximal voluntary wrist flexion.

MAXIMUM VOLUNTARY EMG (mean +/- sem)

DOMINANT HEMISPHERE

NON-DOMINANT HEMISPHERE

control

athlete
Figure 7. A typical FCU threshold curve illustrating the line of best fit to the averaged data at each stimulus intensity.

Figure 8. The mean threshold level recorded for each group.

CALCULATED THRESHOLD (mean +/- sem)

DOMINANT HEMISPHERE     NON-DOMINANT HEMISPHERE
Figure 9. The mean maximum peak-to-peak MEP amplitude as calculated from the MEP maps.

MAXIMUM MEP AMPLITUDE (mean +/- sem)

![Graph showing MEP amplitude comparison between control and athlete groups.]

Figure 10. The mean maximum duration of the EMG silent period.

SILENT PERIOD DURATION (mean +/- sem)

![Graph showing silent period duration comparison between control and athlete groups.]
Figure 11. Typical MEP mapped representations of FCU of the control group.

![Figure 11. Typical MEP mapped representations of FCU of the control group.](image)

Figure 12. Typical SP mapped representations of FCU in the control group.

![Figure 12. Typical SP mapped representations of FCU in the control group.](image)
Figure 13. Typical MEP mapped representations of FCU in the athlete group.

Figure 14. Typical SP mapped representations of FCU in the athlete group.
Figure 15. The area of the representation from the MEP maps for athlete and control groups.

AREA OF MEP MAPS (mean +/- sem)

<table>
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Figure 16. The area of the representation from the SP maps for athlete and control groups.

AREA OF SP MAPS (mean +/- sem)

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<td>athlete</td>
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Figure 17. The optimal latitude of the centre of the MEP maps in the athlete and control groups.

OPTIMAL LATITUDE OF MEP MAPS (mean +/- sem)

DOMINANT HEMISPHERE   NON-DOMINANT HEMISPHERE

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<th>athlete</th>
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Figure 18. The optimal latitude of the centre of the SP maps in the athlete and control groups.

OPTIMAL LATITUDE OF SP MAPS (mean +/- sem)

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Figure 19. The optimal longitude of the centre of the MEP maps in the athlete and control groups.

OPTIMAL LONGITUDE OF MEP MAPS (mean +/- sem)

Figure 20. The optimal longitude of the centre of the SP maps in the athlete and control groups.

OPTIMAL LONGITUDE OF SP MAPS (mean +/- sem)
Figure 21. The diagram represents a view of the scalp looking down, with the X-axis as the nasion-inion line, and the Y-axis as the interaural line. The graph displays the individual centres for both groups of subjects as taken from the MEP maps.

![Optimal sites for MEP maps]

Figure 22. This graph displays the individual centres for both groups of subjects as taken from the SP maps.

![Optimal sites for SP maps]
CHAPTER FIVE

Discussion

This study has compared the cortical motor representations of a wrist flexor muscle FCU in trained elite badminton players, to those of untrained muscle, in order to identify any differences that may occur in the representation on the motor cortex in relation to muscle use. We hypothesised that there would be differences in the size of the representation and excitability of the motor cortex responsible for controlling the FCU. Interhemispheric comparisons in the athletes showed no significant differences in the size of the representation for MEPs or SPs, and no difference in the excitability between the dominant and non-dominant sides.

By selecting subjects for the study that were skilled unilaterally it was possible to examine the FCU contralateral to the dominant, as the untrained sample. A group of non-trained subjects was used to control for any differences in the representations that may be due to factors other than training, such as handedness. There were two main reasons for examining the FCU in this study. Firstly, the muscle is used regularly in badminton and is activated in coordination with other forearm flexors and extensors to produce fine control of the racquet, and is therefore highly skilled in these movements (Lo & Stark, 1991). Secondly, it can be activated in relative isolation and evoked responses to TMS can be recorded using surface electrodes. Since no previous mapping studies of the FCU have been reported, it has been necessary to characterise the cortical representation of the muscle relative to that of more proximal and distal arm muscles.
In this study we found that using TMS to stimulate the cortex and recording the size of the evoked response, it was possible to map the representation of FCU on the cerebral cortex. By repeating the mapping procedure in three of the subjects, we found the maps to be reproducible. The area of the representations for FCU in the control group (dominant= 12.71 ± 0.94cm², non-dominant= 12.29 ± 1.17cm²) were similar to those found in the hand muscle abductor pollicus brevis (APB) (dominant=14.1 ± 4.2cm², non-dominant=13.0 ± 4.8cm²), but smaller than abductor digiti minimi (ADM) (dominant=16.2 ± 5.6cm², non-dominant=13.0 ± 3.9cm²) when compared to a different subject group (Wilson et al., 1993a). The mean latitudes for the control group centre of MEP maps in this study (dominant=5.1 ± 0.15cm, non-dominant=5.16 ± 0.25cm), were slightly medial to those for APB (dominant=5.98 ± 0.04cm, non-dominant=5.81 ± 0.08cm) and ADM (dominant=5.79 ± 0.04cm, non-dominant=5.28 ± 0.06cm). Wassermann et al. (1992) mapped the representations of four upper extremity muscles (APB, flexor carpi radialis, biceps brachii and deltoid) in normal subjects. The results from this study showed distal muscles to have larger representations as would be expected from the motor homunculus where the fingers and hand are shown to have a larger representation on the motor cortex than more proximal muscles.

Interhemispheric comparisons in representational area in the control group of this study were not significantly different between the right and left hand, and this is in agreement with the findings by Wilson et al. (1993a) where no differences in area of the representations were reported between the dominant and non-dominant hemisphere. Wasserman et al. (1992) found that in the biceps and deltoid only, area was significantly greater on the non-dominant hemisphere, whereas with APB the dominant hemisphere showed the larger representations. In this study, comparison of the dominant and non-
dominant hemispheres in the athlete group showed no significant difference in the MEP map area. This indicates that skilful use of the FCU in badminton on a regular basis, is not sufficient to cause prolonged changes to the size of the MEP maps when compared to normal use for the non-dominant hand. Interhemispheric differences in the athletes are consistent with those of the control group and it can be concluded that this type of training does not cause significant long term changes in the size of the representation for FCU.

It appears that although there may be biological asymmetries in the structure and function of the brain relating to cerebral dominance and handedness (Galaburda et al., 1978), this is not reflected at the level of the primary motor cortex, specifically in the representation of single muscles. This study found no trends in dominance or handedness with respect to map representations.

This study also mapped the representation of the silent period following the MEP measured in FCU. In the control group, the size and topography of the SP map varied between hemispheres and between subjects however no trends were evident and there were no statistically significant differences. There was no difference in size of the SP representation between the dominant and non-dominant FCU. The silent period representation was larger than the MEP representation within each hemisphere for all subjects. This is consistent with findings by Wilson et al. (1993b) where the SP representation was found to be significantly larger, and encompass the MEP representation for APB.

The silent period representations in the athlete group were similar to the control group in that they were all larger than the MEP maps, however, the athlete group showed larger silent period maps on the dominant hemisphere and this was not shown in
the control group. Differences in the interhemispheric SP areas have not been described in the literature possibly due to the fact that there have been no noticeable differences in the normal subjects that have been investigated. This suggests that there is probably minimal difference in the area of the silent period representation in normal subjects due to handedness. The larger area of silent period maps on the dominant hemisphere in the athletes was only a trend and possibly more athletes would need to be tested to validate this claim as statistically significant. The silent period after cortical stimulation is generated by several mechanisms (Inghilleri, Berardelli, Crucu, Manfredi, 1993). In the first 50ms, spinal factors operate, with a possible contribution by descending inhibitory fibres. The later part of the silent period probably results from inhibitory effects at the cortical level. Wilson et al. (1993a) suggest that inhibitory processes may act to limit or contain the excitatory output, and that this may contribute to fine discriminatory motor control. Wilson et al. (1993b) found that the centre of the MEP maps and the SP maps were very close. In this study the range of centres between MEP and SP maps were fairly consistent in latitude for the control group and the non-dominant hemisphere of the athlete group, however the dominant hemisphere of the athlete group showed a more lateral SP centre. The longitude of all SP maps were posterior to the MEP map centres. The posterior placement of the athlete dominant hemisphere MEP map centre relative to the other centres, was also reflected in the SP map. A possible explanation is that this may represent the convergence of a greater degree of sensorimotor integration in the dominant hemisphere of the athletes. However other studies examining the changes in cortical representation between skilled and normal muscles have claimed representational area differences, but have not reported changes in location (Pascual-Leone et al., 1993a; Pascual-Leone et al., 1993b).
For both groups in this study threshold differences between hemispheres and between subjects, were negligible and not statistically significant. These findings are in agreement with Macdonell et al. (1991) who found no significant difference in thresholds between the two sides, and contrary to Cohen et al. 1991 who indicate excitability differences with higher amplitude MEPs and lower thresholds in the dominant hemisphere of normal subjects.

The maximal voluntary EMG signals were not significantly different between dominant and non-dominant sides, or between subjects. This gives some indication that the skilled and unskilled muscles have similar characteristics. Strength differences are not always consistent with skill differences and handedness (Porac and Coren, 1981), and although we did not measure wrist flexor strength, no obvious differences were observed in the forearm sizes of the dominant and non-dominant arms in the athletes.

From the results of this study it can be concluded that there are no long term changes to the size of the cortical representation projecting to the dominant FCU in badminton players. These findings are contrary to those found in the case of the Braille subjects that have been exposed to regular skilled training of the hand muscles (Pascual-Leone et al., 1993). These studies found differences in the size of the representations in somatosensory cortex to the reading hand of Braille readers. The cause of these changes is unknown. It may be due to fine control of skilled movement, or it may be due purely to the regular activation of the muscle (Pascual-Leone et al., 1993). The learning of simple motor tasks with regular practise over a short period of time has shown that modulation of the area of the representation can occur while learning the skill but the changes are only short term (Pascual-Leone et al., 1994). This may be the case when learning a new sports skill. Cortical modulation may have occurred in the badminton
players during the learning phase of the skill and then once the skill was learned and automated, the representation may have resumed its normal size. It may be necessary to examine the athletes before, during, and after learning a new skill to determine if modulation occurs.

This study has found that the cortical representations of muscles in unilaterally skilled sportspersons are generally symmetrical, with subtle interhemispheric differences in the centre of the representations and silent period area. The results suggest that performance differences related to general handedness, and between highly skilled and contralateral non-skilled hands during motor tasks, is not reflected in the size of the muscular representation in the motor cortex or the excitability.

Pons et al. (1991) suggests that as a skill becomes automatic (as is the case with the badminton players), there may be structural changes in the intracortical networks and this could be the reason why a larger representation in the dominant hemisphere is not present. It may be possible that the dominant side representational area has experienced a change in the 'wiring' pattern of the corticомotor cells as a result of training. Divergence of neuronal connections to synergist muscles to that of FCU, may assist with fine motor control and explain why an increase in skill is present without an increase in representational area.

In summary, it is evident that badminton players show superior skill in the racquet arm during performance, when compared to the non-dominant arm. This difference is not reflected in the representation of forearm muscles on the motor cortex between cerebral hemispheres. It may be that this method of examining the motor cortex does not detect the changes that that occur in the cortex with skill development, or the changes may occur at higher levels of motor control. Programmed movements may be
stored in subcortical areas such as the cerebellum and basal ganglia which were not investigated in this study. It may be necessary to investigate other motor areas, or higher levels of motor control, to detect significant differences between trained and untrained muscles in the central nervous system.
References


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Merzenich, M.M., Kaas, J.H., Wall J.T., Sur M., Nelson, R.J., Felleman, D.J. (1983) Progression of change following median nerve section in the cortical representation of the hand in areas 3b and 1 in adult owl and squirrel monkeys. *Neuroscience* (10) 639-665


Ransom, S., Clark, S. (1959) The anatomy of the nervous system. Its development and function pp.357-382 W.B. Sanders, Philadelphia


Appendix A

Australian Neuromuscular Research Institute: Consent Form

Mapping of wrist flexor muscles

Procedure: The procedure is non-invasive. Electrode discs will be taped onto the biceps and wrist flexor muscles. The activity in the muscles will be recorded via these electrodes and the information will be fed into the computer. Magnetic stimulation will be used. A snugly fitting cap with pre-marked spacings will be placed on the head. The magnetic coil will be positioned on various sites of the cap and that part of the brain will be stimulated. Each stimulation will be very short, much less than 1 second. This is not painful, but some small movements may be noticed in the target muscle. Occasionally, tingling or a tap on the scalp may be felt. During the session you will be asked to contract muscles in the arm maximally for 5 seconds and submaximally also during the stimulation. You will be shown how to perform these contractions and will be given a chance to practice. We will start the session with a few practice runs, and there will be a rest period after each set of trials. There are very few possible discomforts associated with these procedures. On rare occasions magnetic stimulation may cause a headache. If this occurs, or for any other reason you wish to stop the session, we will stop the session. I understand that I am free to withdraw from the study at any time.

I acknowledge that I have read the above statement which explains the nature and object and the possible risks of the investigation and the statement has been explained to me to my satisfaction. Before signing this document I have been given the opportunity to ask any questions relating to any possible physical or mental harm I might suffer as a result of my participation and I have received satisfactory answers. I agree that research data gathered from the result of the study may be published provided my name is not used.

In the light of the forgoing, I hereby release the Australian Neuromuscular Research Institute or any employee, member or representative thereof, from all or any claim that I may have arising out of my participation on this experiment. I understand that this document in no way limits my rights at law from any damage that might arise from negligence on the part of the investigators.

To the best of my knowledge I am not pregnant. I do not have a cardiac pacemaker and I do not have metal implants in my head.

................................., age........ years, agree to participate as a subject in a study of the type described above.

Signed............................... Witness..............................
Date..............................
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APPENDIX C

CONTROL GROUP MEP MAPS

* Indicates dominant hemisphere
CONTROL GROUP SP MAPS

* Indicates dominant hemisphere

FILE: /home/gary/pc/data/mapping/dylan/dert.dly

SERIES: CONTROL

COMMENTS: map of left flexors
URN: unspecified  AGE: 22 years  SEX: male
FILE: /home/gary/pc/data/mapping/dylan/urn.ffd
SERIES: Control - Weightlifting  CONTROL 7
COMMENTS: L FCU - Non-Dominant

---

URN: unspecified  AGE: 22 years  SEX: male
FILE: /home/gary/pc/data/mapping/dylan/urn.txx
SERIES: Control - Weightlifting  CONTROL 7
COMMENTS: R FCU - Dominant

---
CONTROL GROUP THRESHOLD CURVES

* Indicates dominant hemisphere
SERIES: Control 1
COMMENTS: map of right wrist flexor

SERIES: Control 1
COMMENTS: map of left flexors
Background is 0.00 = 0.06 mV, limit set to 0.36 mV.

Parameters of the fit to \( \frac{a}{1-e^{-(b-x)/(c-b)}} \):
- Amplitude \((a)\): 5.0
- Maximum slope \((b)\): 0.1
- Y-offset \((c)\): 75.0

Plot number: one from column 1

...
Background is 0.11 ± 0.06 mV, limit set to 0.26 mV.

<table>
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<tr>
<th>Parameter of the fit to a(1-exp(b(x-c)/d))+(1-exp(-b(x-c))/d)</th>
<th>Plot data</th>
<th>See guess</th>
<th>Fit</th>
<th>Quiet</th>
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<tr>
<td>Amplitude (a): 5.0</td>
<td>Get bg from epoch: 1-5</td>
<td>dur average</td>
<td>Calculate bg from: middle of epoch</td>
<td>Set bg limit: 1.3 ± 2.9 mV</td>
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<td>Maximum slope (b): 0.8</td>
<td>Starting from epoch 1 in groups of 4</td>
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<td></td>
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<tr>
<td>Y-offset (c): 75.0</td>
<td>Starting from epoch 1 in groups of 4</td>
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</tr>
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<td>Y-offset (d): 5.0</td>
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Plot number: use from column 1

Parameter of the fit to a(1-exp(b(x-c)/d))+(1-exp(-b(x-c))/d):
Amplitude (a): 5.0
Maximum slope (b): 0.8
Y-offset (c): 75.0
Y-offset (d): 5.0

Plot number: use from column 1

**TMS INTENSITY**

**TMS INTENSITY**
Background is 0.00 - 0.10 mV, limits set to 0.00 mV.

Parameters of the fit to $a(1-exp(-b(x-c)))/(1+exp(-b(x-c)))+d$ form

- Amplitude (a): 5.0
- Maximum slope (b): 0.1
- Y-offset (c): 75.0
- Y-offset (d): 5.0

TMS INTENSITY

Plot number: one from column 1.

Background is 0.20 - 0.26 mV, limits set to 0.20 mV.

Parameters of the fit to $a(1-exp(-b(x-c)))/(1+exp(-b(x-c)))+d$ form

- Amplitude (a): 5.0
- Maximum slope (b): 0.1
- Y-offset (c): 75.0
- Y-offset (d): 5.0

TMS INTENSITY

Plot number: one from column 1.
Parameters of the fit is all-exp(-b(x-c)))/1+exp(-b(x-c)))=d

- Amplitude (a): 5.0
- Horizontal slope (b): 0.1
- y-intercept (c): 70.1
- y-intercept (d): 2.0

Background is 0.14 = 0.05 W, Limit set to 0.20 W.

Plot data: Set bag from epoch: 1-4, Ane 2nd calc.
Calculate bag from: middle of epoch.
Set bag limits... 2 nd.
Starting epoch... in steps of 2.
Starting from epoch: 1 in groups of 4.
Plot number: one from column 1.

HAS INTENSITY

Parameters of the fit is all-exp(-b(x-c))/1+exp(-b(x-c)))=d

- Amplitude (a): 3.6
- Horizontal slope (b): 0.1
- y-intercept (c): 73.0
- y-intercept (d): 5.0

Background is 0.09 = 0.03 W, Limit set to 0.14 W.

Plot data: Set bag from epoch: 1-4, Ane 2nd calc.
Calculate bag from: middle of epoch.
Set bag limits... 2 nd.
Starting epoch... in steps of 2.
Starting from epoch: 1 in groups of 4.
Plot number: one from column 1.

HAS INTENSITY
URN: unspecified  AGE: 22 years  SEX: male
FILE: /home/gary/pc/data/mapping/dylan/rsn:flx
SERIES: Control - Weightlifting  CONTROL 7
COMMENTS: R FCU - Dominant

Background is 0.22 ± 0.03 mV, limit set to 0.22 mV.

Parameters of the fit to $a \left(1 - \exp(-b(x-c))\right)/\left(1 + \exp(-b(x-c))\right)$:
- Amplitude ($a$): 5.0
- Maximum slope ($b$): 0.1
- Offset (c): 75.0
- Offset (d): 5.0

Starting from epoch: End of epoch
Plot number: one from column 1

URN: unspecified  AGE: 22 years  SEX: male
FILE: /home/gary/pc/data/mapping/dylan/rsn:flx
SERIES: Control  CONTROL 7
COMMENTS: L FCU - Non-Dominant

Background is 0.06 ± 0.05 mV, limit set to 0.07 mV.

Parameters of the fit to $a \left(1 - \exp(-b(x-c))\right)/\left(1 + \exp(-b(x-c))\right)$:
- Amplitude ($a$): 5.0
- Maximum slope ($b$): 0.1
- Offset (c): 75.0
- Offset (d): 5.0

Starting from epoch: 1 in groups of 4
Plot number: one from column 1
Background is 0.07 - 0.02 mV, limit set to 0.12 mV.

Parameters of the fit to \( \frac{\text{Amplitude}(x)}{1+\text{Exp}\left(-\text{Exp}\left(-\text{Gain}(x)(1-\text{Exp}(1-x))\right)\right)} \):

- Amplitude (a): 5.0
- Maximum slope (b): 0.1
- X-offset (c): 75.1
- Y-offset (d): 0.0

Plot: DATA

Series: Control

Comments: Right hand non-dominant arm.
Background is 0.18 - 0.05 mV, limit set to 0.18 mV.

Parameters of the fit to all1-exp(-b(x-c)/(1-exp(-b(x-c)))):
Amplitude (a) 5.0
Maximum slope (b) 0.1
X-offset (c) 75.0
Y-offset (d) 5.0
Plot number: one from column 1.

Parameters of the fit to all1-exp(-b(x-c)/(1-exp(-b(x-c)))):
Amplitude (a) 5.0
Maximum slope (b) 0.1
X-offset (c) 75.0
Y-offset (d) 5.0
Plot number: one from column 1.
APPENDIX D

ATHLETE GROUP MEP MAPS

* Indicates dominant hemisphere
ATHLETE GROUP SP MAPS

* Indicates dominant hemisphere
URN: unspecified  AGE: 24 years  SEX: male


FILE: /home/gary/po/data/mapping/dylan/sfits

SERIES: BADMINTON  ATHLETE 6

COMMENTS: RIGHT HAND DOMINANT

---

URN: unspecified  AGE: 24 years  SEX: male


FILE: /home/gary/po/data/mapping/dylan/sfits

SERIES: BADMINTON  ATHLETE 6

COMMENTS: LEFT HAND NON DOMINANT
ATHLETE GROUP THRESHOLD CURVES

* Indicates dominant hemisphere
### Background

Background is 0.07 to 0.08 mV, limit set to 0.14 mV.

#### Parameters of the fit to $a(t) = \exp(-b(t-c)/(1+\exp(-d(t-c))))$:

- **Amplitude** $(a)$: 5.0
- **X-offset** $(c)$: 55.0
- **F-offset** $(d)$: 5.0

### Plot Data

- **Plot style**: various
- **Slice date**: unspecified
- **Fit details**: unspecified

### INTENSITY

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<tr>
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<td>3.0mV</td>
</tr>
<tr>
<td>60X</td>
<td>4.0mV</td>
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### Series

#### Series: ATHLETE 1

**Comments**: right eye

---

### Background

Background is 0.05 to 0.06 mV, limit set to 0.10 mV.

#### Parameters of the fit to $a(t) = \exp(-b(t-c)/(1+\exp(-d(t-c))))$:

- **Amplitude** $(a)$: 5.0
- **X-offset** $(c)$: 55.0
- **F-offset** $(d)$: 5.0

### Plot Data

- **Plot style**: various
- **Slice date**: unspecified
- **Fit details**: unspecified

### INTENSITY

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<th>MEP</th>
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<tr>
<td>60X</td>
<td>0.9mV</td>
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### Series

#### Series: ATHLETE 1

**Comments**: left eye
URN: unspecified
AGE: 33 years
SEX: female
FILE: /home/gary/pc/data/mapping/dylan/w.txt
SERIES: ATHLETE Z
COMMENTS: Right FCU - Backmion

Background is 0.068 - 0.02 mV, limit set to 2.19 mV.

Parameters of the fit to \( a(1-\exp(-b(x-c)))/(1+\exp(-b(x-c))) \):
- **Amplitude** \((a)\): 2.0
- Maximum slope \((b)\): 0.1
- \(x\)-intercept \((c)\): 75.0
- \(y\)-intercept \((d)\): 5.3

Plot data: one from column 1

---

URN: unspecified
AGE: unspecified
SEX: unspecified
FILE: /home/gary/pc/data/mapping/dylan/w.txt
SERIES: ATHLETE Z
COMMENTS: Left wrist flexor map

Background is 0.068 - 0.02 mV, limit set to 0.15 mV.

Parameters of the fit to \( a(1-\exp(-b(x-c)))/(1+\exp(-b(x-c))) \):
- **Amplitude** \((a)\): 5.0
- Maximum slope \((b)\): 0.1
- \(x\)-intercept \((c)\): 75.0
- \(y\)-intercept \((d)\): 5.0

Plot data: one from column 1
URN: unspecified  AGE: unspecified  SEX: female
FILE: /home/gary/pc/data/mapping/dylan/fdrt.flx
SERIES: Badminton  ATHLETE 4
COMMENTS: R FCU-Dominant

SERIES: Badminton  ATHLETE 4
COMMENTS: L FCU-NonDominant
URN: unspecified  
AGE: 24 years  
SEX: male  

FILE: /home/gary/pc/data/mapping/dylan/dfn.flx  
SERIES: BADMINTON  
COMMENTS: RIGHT HAND DOMINANT  

---  

URN: unspecified  
AGE: 24 years  
SEX: male  

FILE: /home/gary/pc/data/mapping/dylan/dfn.flx  
SERIES: BADMINTON  
COMMENTS: LEFT HAND NON DOMINANT  

---  

[Graphs showing data related to muscle activity]
Background to 0.07 - 0.11 mV, limit set to 0.12 mV.

Parameters of the fit to \( a + \frac{b}{\text{exp}(-c(x-d))}/(1 + \text{exp}(-c(x-d))) \) are:

- **Amplitude (a):** 5.0
- **Max. slope (b):** 4.1
- **X-off (c):** 75.0
- **Y-off (d):** 3.0

Calculate slope from middle of epoch.

Starting from epoch: 1 in steps of 10.

Plot number: one from column 1.

---

Background to 0.06 - 0.09 mV, limit set to 0.12 mV.

Parameters of the fit to \( a + \frac{b}{\text{exp}(-c(x-d))}/(1 + \text{exp}(-c(x-d))) \) are:

- **Amplitude (a):** 0.9
- **Max. slope (b):** 8.1
- **X-off (c):** 75.0
- **Y-off (d):** 3.0

Calculate slope from middle of epoch.

Set by Intv.: 2 ms

Starting from epoch: 1 in steps of 10.

Plot number: one from column 1.
Background is 0.07 ± 0.02 mV, limit set to 0.12 mV.

| Parameter of the fit to \(|(1-\exp(-b(x-c)))/(1-\exp(-b(x-c)))\) | Amplitude (a) | 5.0 |
| Maximum slope (b) | 0.1 |
| X-coeff (c) | 75.0 |
| Y-coff (d) | 5.0 |

Calculate y-coff: middle of epoch.
Set by y-coff: ....... 2 ed.
Starting TMS:...... 33% in steps of 10
Starting from epoch: 1 in groups of 4
Plot number: one frame column 1
URN: unspecified  AGE: 30 years  SEX: male
FILE: /home/gary/po/data/mapping/dylan/jgpl.flx
SERIES: BADMINTON ATHLETE 8
COMMENTS: RIGHT HAND DOMINANT

URN: unspecified  AGE: 30 years  SEX: male
FILE: /home/gary/po/data/mapping/dylan/jgpl.flx
SERIES: BADMINTON ATHLETE 8
COMMENTS: LEFT HAND NON-DOMINANT