PERSPECTIVES

Corticospinal involvement in volitional contractions

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It is more than 25 years since Merton & Morton (1980) first demonstrated that it was possible to stimulate fast conducting corticospinal connections from motor cortex to spinal motoneurones in conscious humans. The initial stimulators used high-voltage electrical pulses that produced an uncomfortable twitch of scalp muscles under the stimulating electrodes. However, the advent of transcranial magnetic stimulation (TMS) made the procedure painless and it is now widely used to study motor cortical physiology.

Despite its success, one thorny question remains: are the corticospinal connections activated by transcranial stimulation the same as those that are normally recruited in voluntary muscle contractions? It is undoubtedly true that the size of the response to a TMS pulse is modulated by volitional muscle contraction, but that does not mean that the outputs activated by the TMS pulse are the same as those used volitionally. So how do we tackle the question?

One potential solution lies in the observation that stimulation at very low intensities, below the threshold for evoking a muscle twitch, can reduce ongoing volitional muscle activity. This is thought to occur by suppression of ongoing corticospinal excitation to motoneurones by activation of intracortical inhibitory circuits (Davey *et al.* 1994). The result means that TMS pulses can certainly affect volitionally produced activity in corticospinal fibres, but are these the same fibres as are activated by suprathreshold pulses? Unfortunately, the answer appeared to be no. The latency of electromyographic (EMG) suppression after a subthreshold TMS pulse had usually been found to be about 10 ms later than the onset of facilitation caused by a suprathreshold pulse. One explanation for the unusually long delay of this suppression was that in fact the TMS pulse was suppressing activity in slowly conducting corticospinal fibres, and that these, rather than the large diameter ones activated by suprathreshold pulses, were the source of volitional excitation to spinal motoneurones.

However, as Butler *et al.* (2007) point out in this issue of *The Journal of Physiology*, there is one potential flaw in these measurements. They were all made in average recordings of rectified surface EMG activity. Determining the true onset of inhibition in such records is notoriously difficult because muscle action potentials recorded by the surface electrodes have a duration of about 5–10 ms (Widmer & Lund, 1989). Imagine it was possible to stop all the units firing at the same instant. Electrical activity would still continue for 5–10 ms as the final action potentials travelled down the muscle fibres, and it is this that complicates estimation of the onset of inhibition.

In the present experiments, Butler *et al.* bypassed this problem by recording the activity of single motor units with needle electrodes, and constructing a peri-stimulus time histogram (PSTH) of discharge around the time of a TMS pulse to the motor cortex. This should give a true estimate of the difference in timing of facilitation evoked by a suprathreshold TMS pulse and inhibition produced by a subthreshold pulse. However, one last problem remained to be solved. A reliable measure of the onset of inhibition in a PSTH requires there to be a large number of counts in the baseline. Units discharge at around 10 Hz in the weak contractions that are needed to identify single motor units in human muscle. Thus, about 100 trials are needed to fill each 1 ms bin of the PSTHs used in these experiments. Since TMS pulses can be applied at about once every 5 s, this would take just under 10 min of recording. A good number of counts per bin to identify inhibition would be about 10, which would take some 1.5 h of experimenting.

The solution to making the experiments tolerable for subjects is to manipulate the experimental conditions so that the onset of inhibition coincides with the expected time of firing of a unit. That way the number of counts per bin is high in the time interval of interest. This can be arranged by timing the TMS pulse with respect to the previous discharge of the unit such that the presumed onset of inhibition is about 100 ms later.

With this arrangement, Butler *et al*. found that the difference in latency of TMS-evoked suppression was around 2 ms longer than facilitation when recordings were taken from the first dorsal interosseous muscle and 3 ms for the biceps brachii. Assuming an oligosynaptic (or even disynaptic) inhibition, this is compatible with inhibition of activity in the fast conducting fibres of the corticospinal tract. Thus, it appears that TMS is indeed likely to activate the same fast conducting corticospinal fibres as are used in volitional contractions. Perhaps more importantly, we now have a method of testing the extent of this contribution in different types of muscle contraction, from volitional finger and arm movements to walking and breathing.

References

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