Abstract Although it is well known that the corticospinal system exerts more influence over distal (hand and fingers) than proximal (elbow and shoulder) upper limb muscles, differences in the importance of this system for voluntary activation of these muscle groups have not been demonstrated directly. Two investigations were carried out to provide a quantitative comparison of the contribution of fast corticospinal inputs to voluntary activity in proximal and distal muscles of normal subjects. The first study confirmed that the rate of increase in the amplitude of EMG responses to transcranial magnetic stimulation (TMS) with voluntary activation of the muscles was significantly greater in a hand muscle (first dorsal interosseous, 1DI) than in biceps, which was in turn greater than that for deltoid. The second study demonstrated that this result reflected a genuine difference in corticospinal influence over these muscles and was not due to differences in the pattern and type of motor unit recruitment in proximal vs distal muscles. The voluntary activation of a pair of low-threshold single motor units (SMUs) in 1DI and deltoid was compared with their response to TMS. In both muscles only a small amount of additional effort was required to recruit the second SMU; increments were typically within 1% of maximum voluntary contraction, as assessed from EMG measurements. Subjects were asked to voluntarily discharge the lower threshold SMU at a steady rate, and then the threshold of responses of this SMU and that of the second unit to TMS were determined. In 1DI, only small increments in TMS intensity above the threshold for the first SMU were required to activate the second unit [mean 1.4% maximum stimulator output (MSO), SD±1.0%, n=7 subjects]. In contrast, in deltoid a significantly greater intensity increase was needed (mean 6%, SD±1.2%, MSO n=7, P<0.001). Similar results were obtained when TMS thresholds of motor unit pairs were assessed in relaxed subjects. These experiments support the hypothesis that the fast corticospinal input that can be activated by TMS is of greater importance for the voluntary activation of hand than of shoulder muscles. This hypothesis served as a basis for testing deltoid responses in three stroke patients. In two patients smaller responses to TMS were obtained on the affected side than on the unaffected side during the production of equivalent voluntary contractions, suggesting that the patients achieved these contractions using inputs other than the fast corticospinal elements excited by TMS.

Key words Arm · Hand · Magnetic stimulation · Corticospinal · Stroke

Introduction

The corticospinal system is known to exert a greater excitatory influence over distal than over proximal upper limb muscles (Palmer and Ashby 1992a; Colebatch and Gandevia 1989; see Porter and Lemon 1993). This difference is reflected in muscle responses to corticospinal inputs generated by transcranial magnetic stimulation (TMS): in healthy subjects, responses in proximal muscles have higher thresholds, smaller amplitudes and are generally more difficult to elicit than in distal muscles (see Rothwell et al. 1991). It is also well known that the response to TMS is facilitated by voluntary contraction of the test muscle. This facilitation probably involves mechanisms operating at both the cortical level (due to the increased level of cortical excitability associated with the command to contract the muscle) and at the spinal level (due to other sources of excitatory drive necessary...

Given the important differences in corticospinal influence over proximal and distal upper limb muscles, it is likely that the relative contribution of these cortical vs spinal mechanisms may also differ. Kischka et al. (1993) reported that as subjects increased the level of voluntary contraction, there were much steeper increases in the response to TMS in an intrinsic hand muscle than in biceps. There are significant differences in the pattern of voluntary motor unit recruitment in hand versus proximal arm muscles: in 1DI a large proportion of motor units are already activated at low contraction forces, compared to a more gradual recruitment in more proximal muscles such as biceps or deltoid (Milner-Brown et al. 1973; Kukulka and Clamann 1981; DeLuca et al. 1982). These differences could contribute to the contrast in responses to TMS at different levels of voluntary effort, and it is therefore difficult to pinpoint the site of facilitation as spinal and/or cortical.

In this study we have assessed the importance of corticospinal inputs for voluntary activation of different upper limb muscles in three ways. In the first part of the study, we examined the surface EMG response of three upper limb muscles (1DI, biceps and deltoid) to TMS of constant intensity at different levels of voluntary contraction; this confirmed the earlier findings of Kischka et al. (1993). In the second part we demonstrated that the steeper increase in response seen in 1DI, as compared with biceps or deltoid, was not due to differences in motor unit recruitment. This was shown by comparing the thresholds of pairs of motor units activated either voluntarily or by TMS. We found significant differences between proximal and distal muscles in the intensity of TMS required to activate successively each member of a pair of low-threshold units; such differences are most likely to reflect the importance of the corticospinal input for voluntary activation of the respective motoneurone pools.

In the final part of our study we examined the relationship between the response to TMS and voluntary activation in three stroke patients who had recovered the ability to contract their shoulder muscles. In two of these patients we found much smaller responses to TMS with equivalent levels of voluntary activation on the affected side compared to the unaffected side. This indicated that voluntary activation of the affected muscle involved a possible upregulation of inputs other than those derived from fast corticospinal elements excited by TMS. These non-corticospinal inputs, which are particularly prominent for motoneurones supplying more proximal muscles (Kuypers 1981), may compensate for the reduction or even complete loss of the fast corticospinal inputs to shoulder muscles after stroke (Turton et al. 1996), and explain the return of function in these muscles after stroke (Twitchell 1951). This compensatory mechanism appears to be lacking for the more distal muscles (Turton et al. 1996).

Materials and methods

Subjects

Nineteen normal volunteers (nine males) aged 20–57 years participated. Three patients were also studied: JB and DF (both male, aged 59 and 70 years) and RD (female, 52 years). All three had a right hemiparesis of varying degrees of severity; details are given in Table 3. In both DF and RD there was a single infarcted area in the left internal capsule. JB had a large cerebral haemorrhage which affected the left hemisphere both cortically and subcortically. He was the most severely affected of the three patients, having dysphasia, sensory and proprioceptive loss in addition to weakness. All subjects gave informed consent to the procedures used, which were ratified by the local Research Ethics Committee. None of the normal subjects had suffered from epilepsy or other neurological disorder. Three of the subjects (1, 2 and 4) took part in both the surface EMG and single motor unit experiments.

Magnetic stimulation

A Magstim 200 stimulator (maximum output 2.0 T; Magstim Company, Dyfed, UK) with a double-cone coil was used (outer diameter 110 mm, angle between the windings 100°). The coil was secured to the head with Velcro straps; the weight of the coil and its heavy cable were counterbalanced by elastic bands suspended from an overhead gantry.

Surface EMG experiments (12 subjects)

Recording

Surface EMGs were recorded from middle deltoid, biceps and the 1DI on the dominant side (three left-handers) using 10-mm ARBO electrodes placed with their centres 20–30 mm apart over the muscle bellies and a Neurolog NL104 amplifier system (Digitimer Ltd., UK). Recordings from the middle deltoid on both the affected and unaffected sides were taken from the patients. Surface EMG was amplified (>500), filtered (high-pass filter set at 30 Hz) and full wave rectified. EMGs were digitised at 4 kHz and acquired via a 1401 interface (Cambridge Electronic Design) using SIGAVG software. At the beginning and end of each experiment we recorded the EMG while subjects performed a maximum voluntary contraction (MVC) of each tested muscle. Middle deltoid MVC was measured as the seated subject abducted the shoulder, at an angle of 45° to the midline, against a rigid strap secured to the back of the subject’s chair and attached to the arm just above the elbow. For biceps MVC the forearm was supinated and then restrained by a rigid strap at the wrist; the elbow was flexed at about 120°. For 1DI MVC the hand was placed flat on a board in front of the subject and the index finger and thumb positioned between padded blocks; the subject abducted the index finger and pressed against the block. Three successive recordings of each MVC were taken; each lasted 2–3 s. Visual feedback of the level of rectified and smoothed EMG activity was given to subjects as a percentage of their MVC by a vertical row of ten LEDs. In the patients MVC was assessed on the unaffected side first.

Procedure

After determination of the MVC, subjects had a short period in which they practised using visual feedback to produce steady con-