Mapping the cortical representation of the lumbar paravertebral muscles

NE O'Connell MSc Centre for Research in Rehabilitation, Brunel University

DW Maskill MPhil Centre for Research in Rehabilitation Brunel University

J Cossar MSc Centre for Research in Rehabilitation Brunel University

AV Nowicky PhD Centre for Research in Rehabilitation Brunel University

Correspondence Address:

Neil O'Connell Centre for Rehabilitation Research School of Health Sciences and Social Care Brunel University Uxbridge Middlesex UB8 3PH United Kingdom

Tel: 01895 268814 Fax: 0208 847 2030

Email: neil.oconnell@brunel.ac.uk

Objective

The aim of this study was to map the cortical representation of the lumbar spine paravertebral (LP) muscles in healthy subjects.

Methods

Transcranial magnetic stimulation (TMS) was employed to map the cortical representations of the LP muscles at 2 sites. Stimuli were applied to points on a grid representing scalp positions. The amplitude of motor evoked potentials (n=6) were averaged for each position.

Results

The optimal site for evoking responses in the contralateral LP muscles was situated 1cm anterior and 4 cm lateral to the vertex. Ipsilateral responses were evoked from sites lateral to the optimal site for evoking contralateral responses. Contralateral responses were also obtained from areas anterior to the optimal site.

Conclusions

Maps of these muscles can be produced. The results suggest discrete contra and ipsilateral cortical projections. Anterior sites at which excitation can be evoked may indicate projections arising in the SMA are involved.

Significance

This study provides normative data regarding the cortical representation of the paravertebral muscles and provides a technique for evaluating cortical motor plasticity in patients presenting with spinal pathologies.

Introduction

Transcranial magnetic stimulation (TMS) has been extensively employed to map the topographic motor cortical representation in a range of muscles (McMillan et al., 1998; Wasserman et al., 1992; Wilson et al., 1993) and to investigate plastic changes in this representation in response to injury, immobilisation and pathology (Rossini & Pauri, 2000; Schwenkreis et al., 2001; Zanette et al., 1997).

The majority of this research has focused on muscles of the extremities. This being due to difficulty in generating reliable maps of the axial musculature. However various studies have identified direct corticospinal pathways to the muscles of the trunk (Nowicky et al., 2001; Ferbert et al., 1992; Plassman & Gandevia, 1989).

The trunk muscles play a key role in the maintenance of upright posture and balance. The corticospinal drive to the trunk muscles has been considered to have a stronger bilateral hemispherical input than that to the muscles of the extremities. Ferbert et al. (1992) identified contralateral and ipsilateral projections to the erector spinae muscle using TMS. This has also been demonstrated in other proximally situated muscles such as the diaphragm (Maskill et al., 1991) and the sternomastoid (Gandevia & Applegate, 1988) and abdominal muscles (Strutton et al., 2004; Tunstill et al., 2001).

Ferbert et al. (1992) employed TMS to investigate the corticospinal projection to the erector spinae muscles of the lumbar spine. They evoked MEPs in contralateral lumbar erector spinae muscles by cortical stimulation in 5 out of 9 subjects but the methodology employed did not provide an accurate location for the cortical representation of erector spinae.

Animal and human studies have demonstrated that nerve injury and cerebral infarcts lead to associated plastic reorganisation of the motor cortex (Toldi et al. 1996, Nudo et al. 1997, Nudo 1999). Changes include shrinkage of the cortical representation synonymous with the area of injury (Toldi et al. 1996), the corresponding area in the uninjured cortex and invasion of adjacent territory by neighbouring representations (Nudo et al. 1997). This motor cortical reorganisation can be influenced by training tasks and studies have shown that functional recovery mirrors further cortical reorganisation (Nudo et al. 1996, Liepert et al. 2000, Schmidlin et al. 2004).

Cortical plasticity of the representation of the back in the somatosensory cortex has been demonstrated in chronic low back pain (Flor et al. 1997) and plasticity in motor pathways of the thoracic paravertebral muscles has been found following spinal cord injury (Cariga et al. 2002). TMS mapping of paraspinal muscles may be a useful tool in the study of plastic changes in the motor cortex following spinal pathology and in monitoring the cortical effects of therapeutic interventions for these disorders.

As no study to date has generated comprehensive maps of the motor representation of the lumbar paravertebral (LP) musculature, the aim was to address this issue, with the objective of developing a robust methodology for the investigation of plasticity in response to pathologies affecting this muscle group and clinical interventions.

Methods

12 healthy volunteers, 2 male, 10 female aged between 19 and 51 years (mean 25 years) were investigated with the approval of the local Ethics committee. All volunteers gave informed consent prior to participation. 10 right hand dominant subjects with no previous history of low back pain or neurological disease were investigated.

The vertex (Cz) was determined using the international 10/20 system and was marked on a latex swimming cap. A line running laterally through Cz from the left to the right

antitragus and a line running sagittally from the external occipital protuberance to the nasion was drawn. From these reference lines a grid was drawn over the left cortex in 1cm steps antero-posteriorly from 1cm posterior to Cz to 5 cm anterior, and 2cm steps laterally from Cz to 8cm lateral.

Single stimuli were delivered using a repetitive biphasic Magstim Rapid TMS system (Magstim Co. Wales) in single pulse mode with a 70mm figure of 8 coil. Stimulation was applied with the coil handle oriented 45 degrees from the sagittal plane so that the induced current flowed in an anteromedial direction as suggested by Eguchi et al. (1995) and Ziemann et al. (1999). Ferbert et al.(1992) used 100% of maximum stimulator output to elicit responses from these muscles but reduced the stimulus intensity in subjects with readily indentifiable responses. In the current study pilot work mapping at a range of stimulator intensities indicated that the most reliable responses were obtained in most subjects using 100% of maximum stimulator output. Mapping was therefore performed using this intensity in all subjects.

Surface electromyography was recorded bilaterally from 2 sites: 3cm lateral to the L3 spinous process (recording site 1) and 1cm lateral to the spinous process of L5 (recording 2) using self-adhesive electrodes(Arbo, circular 28mm, Henleys Medical Supplies, Welwyn Garden City, UK). These sites have been used previously to successfully record EMG from the paravertebral muscles (Lariviere et al 2003). The ground electrode was placed over the centre of the sacrum.

EMG signals were recorded from the muscles (Quad 1902 signal conditioner ,Cambridge Electronic Design (CED), Cambridge UK), filtered (2nd order Butterworth, 12dB/octave, 20Hz-2kHz), amplified (1000x), and sampled (4kHz) using an A/D acquisition system (micro1401, CED) and personal computer running recording and analysis software (Signal for windows version 2.15 CED). All signals were averaged (n=6) and rectified for subsequent determination of MEP area. Correct electrode placement was confirmed by recording EMG whilst subjects performed a resisted back extension movement.

Pilot work identified difficulty in evoking repeatable responses with the muscles at rest. Subsequently subjects contracted the muscles by sitting forward in a chair and maintaining extension of the lower back. Biofeedback was given using a light box calibrated to a subject's maximum voluntary contraction. Subjects were instructed to maintain 30% of this maximum during stimulation.

With the subject performing this manoeuvre the average MEP response from 6 stimulations was obtained at each point. Stimuli were delivered with an inter-stimulus interval of 2-5 seconds. Stimuli were delivered starting at the midline and moving laterally for each latitude. The subjects were given rest periods between sets of stimuli to reduce the potential impact of fatigue.

Since facilitation was used, MEP amplitude was recorded after subtraction of the mean pre-stimulus EMG. The values of the average MEP area for each stimulation site were calculated and entered into a grid corresponding to the map on the subjects' cortex. All

responses were normalised by expressing them as a percentage of the peak response obtained from the specific recording site in each subject.

Following the first stage of testing, 3 subjects (all male age 33-64, mean age 46) were recruited to be mapped across both hemispheres. Subjects were mapped using a grid in 1cm steps antero-posteriorly from Cz to 5 cm anterior to Cz, and in 1cm steps laterally from Cz to 4cm lateral to Cz over the left and the right cortex to heighten resolution across the transverse axis. To eliminate possible current spread to the opposite hemisphere due to asymmetrical coil orientation the coil handle was oriented posteriorly in the sagittal plane. As before subjects were stimulated at 100% of maximum stimulator output with facilitation.

Results

MEPs were obtained from the contralateral muscles tested in 10 of the 12 subjects. In 2 subjects cortical stimulation evoked no measurable MEPs from the recording sites. It was possible to construct cortical maps for the LP muscles. Figure 1 illustrates the average map obtained for each muscle using the initial mapping protocol.

As in the Ferbert et al. (1992) study, latency of MEPs displayed marked variability both within and between subjects. Table 1 displays latency data for contralateral and ipsilateral responses.

The maps from both recording sites are similar and exhibit considerable overlap. The optimal site for evoking responses was situated 1cm anterior and 4 cm lateral to Cz. The map of contralateral recording site 2 but not recording site 1 reveals another area which produced large responses located 4cm anterior and 2 cm lateral to Cz. On the maps obtained of ipsilateral responses using the initial mapping protocol, large responses were found on the midline with smaller responses evoked from more lateral stimulation sites. Since these responses may have been due to unintentional stimulation of the right hemisphere with the coil orientation of 45°, 3 subjects were mapped as described across both hemispheres using a sagittal coil orientation. Figure 2 illustrates the maps obtained using this protocol.

This mapping protocol produced minimal responses from midline stimulation sites. In the contralateral muscles 2 separate hotspots can be seen at both recording sites. Using this protocol small differences are seen in the optimal site for evoking MEPs with the optimal site located at 2cm anterior and 2cm lateral to Cz for right sided muscles and 1cm anterior and 3 cm lateral for left sided muscles. A separate anteriorly situated hotspot can also be clearly seen. Ipsilateral responses were evoked from lateral stimulation sites.

Discussion

The results indicate that it is possible to map the cortical representation of the lumbar paravertebral muscles using TMS. The range of MEP latencies found is consistent with those found in other studies (Ferbert et al., 1992; Nowicky et al., 2001; (Plassman and Gandevia 1989) indicative of a fast-conducting corticospinal pathway.

Analysis of the contralateral maps illustrates that the hotspot at both sites was the same. Due to the multiple peaks found on some maps centre of gravity calculation was not deemed appropriate.

The results support previous findings (Ferbert et al., 1992), of an ipsilateral projection to trunk muscles from the cortex. As with the findings of Ferbert et al. (1992) in the erector spinae and Ziemann et al. (1999) in hand and arm muscles, ipsilateral responses were evoked when stimulating at lateral sites. The midline ipsilateral responses seen using the original mapping protocol may be the result of unintentional stimulation of the opposite cortex since they disappear when mapping using a sagittal coil orientation. Maskill et al. (1991) found ipsilateral responses in the diaphragm which were stronger near the midline but accepted the possibility that these may be due to stimulation of the opposite hemisphere. The current results support this conclusion.

The location of ipsilateral responses and the coil orientation used in this experiment makes it unlikely that they are the result of current spread to the opposite hemisphere. Ipsilateral responses had a longer latency than contralateral responses of around 4 msecs. These findings are in agreement with those of Ziemann et al. (1999) who demonstrated slower ipsilateral responses in upper limb muscles obtained more prominently from sites lateral to the optimal stimulation site for contralateral responses.

The small differences observed between the hotspots using the different mapping protocols may be the result of the alteration in coil orientation and thus current direction. The increased resolution of the map grid in the bilateral protocol may also have contributed towards this.

The presence of an excitable area of the cortex from anterior stimulation sites is an interesting finding. The bilateral mapping protocol demonstrates that this anterior site is only seen clearly in contralateral muscles. Large responses evoked from relatively anterior stimulation sites may indicate excitation of the supplementary motor area (SMA). Studies have confirmed a somatotopic organization of the pre-motor area (PMA) and the SMA (Fink et al., 1997; Godschalk et al., 1995). Sharshar et al. (2004) demonstrated a discrete anterior site for evoking MEPs in the diaphragm and suggest that this anterior area may represent the SMA. In the current study the latency of large responses obtained from anterior sites was not significantly different to that found at the hotspot. As with the findings of Sharshar et al. (2004) the short latency responses found from anterior stimulation sites in the current study suggest a direct projection from the SMA. Imaging studies would be of use in determining whether these anterior spots do represent the SMA.

The variability seen in the latencies of MEPs may reflect the activation of differing pathways to these muscles due to the necessary use of high stimulation intensity with active motor facilitation. The difficulty in determining onset latency in the presence of background EMG may mitigate this variability. The shorter latency responses may reflect a fast-conducting direct motor pathway. Longer latency responses may indicate an oligosynaptic pathway.

The wide distribution of responses observed and the finding of multiple peaks in some maps limits the use of CoG in accurately ascertaining map position. Map area was not analysed for similar reasons. This is primarily due to the chosen protocol of stimulating all subjects at 100% of stimulator output with voluntary activation of the muscle. The bilateral mapping methodology using a sagittal coil orientation produced good quality maps from which ipsi and contralateral responses could be clearly distinguished. It is therefore recommended that future mapping studies for these muscles adopt this approach.

It is important to consider what influence the use of a biphasic stimulator may have had on the results. Studies have shown that biphasic stimulation may excite larger populations of neurons than monophasic stimulation and that the resting motor threshold for biphasic stimulation is lower than that for monophasic stimulation (Arai et al. 2005, Sommer et al. 2002). Thus maps obtained with monophasic stimulation may vary from those found in the current study. Since biphasic stimulation presents a more powerful stimulus it may be more successful in eliciting MEP's from these muscles from a wider cortical area. Given the technical difficulties encountered in obtaining repeatable responses from these muscles at lower stimulus intensities it appears unlikely that monophasic stimulation would provide more information at these intensities. The influence of different stimulation waveforms on the results of cortical mapping is an interesting direction for further study.

Using this methodology the parameters of hotspot location, response latency and MEP amplitude may provide parameters by which plastic changes may be investigated and monitored in response to pathology and therapeutic intervention.

References

Arai N, Okabe S, Furubayashi T et al. Comparison between short train, monophasic and biphasic repetitive transcranial magnetic stimulation (rTMS) of the human motor cortex. Clin Neurophysiol 2005;116:605-613

Cariga P, Catley M, Nowicky AV et al. Segmental recording of cortical motor evoked potentials from thoracic paravertebral myotomes in complete spinal cord injury. Spine 2002; 27:13:1438-43

Eguchi K, Fujii H, Suzuki E et al. The effect of coil orientation on the reliability of transcranial magnetic stimulation for mapping a cortical representation of the first dorsal

interosseous muscle. Electroencephalogr Clin Neurophysiol Motor Control 1999; 97:4:S107

Ferbert A, Caramia D, Priori A et al. Cortical projection to erector spinae muscles in man as assessed by focal transcranial magnetic stimuation. Electroencephalogr Clin Neurophysiol 1992;85:382-387

Fink GR, Frackowiak RS, Pietrzyk U et al. Mulitple non-primary motor areas in the human motor cortex. J Neurosci 1997;77:2164-2174

Flor H, Elbert T, Braun C et al. Extensive cortical reorganization in chronic back pain patients. NeuroImage 1997; 5:4:S216

Gandevia SC, Applegate C. Activation of neck muscles from the human motor cortex. Brain 1988;111:801-813

Godschalk M, Mitz AR, van Duin B et al. Somatotopy of monkey premotor cortex with microstimulation. Neurosci Res 1995;23:269-279

Lariviere C, Arsenault AB, Gravel D et al. Surface electromyography assessment of back muscle intrinsic properties. J EMG Kinesiol 2003; 13:4:305-318

Liepert J, Graef S, Uhde I et al. Training induced changes of motor cortex representations in stroke patients. Acta Neurol Scand 2000;321-326

Maskill DW, Murphy K, Mier A et al. Motor cortical representation of the diaphragm in man. J. Physiol 1991;443:105-121

McMillan AS, Watson C, Walshaw D. Transcranial magnetic stimulation mapping of the cortical topography of the human masseter muscle. Arch Oral Biol 1998;43:925-931

Nowicky AV, Mcgregor AH, Davey NJ. Corticospinal control of human erector spinae muscles. Motor Control 2001;3:270-280

Nudo RJ, Wise BM, Sifuentes F et al. Neural substrates for the effects of rehabilitative training on motor recovery after ischaemic infarct. Science 1996;1791-1794

Nudo RJ, Plautz EJ, Milliken GW Adaptive plasticity in primate motor cortex as a consequence of behavioural experience and neuronal injury. Semin Neurosci 1997;9:13-23

Nudo RJ. Recovery after damage to motor cortical areas. Curr Opinion Neurobiol 1999;9:740-747

Plassman BL, Gandevia SC. Comparison of human motor cortical projections to abdominal muscles and intrinsic muscles of the hand. Exp Brain Res 1989;78:301-308

Rossini PM, Pauri F. Neuromagnetic integrated methods tracking human brain mechanisms of sensorimotor areas "plastic" organization. Brain Res Revs 2000;33:131-154

Schmidlin E, Wannier T, Bloch J et al. Progressive plastic changes in the hand representation of the primary motor cortex parallel incomplete recovery from a unilateral section of the corticospinal tract at cervical level in monkeys. Brain Research 2004; 1017:172-183

Schwenkreis P, Witscher K, Janssen F et al. Assessment of reorganization in the sensorimotor cortex after upper limb amputation. Clin Neurophysiol 2001;627-635

Sharshar T, Hopkinson NS, Jonville S et al. Demonstration of a second rapidly conducting cortico-diaphragmatic pathway in humans. J Physiol 2004;560:3:897-908

Sommer M, Lang N, Tergau F et al. Neuronal tissue polarization induced by repetitive transcranial magnetic stimulation? Neuroreport 2002; 13:6:809-811

Strutton PH, Beith ID, Theodorou S et al. Corticospinal activation of internal oblique muscles has a strong ipsilateral component and can be lateralized in man. Exp Brain Res 2004;158:474-479

Toldi J, Laskawi R, Landgrebe M et al. Biphasic reorganization of somatotopy in the primary motor cortex follows facial nerve lesions in adult rats. Neurosci Letters 1996;203;179-182

Tunstill SA, Wynn-Davies AC, Nowicky AV et al. Corticospinal facilitation studied during voluntary contraction of human abdominal muscles. Exp Physiol 2001;86:131-136

Wasserman EM, McShane LM, Hallett M et al. Noninvasive mapping of muscle representations in human motor cortex. Electroenceph & Clin Neurophysiol 1992;1-8

Wilson SA, Thickbroom GW, Mastaglia FL. Transcranial Magnetic Stimulation mapping of the motor cortex in normal subjects. The representation of two intrinsic hand muscles. J Neurol Sci 1993;118:2:134-144

Zanette G, Tinazzi M, Bonato C et al. Reversible changes of motor cortical outputs following immobilization of the upper limb. Electroenceph & Clin Neurophysiol 1997;105:269-279

Ziemann U, Ishii K, Borgheresi A et al. Dissociation of the pathways mediating ipsilateral and contralateral motor-evoked potentials in human hand arm muscles. J Physiol 1999;518:3:895-906

Figures and Tables

Figure 1. Averaged cortical maps (n=10) for the 4 recording sites using the initial mapping protocol of stimulating the left cortex with the coil handle oriented at 45° . %MR= amplitude expressed as a percentage of the maximum response obtained from that recording site.

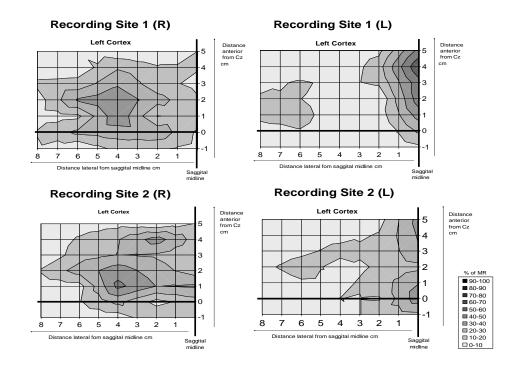


Figure 2. Averaged cortical maps (n=3) for the 4 recording sites using the second mapping protocol of stimulating the left and right cortex with the coil handle oriented in the sagittal plane.

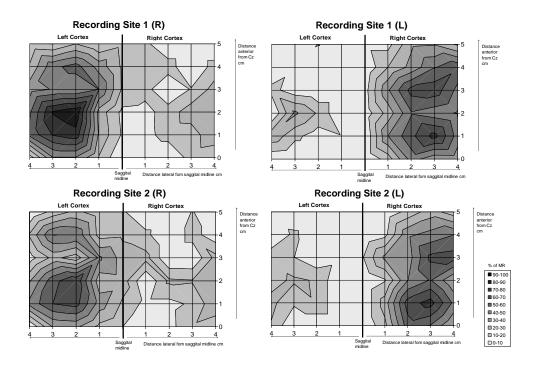


Table 1. Mean (±Standard Deviation) and range of MEP latencies (msecs) obtained from the optimal site for obtaining responses at each recording site in each subject, and for all responses obtained in each subject.

	Contralateral Responses	Ipsilateral Responses
Mean latency at optimal site	19.53 ± 4.78	24.19 ± 3.77
Range of latencies at optimal site	12.10 –27.70	18.60 – 31.53
Mean Latency all responses	19.72 ± 4.41	24.3 ± 5.97
Range of latencies all responses	12.05 - 30.48	14.06 - 36.45