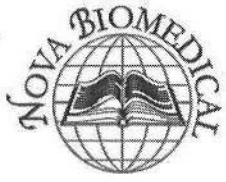
HORIZONS IN NEUROSCIENCE RESEARCH

HORIZONS IN NEUROSCIENCE RESEARCH. VOLUME 3

ANDRES COSTA
AND
EUGENIO VILLALBA
EDITORS



Nova Science Publishers, Inc.
New York

Library of Congress Cataloging-in-Publication Data

ISSN: 2159-113X
ISBN: 978-1-61728-027-6

Published by Nova Science Publishers, Inc. † New York

In: Horizons in Neuroscience Research. Volume 3
Editor: Andres Costa and Eugenio Villalba
ISBN: 978-1-61728-027-6

Chapter 2

Neuroplasticity Following Skill and Strength Training: Evidence from Transcranial Magnetic Stimulation Studies

Alan J Pearce[1] and Dawson J Kidgell[2]
[1] School of Sport and Exercise Science, Victoria University, Melbourne Australia;
[2] School of Exercise and Nutrition Science, Deakin University, Melbourne Australia.

Abstract

The ability for the human motor cortex to change based on experience is now well accepted. Termed neuroplasticity, convincing evidence is available to challenge the long held view of a functionally stable neocortex that is unable to change. The aim of this chapter is to provide data collected in our laboratory, using the technique of transcranial magnetic stimulation (TMS), as evidence of neuroplasticity following short and long term skill and strength training. Short-term skill training studies have shown significant increases in corticospinal excitability (12%) projecting to an intrinsic hand muscle (first dorsal interosseous, FDI) during the performance of a simple fine motor task associated with greater degree of precision compared to an identical but less difficult task. These findings were then reproduced in both anisometric (ramped) and dynamic visuomotor tracking tasks (in FDI), with corticospinal excitability increased during both slow and precise anisometric and dynamic tasks (increases of 42% and 56% respectively), compared to the same tasks being performed fast and rhythmically. Neuroplasticity has also been demonstrated in individuals who have practiced complex motor skills over long-periods of time. Using the technique of TMS mapping, allowing for the construction of topographical maps, we investigated the representation of the FDI, in Olympic badminton athletes. In comparison to sub-elite competitive players and control subjects, Olympic athletes showed increased corticospinal excitability and alterations in

the map representation suggesting functional reorganisation following long-term motor practice. Strength exercise has been considered a form of motor skill training and for this reason we have also investigated neural adaptations following short-term strength training. Four weeks of maximal isometric strength training in the FDI demonstrated a significant increase in strength (34%) and changes in corticospinal inhibition (seen as a reduction in the cortical silent period of 25 ms). Similar increases in strength (28%) were observed following four weeks unilateral training of the biceps brachii muscle, however, neural adaptations were reflected in greater corticospinal excitability rather than corticospinal inhibition. In a second part of the same study, we also found significant increases in strength (19%) and corticospinal excitability projecting to the biceps brachii muscle in the non-trained limb, in the absence of muscular hypertrophy in either arm. The studies presented in this chapter has extended on previous research, however by using TMS we are able to provide evidence of neuroplastic changes in the human motor cortex, in relation to motor skill and strength training.

Introduction

The ability for the human primary motor cortex (M1) to adapt and reorganise, based on experience and/or training, is now accepted and ubiquitously termed neuroplasticity. With evidence collected from a range of non-invasive neuroimaging techniques, to challenge the long held view of a functionally stable neocortex, immutable to change, investigations now focus on the degree and variations in the forms of neuroplasticity following experience or training interventions [1].

Neuroplasticity in humans has been demonstrated across a range of conditions, including acute sensory and peripheral manipulations, such as electrical stimulation [2], amputation [3], nerve deafferentation or anaesthesia [4]. Reorganisation in topographical representation, providing evidence of experience dependant neuroplasticity, have been shown in neurological conditions such as stroke [5], dystonia [6], Parkinson's disease [7] and multiple sclerosis [8].

Neural adaptations have also been demonstrated following training interventions such as motor skill acquisition and strength training. We have used the technique of transcranial magnetic stimulation (TMS) to investigate neural adaptations and functional reorganisation following fine motor skill performance, long-term motor skill training, short-term strength training, and cross-education to the untrained limb following unilateral strength training. This has been the focus of the research in our laboratory and will form the basis of this chapter. The chapter will briefly describe the technique of TMS before the presentation of these studies.

Transcranial Magnetic Stimulation (TMS)

It is possible to study the brain using electromagnetic stimulation. Non invasive brain stimulation, particularly TMS, was first developed in 1985. Commercially available stimulators suitable for human study have been available since the middle 1980s [9] with minimal risk to healthy individuals [10]. Although quite rare, individuals may experience

mild local discomfort on the scalp musculature or a headache. However, since the introduction of commercially available single pulse stimulators, only three reported cases of seizure have been reported in adult patients with large cerebral infarcts or other lesions, with no adverse reports in healthy adults [10] or children [11,12], making single pulse TMS very safe [13,14].

The mechanisms and principles of TMS are well covered in reviews by Hallet [13, 14]. However, briefly, TMS employs time varying magnetic fields that induce electrical currents in conductive neural tissue, with the induced electric field being proportional to the rate of the magnetic field. If the current induced by the electric field is of sufficient amplitude and duration, it will depolarise neural tissue, recorded and measured as a motor evoked potential (MEP) in the surface electromyogram (sEMG) of the target muscle. There are several quantifiable components with TMS. The motor threshold (MT) being the minimum amount of stimulation required to produce a MEP. The MT may be observed in both resting muscle or during a tonic voluntary contraction, and may be termed active motor threshold or AMT [15]. The measurable components of a MEP are illustrated in Figure 2: latency, MEP amplitude and silent period duration. The latency of the MEP is a reproducible measure of the corticospinal conduction time and measured from the time of stimulation to the onset of the MEP [13,14]. For example, it is well known that conduction time from stimulation to a hand muscle is approximately 20 ms of which Hess et al. [16] estimated that approximately 13 ms is from peripheral mechanisms with the remaining time comprising central conduction, synaptic delay at the motoneuron and conduction down a short intradural segment of the motor root [17]. Further, it is also well known that tonic voluntary activation can reduce the corticomotor conduction time of an average of 2 – 3 ms [18] due to facilitation of spinal motoneurons.

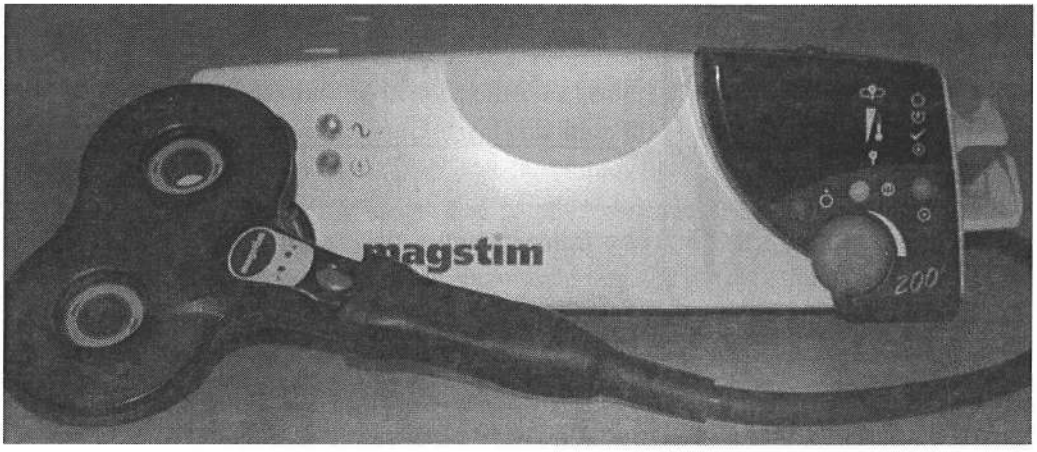


Figure 1. Magstim 200^2 single pulse stimulator with a 'figure of eight' magnetic coil. The figure of eight coil contains two small magnets in reverse polarity enabling a more focused electromagnetic impulse than the standard circular coil (from author's own image collection).

The MEP is a relatively synchronous muscle response that can be measured on the sEMG [13, 14]. The amplitude of a MEP is influenced by inhibitory and excitatory interconnections

descending on the motor neurone pool at the time of stimulation [19] and is usually quantified by measuring the peak to peak amplitude of the MEP "spike" waveform on the sEMG (see Figure 2). Similar to latency, facilitation of a muscle will reduce the threshold for a MEP to occur, lowering the intensity of stimulation applied to the participant. Individuals respond differently to TMS, that is, the MEP response for similar stimulus intensity between individuals will differ in amplitude [20]. However, when controlled for torque and type of motor task, the MEP is a reliable intra-partipant measure [21, 22], allowing for confident interpretation of changes following acute or chronic interventions. However, it is not uncommon to see investigators using a MEP/M-wave ratio to normalise individual MEP variations between participants [23].

In facilitated muscle contractions, the MEP will be followed by a distinctive period of EMG non activity, termed the silent period or SP (Figure 2). The SP can last anywhere between 50 ms to 300 ms following stimulation, and provides a measure of the strength of inhibition in the corticospinal pathway [25, 26]. It is now recognised that the SP arises from a combination of peripheral (spinal cord) and cortical inhibition [14, 27] and with single pulse TMS the duration of the silent period is thought to reflect γ-aminobutyric acid ($GABA_b$) mediated inhibitory processes [28].

The SP has been shown to correlate with MEP [29], despite the evidence that the SP represents different aspect of cortical excitability [26, 30]. It is well recognised that the SP will be recruited at lower levels of stimulation, including below AMT [31], and saturate earlier than the MEP [29].

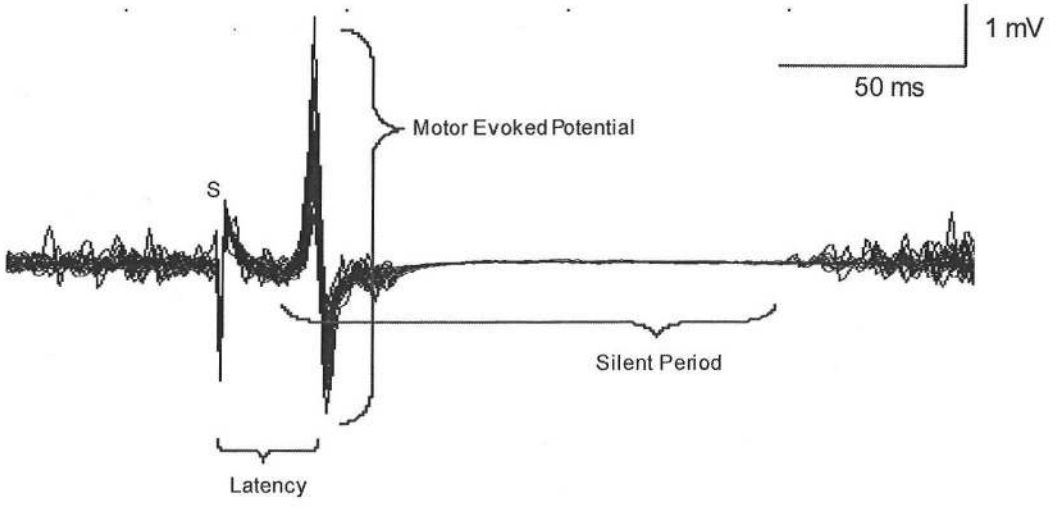


Figure 2. The components of a MEP as recorded on sEMG. Overlayed image of 30 sweeps, obtained from the first dorsal interosseous (FDI) muscle following TMS over the contralateral motor cortex, illustrating the reliability of the MEP when the muscle is controlled for contraction level. The MEP is visible approximately 20-25ms following cortical stimulation (S). This is termed latency (L). MEP amplitude is calculated by cursoring the peak to peak value of the MEP. Silent period (SO) is measured by cursoring onset of MEP to return of sEMG activity (Image from [24]).

Experimental and clinical studies have shown that the SP can be altered. Hess et al [32] has demonstrated task-dependant changes in SP duration of various tasks. During tactile exploration of a small object, SP duration was longer than for an isometric contraction. Conversely, SP duration was shortened during visuomotor tasks compared to the isometric contraction. Clinical studies of individuals with neurological conditions have shown reduced SP in Parkinson's disease, thought to be due to decreased effectiveness of cortical inhibitory inputs following TMS [33]. However, lengthening of the SP has been shown in epilepsy patients [34] and in stroke patients [35] with these authors suggesting that the SP lengthening was due to a reduced drive of afferent signals to the M1. For further information on evoked potentials (latency, MEP and SP) from TMS, the reader is directed to Hallet [13,14], Pascual-Leone et al [36], Boniface & Ziemann [37], and Wasserman et al [20].

TMS Compared to Other Neuroimaging Techniques

A major difference between neuroimaging techniques, such as functional magnetic resonance imaging (fMRI), and TMS is that TMS has the ability to show excitation and inhibition of the corticospinal pathway, thereby demonstrating an area's direct influence on a motor performance. There are a number of studies that have directly shown increased excitability [38, 39] in a cortical motor area during task activation or, conversely, using TMS to transiently suppress regions of the cerebral cortex, providing stronger evidence of motor regions directly involved in a task [40]. It is for this reason that TMS has been pivotal in a variety of research areas within neurophysiology and motor control. Furthermore, many studies now combine both spatial imaging of techniques such as fMRI with temporal techniques such as TMS to investigate areas of the brain directly involved with motor function [41, 42].

TMS also has the ability to be used as a neuroimaging tool, illustrating topographical representation of muscles on the motor cortex in healthy [26], athletic [43], or conversely individuals who have suffered a neurological condition [5, 7]. Using a methodology based on a latitude and longitude system, TMS can be employed to stimulate a number of areas overlying the motor cortex systematically (Figure 3a). Averaging the MEP waveforms collected over each site (Figure 3b), a topographical matrix of MEP vs stimulus site can be generated to provide a representation of the muscle targeted during stimulation. An example of a map calculated from the topographical matrix is presented in Figure 3c. For an in depth discussion on the technique and methodology of TMS mapping, the reader is directed to Wilson et al [26] or Thickbroom & Mastaglia [44].

Neural Adaptations Following Motor Skill Training

Neural adaptations may occur at a number of sites within the neuromuscular pathway [45]. Changes may be as a result of strength training, but may also be the result of skill training, as it has been argued that strength training and skill training are closely connected in terms of adaptations occurring by similar mechanisms originating in the cerebral cortex [46].