Arm posture-dependent changes in corticospinal excitability are largely spinal in origin

James L. Nuzzo,1,2 Gabriel S. Trajano,1 Benjamin K. Barry,1,2 Simon C. Gandevia,1,3 and Janet L. Taylor1,2
1Neuroscience Research Australia, Randwick, New South Wales, Australia; 2School of Medical Sciences, University of New South Wales, Kensington, New South Wales, Australia; and 3Prince of Wales Clinical School, University of New South Wales, Kensington, New South Wales, Australia

Submitted 15 September 2015; accepted in final form 6 February 2016

Nuzzo JL, Trajano GS, Barry BK, Gandevia SC, Taylor JL. Arm posture-dependent changes in corticospinal excitability are largely spinal in origin. J Neurophysiol 115: 2076–2082, 2016. First published February 10, 2016; doi:10.1152/jn.00885.2015.—Biceps brachii motor evoked potentials (MEPs) from cortical stimulation are influenced by arm posture. We used subcortical stimulation of corticospinal axons to determine whether this postural effect is spinal in origin. While seated at rest, 12 subjects assumed several static arm postures, which varied in upper-arm (shoulder flexed, shoulder abducted, arm hanging to side) and forearm orientation (pronated, neutral, supinated). Transcranial magnetic stimulation over the contralateral motor cortex elicited MEPs in resting biceps and triceps brachii, and electrical stimulation of corticospinal tract axons at the cervicomedullary junction elicited cervicomedullary motor evoked potentials (CMEPs). MEPs and CMEPs were normalized to the maximal compound muscle action potential (Mmax). Responses in biceps were influenced by upper-arm and forearm orientation. For upper-arm orientation, biceps CMEPs were 68% smaller (P = 0.001), and biceps MEPs 31% smaller (P = 0.012), with the arm hanging to the side compared with when the shoulder was flexed. For forearm orientation, both biceps CMEPs and MEPs were 34% smaller (both P < 0.0046) in pronation compared with supination. Responses in triceps were influenced by upper-arm, but not forearm, orientation. Triceps CMEPs were 46% smaller (P = 0.007) with the arm hanging to the side compared with when the shoulder was flexed. Triceps MEPs and biceps and triceps MEP/CMEP ratios were unaffected by arm posture. The novel finding is that arm posture-dependent changes in corticospinal excitability in humans are largely spinal in origin. An interplay of multiple reflex inputs to motoneurons likely explains the results.

biceps brachii; cervicomedullary motor evoked potential; motoneuron; motor cortex; spinal cord

CORTICOSPINAL EXCITABILITY to arm muscles is influenced by arm posture. In upper-arm, forearm, and hand muscles, sizes of motor evoked potentials (MEPs) from transcranial magnetic stimulation change depending on the orientation of the upper arm and forearm (Dominici et al. 2005; Ginanneschi et al. 2005, 2006; Mazzocchio et al. 2008a, 2008b; Mitsuhashi et al. 2007; Mogk et al. 2014; Perez and Rothwell 2015). In resting biceps brachii, MEPs increase when the upper arm is abducted and the forearm supinated (Mogk et al. 2014; Peterson et al. 2014). Peripheral or methodological factors (e.g., muscle length, muscle orientation relative to electrodes) do not fully account for such effects and indicate a central mechanism (Mogk et al. 2014). Changes in MEPs are often thought to reflect cortical changes, but processes within the spinal cord also play a role (Di Lazzaro et al. 1998; Hess et al. 1987; Ugawa et al. 1995). Thus it is unclear if the effect of arm posture on corticospinal excitability is due to processes within the cortex or spinal cord, or both.

Responses to subcortical stimulation of corticospinal axons (i.e., cervicomedullary evoked potentials, CMEPs) can help address cortical vs. spinal contributions. CMEPs reflect the efficacy of corticospinal-motoneuronal transmission and/or excitability of the motoneurons. CMEPs travel along the same corticospinal axons as MEPs (Gandevia et al. 1999); they have a large monosynaptic component in biceps brachii (Petersen et al. 2002), and they are not subject to conventional presynaptic inhibition (Jackson et al. 2006; Nielsen and Petersen 1994). Only one study of a hand muscle has examined the effect of arm posture on CMEPs (Perez and Rothwell 2015). It was found that during a static finger grasp, MEPs, but not CMEPs, of the first dorsal interosseous increased when the forearm was neutral compared with when it was supinated or pronated. This suggests that, in intrinsic hand muscles, posture-dependent changes in corticospinal excitability are due to processes within the cortex.

In contrast, findings from other studies suggest that arm posture-dependent changes in corticospinal excitability have a spinal origin. In monkeys, responses evoked in resting hand and forearm muscles from subcortical stimulation are influenced by upper-arm orientation, and this effect remains after the spinal cord has been transected above the site of stimulation (Yaguchi et al. 2015). In humans, F waves, as well as MEPs, in resting abductor digiti minimi increase when the upper arm is horizontally adducted compared with horizontally abducted (Ginanneschi et al. 2005; Mazzocchio et al. 2008a).

In human biceps (Mitsuhashi et al. 2007; Mogk et al. 2014; Peterson et al. 2014; Renner et al. 2006) and triceps brachii (Mitsuhashi et al. 2007; Renner et al. 2006), posture-dependent changes in corticospinal excitability have only been investigated with transcranial magnetic stimulation. Thus it remains uncertain as to where in the central nervous system these effects are modulated. Therefore, to determine whether arm posture-dependent changes in MEPs are spinal in origin, we obtained MEPs and CMEPs in resting biceps and triceps brachii while subjects assumed several static arm postures. These postures differed in both upper-arm and forearm orientation. In this article, the term “arm” refers to the entire upper limb, whereas “upper arm” refers to the region from shoulder...
to elbow and “forearm” refers to the region from elbow to wrist.

METHODS

Ethical approval. The study protocol was approved by the University of New South Wales Human Research Ethics Committee and complied with the Declaration of Helsinki (2008). All subjects provided written informed consent to participate.

Subjects. Twelve right-handed subjects (age 22.6 ± 4.4 yr, 7 males) participated. Subjects were included if they reported no contraindications to transcranial magnetic stimulation (e.g., epilepsy, metal in the body), were not currently taking medications that may contraindicate transcranial magnetic stimulation (e.g., epilepsy, males) participated. Subjects were included if they reported no con-

Experimental setup. Subjects were seated in an adjustable chair.

The right arm was configured into the desired posture (see Experiment protocol) using rigid arm bars and adjustable straps (Fig. 1A). Surface electrodes (Ag-AgCl) recorded electromyographic (EMG) activity from the right biceps and triceps brachii. For biceps brachii, the proximal electrode was placed over the mid belly, and the distal electrode was placed over the distal tendon. For triceps brachii, the proximal electrode was positioned halfway between the humerus head and olecranon process, and the distal electrode was placed over the distal tendon. The signals were filtered and amplified (16–1,000 Hz; CED 1902 amplifier; Cambridge Electronics Design, Cambridge, UK), and the sampling rate was 2,000 Hz. All analog signals were digitized and stored onto a personal computer using a laboratory interface (CED 1401 and Spike2 software; Cambridge Electronics Design).

Transcranial magnetic stimulation. Magnetic stimulation (Mags- tim 200; Magstim, Whitland, UK) of the corticospinal axons at the cervicomедullary junction was used to obtain CMEPs in the right biceps and triceps brachii at rest. The stimulating electrodes (self-adhesive Ag-AgCl) were positioned at the grooves between the mastoid processes and occipital bone. The cathode and anode were positioned on the left and right sides, respectively. Stimulation intensity (range 123–170 mA) was that which induced a CMEP in biceps brachii equal to ~5% of Mmax, with the arm in the Shoulder Abducted Supinated position.

Brachial plexus stimulation. Electrical stimulation (200-μs duration; DS7AH constant current stimulus) of the brachial plexus was delivered at Erb’s point to obtain the M wave for the right biceps and triceps brachii at rest. The cathode was positioned over the supracla-

Fig. 1. Schematic representation of the setup (A) and experimental protocol (B). A: depiction of Hanging Neutral, Shoulder Flexed Neutral, and Shoulder Abducted Supinated arm postures. B: sets of evoked responses were obtained in 7 different arm postures. Each set (indicated by black arrows) consisted of 5 cervicomедullary motor evoked potentials (CMEPs), 10 mo-

tor evoked potentials (MEPs), and 2 maximal compound muscle action potentials (Mmax). The 7 postures were selected because they have been used in studies assessing changes in corticospinal excitability to biceps brachii with different arm postures (Mogk et al. 2014) and different strength training regimens (Nuzzo et al. 2016). In the Shoulder Flexed postures, the shoulder and elbow were both flexed 90°. In the Shoulder Abducted postures, the shoulder was abduced ~75° and horizontally adducted ~30° while the elbow was flexed ~120°.

Sets of evoked responses were obtained in each posture. Measures within each set occurred in the following order: 5 CMEPs, 1 Mmax, 10 MEPs, and 1 Mmax. Each stimulus was delivered 10 s apart, and EMG was monitored to ensure subjects were relaxed. Each of the seven arm postures was assumed twice with a set of measurements obtained on each occasion, resulting in 10 CMEPs, 20 MEPs, and 4 Mmax measures per posture (Fig. 1B).

Data analysis. Areas of each individual MEP, CMEP, and Mmax waveform were measured. For each posture, the areas of the 20 MEPs were then averaged to represent one value, as were the areas of the 10
CMEPs and 4 $M_{\text{max}}$. MEPs and CMEPs in a given posture were normalized to $M_{\text{max}}$ from the same posture. Root mean square (RMS) amplitude of biceps and triceps brachii EMG was measured across the 100 ms preceding each stimulus, and for this calculation, a digital second-order Butterworth notch filter (bandstop of 49 and 51 Hz) was used to eliminate any 50-Hz noise in the EMG.

Statistical analysis. Statistical analyses were conducted with SPSS (version 21; IBM, Armonk, NY). A two-way repeated-measures ANOVA was used to assess the effects of upper-arm and forearm orientation (and any interaction) on each of the evoked potentials. This analysis (2 upper-arm orientations × 3 forearm orientations) did not include data from the hanging neutral posture. Mauchly’s test was used to assess sphericity of repeated measures. In cases where sphericity was violated, Huynh-Feldt corrections were applied. Post hoc pairwise comparisons were made with Bonferroni correction. In a further analysis of the evoked potentials, data from the Hanging Neutral posture were compared with data from the Shoulder Flexed Neutral and Shoulder Abducted Neutral postures using dependent $t$-tests with Bonferroni correction. Statistical significance was set at the 0.05 alpha level. Data are means ± SD. Some data were excluded from analysis. First, individual MEPs or CMEPs were excluded if the RMS amplitude of the EMG in the 100 ms preceding stimulation was $\geq 0.004$ mV. A total of 76 of the 2,520 evoked potentials were excluded for this reason. Second, a subject’s entire set of MEPs or CMEPs were excluded if, in more than one arm posture, peak-to-peak amplitudes of half of the potentials with no preceding EMG were $\leq 50$ µV. Also, in one subject, biceps and triceps brachii CMEPs were excluded because of a technical issue with the cervicomedullary stimulation. With these criteria, the final sample sizes for each variable were as follows: biceps brachii CMEPs ($n = 11$), biceps brachii MEPs ($n = 11$), triceps brachii CMEPs ($n = 9$), and triceps brachii MEPs ($n = 6$). Biceps brachii MEP/CMEP ratios were also computed ($n = 10$), as were triceps MEP/CMEP ratios ($n = 5$).

RESULTS
A summary of key findings is presented in Table 1.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Forearm</th>
<th>Upper Arm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biceps</td>
<td>MEP</td>
<td>N, S</td>
</tr>
<tr>
<td></td>
<td>CMEP</td>
<td>n &lt; S</td>
</tr>
<tr>
<td></td>
<td>$M_{\text{max}}$</td>
<td>n &gt; S</td>
</tr>
<tr>
<td></td>
<td>MEP/CMEP</td>
<td>n effect</td>
</tr>
<tr>
<td></td>
<td>$M_{\text{max}}$</td>
<td>n effect</td>
</tr>
</tbody>
</table>

Some data were excluded from analysis. First, individual MEPs or CMEPs were excluded if the RMS amplitude of the EMG in the 100 ms preceding stimulation was $\geq 0.004$ mV. A total of 76 of the 2,520 evoked potentials were excluded for this reason. Second, a subject’s entire set of MEPs or CMEPs were excluded if, in more than one arm posture, peak-to-peak amplitudes of half of the potentials with no preceding EMG were $\leq 50$ µV. Also, in one subject, biceps and triceps brachii CMEPs were excluded because of a technical issue with the cervicomedullary stimulation. With these criteria, the final sample sizes for each variable were as follows: biceps brachii CMEPs ($n = 11$), biceps brachii MEPs ($n = 11$), triceps brachii CMEPs ($n = 9$), and triceps brachii MEPs ($n = 6$). Biceps brachii MEP/CMEP ratios were also computed ($n = 10$), as were triceps MEP/CMEP ratios ($n = 5$).
was affected by upper-arm orientation ($F_{1,10} = 7.39$, $P = 0.022$; Shoulder Flexed > Shoulder Abducted). To assess whether the prestimulus EMG in the Shoulder Flexed postures influenced subsequent biceps brachii CMEPs, correlations were performed between these two variables for each individual subject. The mean of these intrasubject correlations was $r = 0.10$ (range: $r = -0.36$ to 0.66). Thus the increased prestimulus EMG in the Shoulder Flexed postures did not consistently influence biceps brachii CMEPs.

Biceps brachii MEP/CMEP ratios were generally not affected by arm posture. There were no main effects for forearm orientation ($F_{2,18} = 1.124$, $P = 0.347$) or upper-arm orientation ($F_{1,9} = 3.598$, $P = 0.090$), and no interaction ($F_{1,194,10,748} = 0.63$, $P = 0.472$). The ratio tended to be larger in Hanging Neutral (1.26 ± 1.53) compared with Shoulder Flexed Neutral (0.42 ± 0.45) and Shoulder Abducted Neutral (0.48 ± 0.42), but these comparisons were not significantly different ($P = 0.059$ and 0.061, respectively).

Biceps brachii $M_{\text{max}}$ was smallest when the forearm was supinated or the arm hung to the side (Fig. 3C). ANOVA showed a main effect of forearm orientation ($F_{1,17,12.875} = 12.878$, $P = 0.003$) but not upper arm orientation ($F_{1,11} = 1.952$, $P = 0.19$). For forearm orientation, biceps brachii $M_{\text{max}}$ in Supinated postures was 12% smaller than in Neutral postures ($P = 0.009$) and 15% smaller than in Pronated postures ($P = 0.012$). Also, there was an interaction between upper-arm and forearm orientation ($F_{3,182,15,2} = 5.173$, $P = 0.029$) wherein responses tended to be smaller in Neutral compared with Pronated postures in Shoulder Flexed ($t = -2.204$, $P = 0.05$) but not in Shoulder Abducted position ($t = -0.497$, $P = 0.629$). Separate analysis showed that responses in Hanging Neutral were 11% smaller than in Shoulder Abducted Neutral ($t = -5.24$, $P < 0.001$) and 13% smaller than in Shoulder Flexed Neutral ($t = -5.257$, $P < 0.001$).

Triceps brachii responses. Figure 4 shows an individual subject’s traces for triceps brachii MEPs, CMEPs, and $M_{\text{max}}$ in the seven arm postures. Triceps brachii MEPs were not affected by arm posture (Fig. 5A). There were no main effects for forearm orientation ($F_{2,10} = 2.917$, $P = 0.100$) or upper-arm orientation ($F_{1,5} = 0.152$, $P = 0.712$), and no interaction ($F_{2,10} = 0.085$, $P = 0.919$). Triceps brachii EMG RMS in the 100 ms preceding the MEP was generally not affected by arm posture, although there was an interaction between upper-arm and forearm orientation ($F_{2,10} = 4.151$, $P = 0.049$), which yielded no significant post hoc results.

Triceps brachii CMEPs were unaffected by forearm orientation but decreased when the arm hung to the side (Fig. 5B). ANOVA showed no main effect for forearm orientation ($F_{2,16} = 1.58$, $P = 0.237$) or upper-arm orientation ($F_{1,8} = 0.551$, $P = 0.479$), and no interaction ($F_{2,16} = 2.497$, $P = 0.114$). Separate analysis showed that responses in Hanging Neutral were 42% less than in Shoulder Abducted Neutral ($t = -4.354$, $P = 0.002$) and 46% less than in Shoulder Flexed Neutral ($t = -3.567$, $P = 0.007$). Triceps brachii EMG RMS in the 100 ms preceding the CMEP was not affected by arm posture.

Triceps brachii MEP/CMEP ratios were not affected by arm posture. There were no main effects for forearm orientation ($F_{2,8} = 0.619$, $P = 0.562$) or upper-arm orientation ($F_{1,4} = 0.511$, $P = 0.514$), and no interaction ($F_{2,8} = 1.261$, $P = 0.334$). The ratio tended to be larger in Hanging Neutral (2.94 ± 2.55) compared with Shoulder Flexed Neutral (1.36 ± 0.67) and Shoulder Abducted Neutral (1.88 ± 2.19), but these differences were not statistically significant (both $P > 0.148$).

Triceps brachii $M_{\text{max}}$ was not affected by arm posture (Fig. 5C). There were no main effects for forearm orientation ($F_{2,22} = 1.267$, $P = 0.302$) or upper-arm orientation ($F_{1,11} = 0.150$, $P = 0.706$), and no interaction ($F_{2,22} = 3.151$, $P = 0.063$).

**DISCUSSION**

Our novel result is that changes in corticospinal excitability between different arm postures are largely spinal in origin. Sizes of biceps brachii MEPs are known to be influenced by arm posture (Mitsuhashi et al. 2007; Mogk et al. 2014; Peter-
son et al. 2014). In this study, we demonstrate that CMEPs in resting biceps brachii are also influenced by arm posture. More specifically, biceps brachii CMEPs are smallest when the arm hangs to the side or when the forearm is pronated and are largest when the upper arm is flexed at the shoulder or when the forearm is supinated. Triceps brachii CMEPs are smallest when the arm hangs to the side but are unaffected by forearm orientation.

Before the physiological mechanisms underlying arm posture-dependent changes in corticospinal excitability are considered, potential confounding methodological factors should be taken into account. Arm posture influenced biceps brachii \(M_{\text{max}}\). Biceps brachii \(M_{\text{max}}\) was smallest when the arm hung to the side or when the forearm was supinated. These differences may be attributed to changes in the spatial configuration of muscle fibers under the recording electrodes. Such changes may occur as a result of muscle lengthening or shortening in different postures (Frigon et al. 2007). Because the same electrode pair is used to record \(M_{\text{max}}\), MEPs, and CMEPs, changes in MEPs and CMEPs could simply represent a methodological artifact from the periphery. To avoid this problem, MEPs and CMEPs at a given posture were normalized to \(M_{\text{max}}\) in the same posture to account for postural changes in the compound muscle fiber action potential. When normalized to \(M_{\text{max}}\), MEPs and CMEPs still changed with arm posture, indicating that the observed changes were due to a central mechanism. A second methodological factor is that elbow angle was not standardized between the three different upper-arm orientations, because we were replicating postures that have been used in related work (Mogk et al. 2014; Nuzzo et al. 2016). Thus changes in excitability between the three upper-arm orientations reflect postural deviations at both the shoulder and elbow. Moreover, biceps and triceps brachii cross both the shoulder and elbow so that muscle lengths depend on both joints.

The identification of a spinal origin for this postural effect in humans is consistent with findings in anesthetized monkeys (Yaguchi et al. 2015). Using cervical spinal cord stimulation, Yaguchi et al. observed that sizes of evoked responses in arm muscles changed with shoulder and elbow angle, an effect that remained after spinal transection above the stimulation site. However, the specific spinal mechanisms underlying arm posture-dependent changes in corticospinal excitability are not revealed by our study. Use of the existing literature to identify mechanisms is difficult because measurements have been acquired with the muscle in a non-resting state (e.g., Barry et al. 2008). Moreover, if reflex inputs underlie the changes, these may well differ between motoneuron pools. The changes in CMEPs in biceps brachii are consistent with the actions of some reflex pathways, and those in triceps brachii are consistent with others.

For biceps brachii motoneurons, spinal reflex pathways from heteronymous muscles are one of the more likely mechanisms to explain the findings with regard to forearm posture. Both brachioradialis (Barry et al. 2008; Naito et al. 1996) and pronator teres (Naito et al. 1998) exert an inhibitory effect onto single motor units of biceps brachii during voluntary contraction. The effect from brachioradialis is greatest during forearm pronation (Barry et al. 2008), and this is consistent with our observation that biceps brachii CMEPs decrease in pronation. This pathway is thought to involve group I afferents from brachioradialis synapsing on inhibitory interneuronal inputs to the biceps brachii motoneuron pool. Variation in the excitability of the inhibitory interneuronal connections rather than variation in Ia firing is likely to account for altered strength of the reflex across forearm postures, but the mechanisms responsible for this are not known. Moreover, as all previous studies of the reflex have been undertaken during voluntary contraction, it is unclear to what extent it acts during rest.

Other spinal pathways can be excluded as major mechanisms influencing biceps brachii motoneuron excitability. The observed effects cannot be attributed to facilitation from homonymous group Ia muscle afferents, which are most active at long muscle lengths (Burke et al. 1978), because biceps brachii MEPs and CMEPs were smallest at long muscle lengths (i.e., Hanging Neutral) and largest at short lengths (i.e., Shoulder Flexed Supinated). This behavior also excludes reciprocal inhibition. However, it is consistent with previous reports that MEPs in various arm muscles are largest at short muscle lengths (Lewis et al. 2001; Renner et al. 2006), as are soleus H-reflexes (Gerilovsky et al. 1989; Hwang 2002). Alterations in H-reflexes after passive changes in muscle length have been attributed to homosynaptic postactivation depression of the Ia afferents (Hultborn et al. 1996). This mechanism cannot explain our results, because MEPs and CMEPs access the motoneurons via descending paths.

For triceps brachii motoneurons, forearm posture (pronation, supination) had no effect on CMEPs, despite the strong influence on biceps brachii, but with changes in shoulder posture, triceps and biceps brachii CMEPs changed in the same direction. Both responses were smallest in Hanging Neutral, which is the posture where triceps brachii was shortest and biceps
brachii longest. Thus, for the triceps brachii motoneuron pool, the change in CMEPs agrees with the predicted actions of homonymous Ia facilitation and reciprocal inhibition, although it is not in accordance with previous results of increased corticospinal excitability to this muscle when it was shortened through changing elbow angle (Renner et al. 2006). Comparison of biceps and triceps responses suggests that excitability of motoneurons depends on a different mix of afferent input for the two muscles. Alternatively, postural changes in muscle afferent input may have little influence on either motoneuron pool.

Afferent pathways associated with skin and joint receptors also act on motoneurons. Skin afferents from the fingers can inhibit biceps brachii via propriospinal neurons (Nielsen and Pierrot-Deseilligny 1991). Skin contact was not directly controlled in the current study. In the Shoulder Flexed and Shoulder Abducted postures, skin from the forearm and hand contacted the experimental setup, whereas in Hanging Neutral, when sizes of biceps and triceps CMEPs were smallest, there was no skin contact with the setup. Afferents within the shoulder capsule (including the glenohumeral ligament) can inhibit the long head of biceps brachii (Voigt et al. 1998). This makes them potential contributors to the effect of upper-arm orientation on corticospinal excitability. However, these joint receptors are probably most active at the limits of range of motion (Burke et al. 1988), and the shoulder was not near end of range in any of the postures in the current study.

Whereas spinal mechanisms underlie the changes in CMEPs, we cannot rule out an influence on MEPs from additional cortical mechanisms. The MEP/CMEP ratios calculated for biceps and triceps responses tended to be larger in Hanging Neutral compared with Shoulder Flexed Neutral and Shoulder Abducted Neutral. Differential changes in cortically and spinally evoked responses imply different effects at the cortex and spinal cord for some postures. In this study, MEPs could be influenced by either facilitation at the cortex when the arm hangs to the side or inhibition when the shoulder is flexed or abducted. Previously, intracortical facilitation, but not inhibition, has been observed to play a role in corticospinal excitability to resting arm muscles when upper-arm orientation is manipulated (Ginanneschi et al. 2005, 2006). In addition, the relationship that exists between muscle length and biceps brachii MEPs in healthy controls is abolished after cortical stroke, but not subcortical stroke (Renner et al. 2006). Therefore, we conclude that the observed arm posture-dependent changes in corticospinal excitability are largely spinal in origin, but cortical processes may also contribute.

In conclusion, forearm and upper-arm orientation influence sizes of biceps brachii MEPs and CMEPs. The changes in CMEPs indicate that the mechanisms underlying arm posture-dependent changes in corticospinal excitability are largely spinal in origin. Multiple pathways likely contribute to this effect by altering the balance of inhibitory and excitatory inputs to the motoneurons.

GRANTS

This work was supported by the National Health and Medical Research Council of Australia. J. L. Nuzzo is supported by a University of New South Wales International Postgraduate Research Scholarship and a Neuroscience Research Australia Supplementary Scholarship. G. S. Trajano is supported by The National Council for Scientific and Technological Development (CNPq) Brazil.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

REFERENCES


