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RESEARCH ARTICLE

Short-interval intracortical inhibition to the biceps brachii is present during arm cycling but is not different than a position- and intensity-matched tonic contraction

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Abstract

We have previously shown that supraspinal excitability is higher during arm cycling than a position- and intensity-matched tonic contraction. The present study sought to determine if short-interval intracortical inhibition (SICI) was present during arm cycling and if so, if the amount of SICI was different from an intensity-matched tonic contraction. SICI was assessed using conditioning stimuli (CS) of 70 and 90% of active motor threshold (AMT) and a test stimulus (TS) of 120% AMT at an interstimulus interval (ISI) of 2.5 ms. SICI was elicited in all participants; on average (i.e., cycling and tonic contraction grouped) test MEP amplitudes were reduced by 64.2% ($p < 0.001$) and 62.8% ($p = 0.001$) following conditioning stimuli of 70% and 90% AMT, respectively. There was no significant difference in extent of SICI between tasks ($p = 0.360$). These data represent the novel finding that SICI is present during arm cycling, a motor output partially mediated by spinal interneuronal networks. The amount of SICI, however, was not different from that during a position- and intensity-matched tonic contraction, suggesting that SICI is not likely a cortical mechanism contributing to higher supraspinal excitability during arm cycling compared to tonic contraction.

Keywords Paired-pulse TMS · Cortical · Task-dependent · Arm cranking · Pedaling

Introduction

The basic pattern of rhythmic motor outputs, such as locomotion and cycling, is partially generated by spinal circuits known as central pattern generators (CPGs) (Grillner 1981; Zehr 2005; Hultborn and Nielsen 2007). Descending input arising from the motor cortex contributes to human locomotor outputs such as locomotion (Capaday et al. 1999; Christensen et al. 2001; Petersen et al. 2001; Barthelemy and Nielsen 2010; Barthelemy et al. 2011), leg cycling (Christensen et al. 2000; Pyndt and Nielsen 2003; Sidhu et al. 2012) and arm cycling (Forman et al. 2014, 2015, 2016a; Spence et al. 2016; Lockyer et al. 2018; Power et al. 2018).

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The modulation of human cortical excitability by various local circuits, such as short-interval intracortical inhibition (SICI), can be assessed through the use of paired-pulse transcranial magnetic stimulation (ppTMS). To assess SICI using ppTMS, a subthreshold conditioning stimulus (CS), which activates cortical inhibitory neurones, is administered approximately 1–5 ms prior to a suprathreshold test stimulus (TS), which indirectly activates cortical pyramidal neurones (Di Lazzaro et al. 2005; Rossini et al. 2015). The resulting motor-evoked potential (MEP) amplitude is compared to the MEP elicited via the TS in the absence of a CS. If a decrease in MEP amplitude is observed with the CS present, SICI is thought to be occurring and likely represents postsynaptic inhibition mediated by $GABA_A$ receptors (Rossini et al. 2015).

Only two studies have assessed SICI during locomotor output (Barthelemy and Nielsen 2010; Sidhu et al. 2013). Barthelemy and Nielsen (2010) showed that SICI in the posterior deltoid was enhanced when muscle activity was low and reduced when muscle activity was high. In a more recent study, Sidhu and colleagues assessed SICI projecting to the knee extensor muscles during leg cycling using four different

CS intensities [70, 80, 90 and 95% of active motor threshold (AMT)]. Similar to Barthelemy and Nielsen (2010), SICI was reduced during muscle activation and enhanced during deactivation, independent of CS intensity. Importantly, SICI also occurred during a tonic contraction of the same muscle; however, the two motor outputs (cycling and tonic contraction) were not directly compared. Thus, it is unclear whether the degree of SICI was task dependent.

We recently demonstrated that TMS-evoked MEPs to the biceps brachii were larger during the elbow flexion phase of arm cycling than during position- and intensity-matched tonic contractions, indicating a higher level of corticospinal excitability during arm cycling. Cervicomedullary motor-evoked potentials (CMEPs) elicited through transmastoid electrical stimulation (TMES) of the corticospinal tract were not different, suggesting that supraspinal excitability was higher during arm cycling than the tonic contraction (Forman et al. 2014, 2016b). The mechanism(s) responsible for this task-dependent difference in supraspinal excitability was(were) not examined. Importantly, SICI has been previously shown to be both state- and task dependent (Kujirai et al. 1993; Ortu et al. 2008; Opie et al. 2015; Hunter et al. 2016). Thus, it is possible that the higher supraspinal excitability we observed during arm cycling as compared to tonic contraction was due to a reduction in the activation of the SICI circuity, thus allowing greater corticospinal activation.

The purpose of the current study was to determine if: (1) SICI was present during arm cycling and (2) the amount of SICI between arm cycling and a tonic contraction was different. Provided SICI was present during arm cycling, a secondary objective was to determine whether CS intensity differentially modulated the degree of SICI. We hypothesized that SICI would be present during arm cycling, that the amount of SICI would be less during arm cycling compared to a tonic contraction and that CS intensity would not affect the degree of SICI present.

Methods

Participants

Twelve healthy participants (10 males and 2 females) between the ages of 20 and 37 participated in all aspects of this study. All participants completed a magnetic stimulation safety checklist (Rossini et al. 2015), a Physical Activity Readiness Questionnaire [PAR-Q+; Canadian Society for Exercise Physiology (CSEP)] (Bredin et al. 2013), and an Edinburg handedness questionnaire to identify the dominant limb for testing (Veale 2014). Participants had no known neurological impairments. The procedure was verbally explained to the participants and written consent was obtained prior to starting the study. The experimental procedure conformed to the Helsinki declaration and was approved by the Interdisciplinary Committee on Ethics in Human Research at Memorial University of Newfoundland (ICEHR no. 20161507-HK). Procedures were in accordance with the Tri-Council guideline in Canada, with potential risks explained to participants.

Experimental set-up

EMG recordings

Electromyography recordings were taken from the dominant biceps and triceps brachii using pairs of surface electrodes (Medi-Trace 130 ECG conductive adhesive electrodes) in bipolar configuration (Ag–AgCl, 2-cm interelectrode distance). Electrodes were placed over the midline of the biceps and triceps brachii (lateral head) muscles. Prior to electrode placement, the skin was thoroughly prepared through shaving, abrading, and cleaning with alcohol swabs to reduce EMG recording impedance. An additional ground electrode was placed over the lateral epicondyle. EMG data were collected online at 5 kHz using CED 1401 interface and Signal 4 software program [Cambridge Electronic Design (CED), Cambridge, UK]. Signals were amplified and filtered using a three-pole Butterworth with cut-off frequencies of 10–1000 Hz (Fig. 1).

Cycle ergometer set-up

Participants were in a seated position to perform arm cycling and tonic contraction tasks using an arm cycle ergometer (SCIFIT ergometer, model PRO2 Total Body). It was ensured that participants were in a comfortable position and at a distance from the arm cranks such that they were not leaning forward or backward and were maintaining upright trunk posture. The arm cranks on the bike were fixed 180° out of phase. Forearms were in the pronated position and stabilized with wrist braces to reduce interference from joint movement and subsequent heteronymous reflex connections between the wrist flexors and extensors and the biceps brachii (Manning and Bawa 2011).

Crank position was made relative to a clock face with respect to the arm from which the recordings were made (dominant arm; 12, 3, 6, and 9 o'clock), with 'bottom dead centre' as the 6 o'clock position. The biceps brachii was the main muscle of interest; therefore, cycling movement was defined hereafter with reference to the dominant elbow joint position. Thus, elbow flexion was defined as the movement from the 3- to the 9 o'clock position (Fig. 2). Cycling throughout the experiment was fixed at a workload of 25 W and at a cadence of 60 rpm as per our previous work upon which the current study is based (Forman et al. 2014).

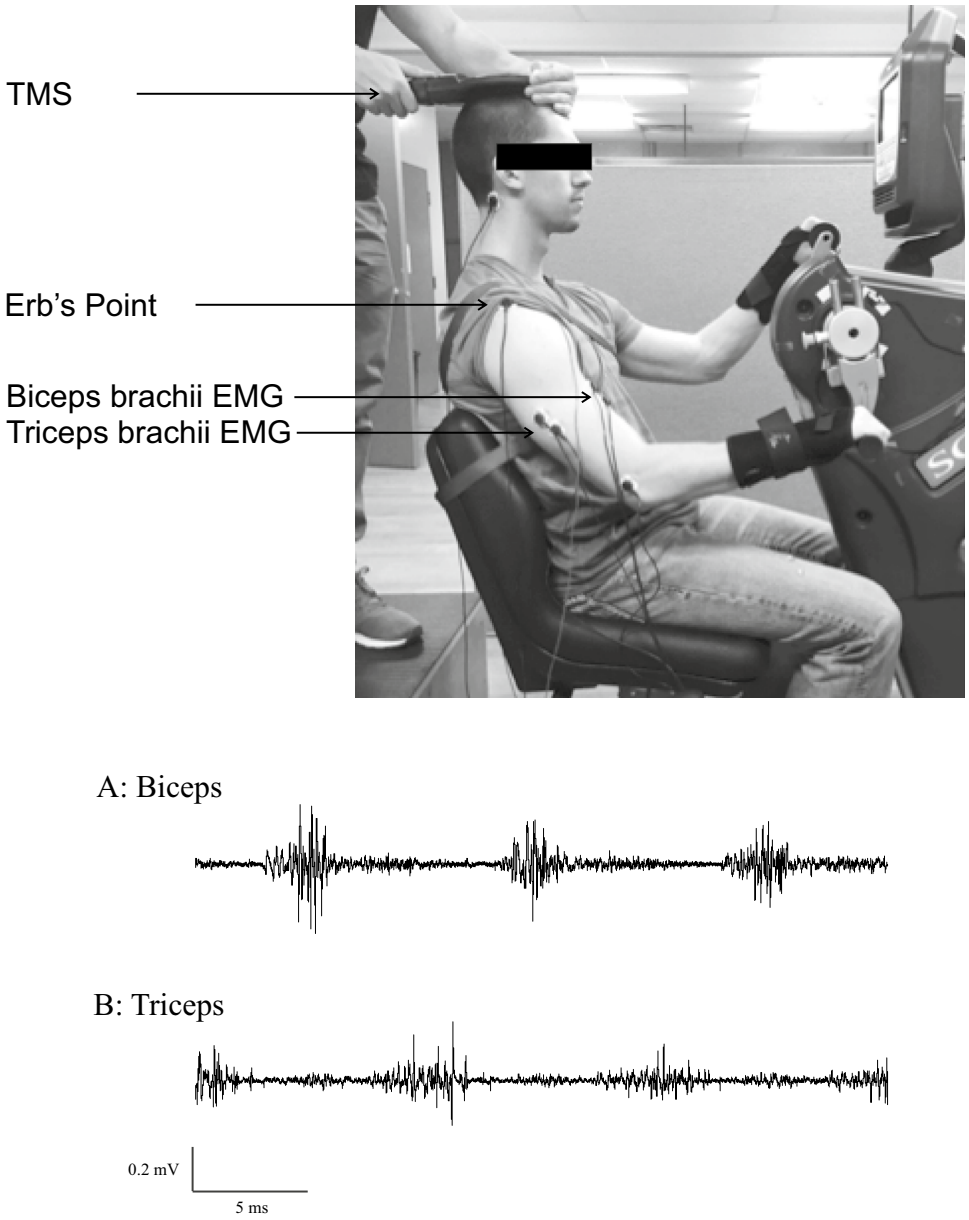

Fig. 1 Experimental design with participant at arm cycle ergometer. Labels indicate TMS paddle, Erb's point stimulating electrodes, biceps and triceps brachii EMG recording electrodes. EMG trace shows bursts from biceps brachii (**a**) and triceps brachii (**b**), with an arrow indicating approximate point of stimulation during mid-flexion of the elbow joint

Stimulation conditions

Motor responses recorded from the biceps brachii were elicited via (1) single-pulse TMS, (2) paired-pulse TMS (i.e., ppTMS) and (3) brachial plexus electrical stimulation at Erb's point. During cycling, stimulation intensities were determined in reference to the dominant arm crank position and were automatically triggered as the arm crank passed the 6 o'clock position, the mid-flexion point of the dominant biceps brachii. During tonic contraction, stimulations were triggered with the dominant arm crank fixed at the 6 o'clock position as we have done previously (Forman et al. 2014; Copithorne et al. 2015).

Transcranial magnetic stimulation

Motor-evoked potentials (MEPs) from the dominant arm biceps brachii were elicited during cycling and intensity-matched tonic contractions using a circular coil (13.5 cm outside diameter) attached to a BiStim module connected to two magnetic stimulators (Magstim 200, Dyfed, United Kingdom). The coil was held 1 cm lateral to vertex, parallel to the floor with direction of current flow preferentially activating the dominant motor cortex. Vertex was located by measuring nasion to inion and tragus to tragus; marking the location on the scalp halfway between them; and defining vertex as the intersection of the halfway marks (Forman

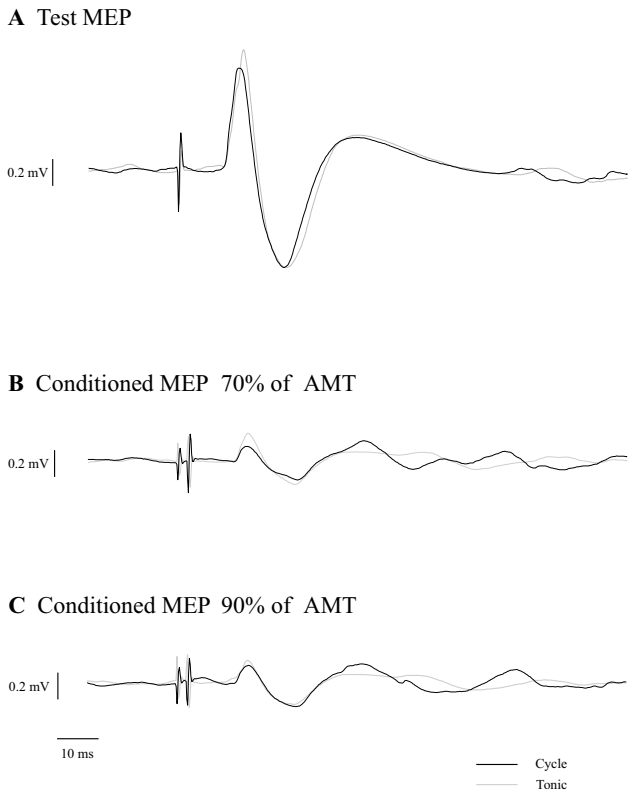

Fig. 2 Average test (**a**), conditioned using 70% of AMT (**b**), and conditioned using 90% of AMT (**c**) MEP traces after receiving stimuli during arm cycling (black lines) and tonic contraction (gray lines) from one participant ($n = 1$)

et al. 2014; Copithorne et al. 2015). Stimulation was delivered every 7 s. During tonic contraction trials, participants relaxed between stimulations.

Active motor threshold

Active motor threshold (AMT) was defined as the minimum intensity at which a MEP was clearly discernible from the background EMG (bEMG) in 50% of the trials (i.e., 8/16 during cycling and tonic contraction) (Sidhu et al. 2013; Forman et al. 2018). AMT recorded from the biceps brachii was determined using TMS in two separate conditions, (1) while participants cycled at 25 W and 60 rpm and (2) while participants completed intensity-matched tonic contractions.

Test and conditioning stimulus intensity

Test stimulus (TS) intensity was defined as a suprathreshold TMS of approximately 120% AMT. For cycling trials, TS was set as 120% of cycling AMT. For tonic contraction trials, TS was set as ~ 120% such that the MEP amplitude was matched to the average TS MEP amplitude recording during cycling. This was to ensure that SICI measurements were not affected by test MEP size-dependent differences (Sidhu et al. 2013). Conditioning stimulus (CS) intensities of 70% and 90% of AMT (Sidhu et al. 2013) were investigated during both cycling and tonic contraction trials. In all conditioned stimulations, the TS was preceded by a CS at an ISI of 2.5 ms, based on a previous study which indicated that an ISI of 2.5 ms was optimal to activate inhibitory interneurons in the motor cortex (Ortu et al. 2008). All TS and CS stimulation intensities during cycling and tonic contractions were percentages of their respective AMT values.

Brachial plexus stimulation

Stimulating electrodes were placed at Erb's point (pulse duration of 200 µs), with the cathode in the supraclavicular fossa and the anode over the acromion process, to stimulate the brachial plexus (DS7AH, Digitimer, Welwyn Garden City, Hertfordshire, UK). Resting M_{max} of the biceps brachii was determined by increasing stimulation intensity until the M-wave reached a plateau. This stimulation intensity was then increased by 20% to ensure maximal M-waves (i.e., M_{max} stimulations) were elicited throughout the study. M_{max} stimulations were elicited once during each cycling and tonic contraction condition as a measure of peripheral neuromuscular excitability.

Experimental protocol

Active motor threshold was first established during cycling as noted above. The bEMG from the cycling period was then used to complete intensity-matched tonic contractions as we have previously done (Forman et al. 2014, 2016b). bEMG activity was found by averaging the rectified amplitude of the 50-ms window preceding stimulation during previous cycling trials and maximum EMG burst at the 6 o'clock position. Participants then performed tonic elbow flexion contractions that were intensity matched to arm cycling based on the bEMG. With the arm crank fixed at the 6 o'clock position, participants were provided with visual feedback of a horizontal line on a computer screen equal to the averaged bEMG level during cycling. AMT was found during tonic contractions in the same manner as the cycling trials, with stimulation administered at increasing intensities until a visible MEP was found as noted above.

Following the determination of AMT, participants cycled for approximately 1 min during which they received 10 control TS at 120% AMT and one M_{max} in a random order at approximately 7 s intervals (i.e., one stimulation every seven cycles). TS intensity (~ 120% AMT) for tonic contractions was determined by eliciting a MEP during tonic contraction that was size matched to the average MEP response from the previous cycling TS control trial. Participants then completed 13 tonic contraction trials during which they

received 12 control TS (~ 120% AMT) and one M_{max} in a random order at 7 s intervals. Participants briefly relaxed for approximately 4 s following the stimulation and then contracted again prior to the next stimulation. Participants were always given two extra stimulations during tonic trials (i.e., 12 during tonic contraction and 10 during cycling).

Next, participants cycled for approximately 2 min, during which they received the following conditioned stimulations in a random order: 10 ppTMS stimuli with CS and TS set at 70 and 120% AMT, respectively; 10 ppTMS stimuli with CS and TS set at 90 and 120% of AMT, respectively; and two M_{max}. Participants then performed 2 min of intensity-matched tonic contractions during which the participant repeated a sequence of contractions that involved active contraction for approximately 2 s followed by approximately 4 s of relaxation. During tonic contractions, they received the following stimulations in a random order: 12 ppTMS stimuli with CS and TS set at 70 and ~ 120% AMT, respectively; 12 ppTMS stimuli with CS and TS set at 90 and ~ 120% AMT, respectively; and two M_{max}.

A cycling trial with 16 stimulations at original AMT intensity was performed again upon completion of the experimental protocol. MEP amplitudes elicited via AMT stimulation intensity were then compared between pre- and post-protocol to determine whether any changes in AMT occurred throughout the experiment, which would affect the relative percent intensity of the CS. Similar comparisons were made for the tonic contraction trials.

Data analysis

For analysis of MEPs and M_{max}, the averaged peak-to-peak amplitudes from each cycling trial were measured from the biceps brachii of the dominant arm. Because MEPs evoked via ppTMS (CS) were compared to those during the single-pulse TMS (TS), MEPs were made relative to each other as opposed to M_{max} as is normally done with single-pulse TMS studies. M_{max} was instead used to assess peripheral excitability and to ensure it was similar between tasks. Additionally, since the level of voluntary muscle contraction can influence MEP amplitudes, pre-stimulus EMG was measured from the rectified virtual channel created for the biceps and triceps brachii as the mean of a 50-ms window immediately prior to the stimulation artifact (Forman et al. 2015).

Statistical analysis

SICI is presented as a ratio of conditioned MEP amplitude over test MEP amplitude. The ratio is then multiplied by 100 to give the amplitude of the conditioned MEP as a percentage of the test MEP amplitude. All statistics were performed using IBM's SPSS Statistics (IBM SPSS Statistics for Windows, Version 23.0. Armonk, NY: IBM Corp.). Mauchly's test was employed to assess the assumption of sphericity for repeated measures analysis. In cases where sphericity was violated, the Greenhouse–Geisser correction was applied and the degrees of freedom were adjusted. Two-way (task × stimulation intensity) repeated-measures ANOVAs were used to determine whether statistically significant differences in SICI or bEMG were present. To determine whether MEP amplitudes elicited via AMT stimulation intensities changed over the course of the experiment (pre- to post-protocol), separate paired-sample t tests were performed for cycling and tonic conditions. M_{max} was assessed using a paired t test to compare amplitudes between cycling and tonic trials. All statistical analyses were performed on group data, and a significance level of $p < 0.05$ was used. Data are presented and shown in figures as mean ± SD.

Results

Active motor threshold

Test and conditioning stimulation intensities (and the MEP amplitudes they elicit) used throughout the experiment are a percentage of the AMT; therefore, it is important to replicate the AMT trial following the protocol to know if it changed throughout. MEPs elicited at AMT stimulation intensities (same stimulation intensity used pre- and post-protocol) were not significantly different for the cycling or tonic tasks ($p = 0.753$ and $p = 0.755$, respectively; see Table 1) following the experimental protocol.

Stimulation intensities

Test stimulus intensities ranged from 32 to 68% of maximum stimulator output (MSO). There was no main effect of task ($p = 0.113$), nor was there an interaction effect between task and stimulation intensity ($p = 0.934$) indicating that stimulator output intensity was comparable between tasks within conditions (test and conditioning stimulation intensities; see Table 2). MEP amplitudes elicited via the TS intensity for both tasks were not significantly different, thus allowing a direct comparison between tasks [see Table 2; TS MEPs

Table 1 AMT MEP amplitudes immediately pre- and post-protocol for cycling and tonic tasks ($n = 12$)

	Cycle	Tonic
AMT		
Pre (mV)	1.13 ± 0.58	0.76 ± 0.41
Post (mV)	1.10 ± 0.53	0.78 ± 0.41

Values are in mean ± SD

Table 2 Average percent MSO used throughout the experiment for each task and condition ($n = 12$)

Condition	Cycle	Tonic
TS ~ 120%	46.4 ± 10.0	48.7 ± 10.5
MEP amplitude	2.2 ± 1.2	2.1 ± 1.2
CS 70%	27.0 ± 5.7	29.2 ± 7.9
CS 90%	34.9 ± 7.6	37.5 ± 10.2

Average MEP amplitude (mV) elicited using the test stimulus intensity (TS ~ 120%) is shown for cycling and tonic tasks

Values are in mean ± SD

(mV): cycling, 2.2 ± 1.2 vs tonic, 2.1 ± 1.2; paired *t* test, $p = 0.273$].

SICI

Figure 2 shows an example from a single participant of both test and conditioned MEPs during cycling and tonic tasks using conditioning stimulation intensities of 70% and 90% of AMT. In this example, MEP amplitudes were reduced during cycling from 3.0 mV (Fig. 2a) to 0.5 mV (Fig. 2b) and 0.6 mV (Fig. 2c), a reduction of 83% and 80%, respectively. During tonic contraction, MEP amplitudes were reduced from 3.3 mV (Fig. 2a) to 0.8 mV (Fig. 2b) and 0.7 mV (Fig. 2c), a reduction of 76% and 79%, respectively.

The SICI ratio shows the size of the conditioned MEP as a percentage of the test MEP. Therefore, a value below 100% shows MEP amplitude reduction and a value above 100% would show an increase in MEP amplitude. As a group, SICI was evident in both tasks using both CS intensities (Fig. 3). There were no main effects for 'task' ($p = 0.360$) or 'stimulation intensity' ($p = 0.301$), nor was there an interaction effect between task and stimulation intensity ($p = 0.181$).

Background EMG

Group data for bEMG of the biceps and triceps brachii can be seen in Fig. 4a, b. For the biceps brachii, there were no main effects for task ($p = 0.134$) or stimulation intensity ($p = 0.744$), nor was there an interaction effect between task and stimulation intensity ($p = 0.238$). For the triceps brachii, there were no main effects for task ($p = 0.142$) or stimulation intensity ($p = 0.578$), nor was there an interaction effect between them ($p = 0.980$). Mauchly's test of Sphericity was violated and the Greenhouse–Geisser correction was applied to both biceps and triceps brachii data.

M_{max}

There was no significant difference in M_{max} amplitudes between tasks as recorded from the biceps brachii ($p = 0.413$: cycling 11.0 ± 4.4 mV and tonic 10.5 ± 4.1 mV).

Discussion

Main findings

The two main objectives of this study were to determine whether SICI was present during arm cycling and if so,

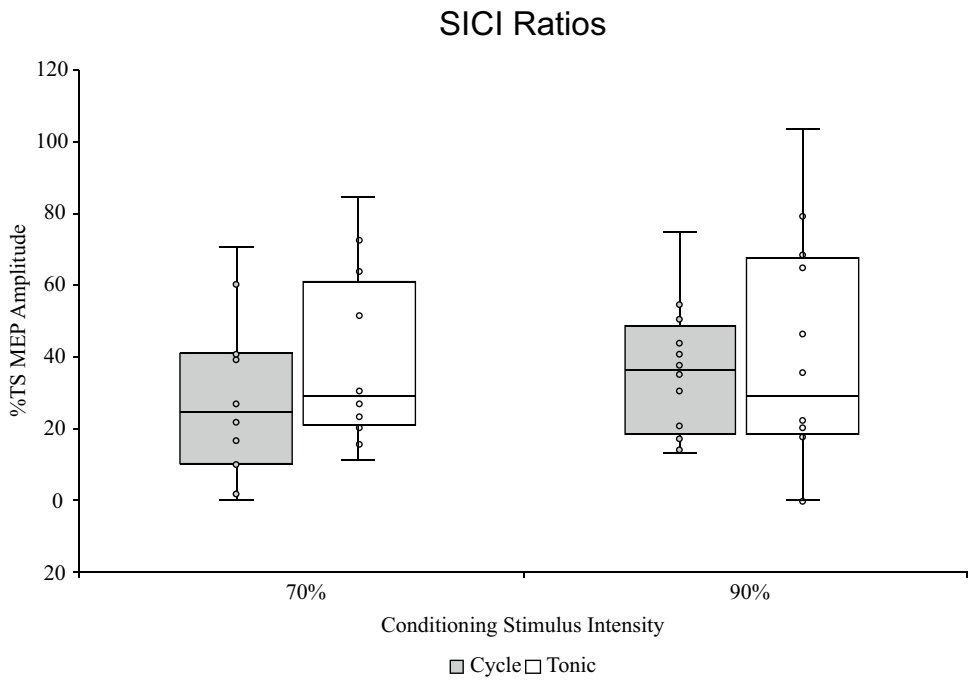

Fig. 3 Conditioned MEP amplitudes as a percentage of the test MEP response in the biceps brachii for both cycling and tonic tasks ($n = 12$). No significant differences between conditions

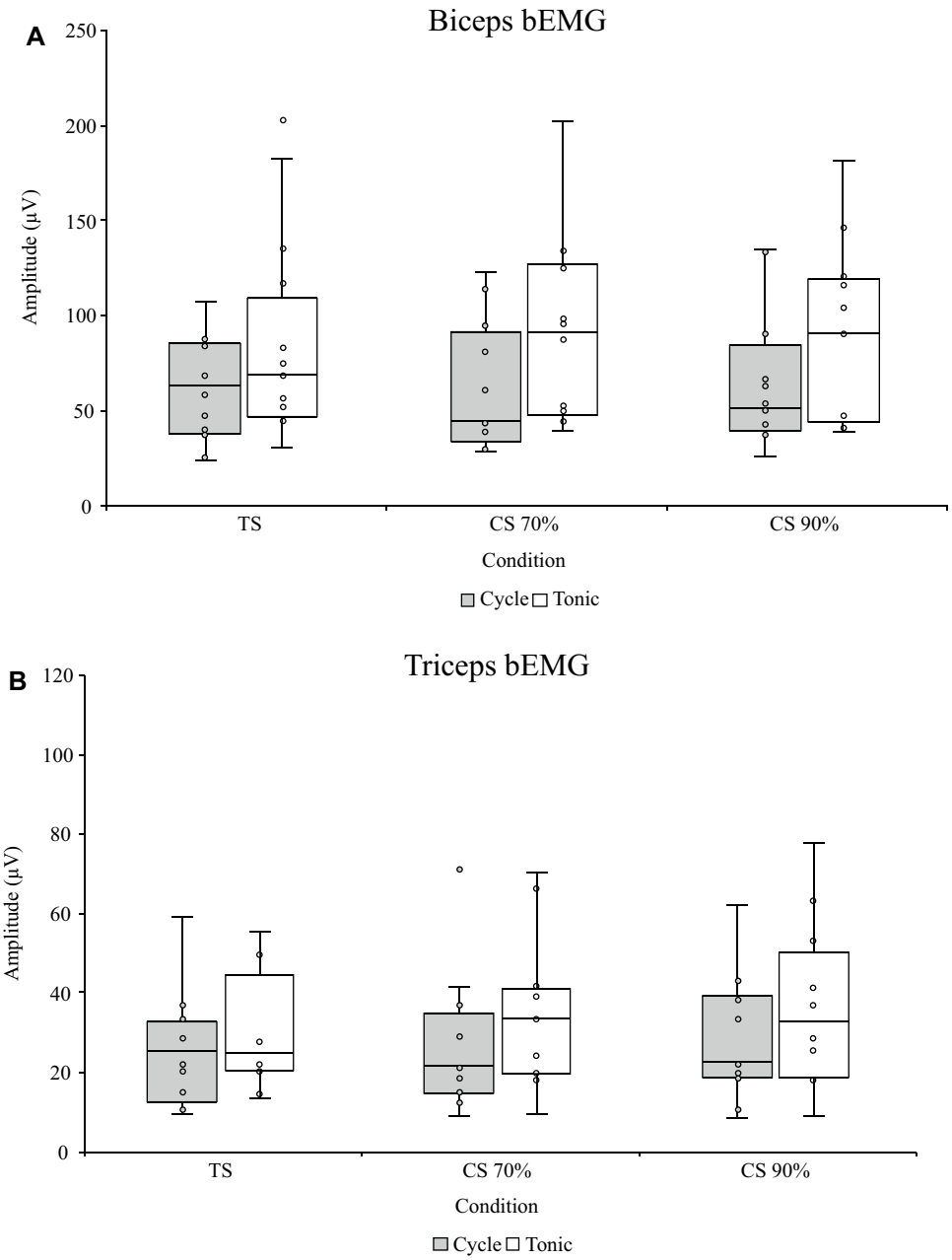

Fig. 4 Background EMG during cycling and tonic contractions for all conditions in the biceps brachii (**a**) and triceps brachii (**b**). No significant differences between conditions ($n = 12$)

was the amount of SICI different than a position- and intensity-matched tonic contraction. This is the first report to show that SICI is present during arm cycling; however, the amount of SICI was not different from that of an intensity-matched tonic contraction. A secondary objective was to determine whether CS intensity (70 vs 90% AMT) influenced the degree of SICI during arm cycling and tonic contraction. Here, we show that both CS intensities are equally effective in eliciting SICI during arm cycling.

SICI was present during arm cycling but was not task dependent

We have previously shown that corticospinal excitability to the biceps brachii, as assessed via TMS-elicited MEPs (single-pulse), was higher at the mid-flexion point of arm cycling when compared to a position- and intensity-matched tonic contraction (Forman et al. 2014). Given that spinal excitability as assessed via CMEPs was not different between the

two motor outputs, we suggested that changes in corticospinal excitability were of supraspinal origin, though the exact mechanism(s) was(were) unknown (Forman et al. 2014). In the present study, we sought to determine if changes in SICI could partially account for this task-dependent difference in supraspinal excitability. Using 70 and 90% CS intensities, here we show that SICI was present during arm cycling as shown via 67 and 64% reductions in TS MEP amplitude (Fig. 3). Though present during arm cycling, SICI was not different than the tonic contraction (Fig. 3). Task-dependent differences in SICI have been previously reported, albeit not during locomotor output (Opie et al. 2015).

Opie et al. (2015) demonstrated that SICI was lower during a gripping task as compared to a finger abduction task. Though both were tonic contractions, the gripping task had higher levels of synergistic muscle activation which likely involves greater cortical activation to activate more muscles. It is worth noting that arm cycling is a bilateral, dynamic motor output and likely involves the activation of greater cortical regions to activate more muscles, similar to that of the gripping task used by Opie et al. (2015). Though SICI was not larger in the cycling task, it is noted that the influence of transcortical and/or different intracortical circuits (Rossini et al. 2015) during locomotor output and how they would interact with SICI are currently unknown.

SICI during locomotor output

This is only the third study to examine SICI during locomotor output. Sidhu et al. (2013) measured changes in SICI as recorded from the knee extensors during the 'activation' (i.e., during ascending EMG activity) and 'deactivation' (i.e., during descending EMG activity) phases of leg cycling. They found that SICI was reduced during activation and enhanced during deactivation and tonic contraction of the knee extensors. Reduction of SICI during the activation phase may allow greater cortical neuronal excitation, and thus enhanced descending input via the corticospinal tract to the motoneurone pools to ensure adequate muscle activation. The reverse would seem reasonable during the deactivation phase. This is similar to the modulation of SICI in the posterior deltoid muscle during human locomotion (Barthelemy and Nielsen 2010), namely that SICI and bEMG were inversely related. Unlike Sidhu et al. (2013), we assessed SICI based on position, not EMG level, in an attempt to gain a better understanding of our prior work (Forman et al. 2014). Though the EMG activity of the biceps brachii was relatively high when SICI was assessed (Fig. 1), the pattern of EMG during arm cycling can be variable between cycles within an individual and certainly between individuals. Thus, it is unclear whether SICI in the present study was assessed as the muscle was activating or at the peak of activation and transitioning to deactivation, which may prove important given the findings of Sidhu et al. (2013). Regardless, the current finding that SICI was not task dependent is an important step in understanding potential task-dependent differences in cortical excitability.

Are there muscle-dependent differences in SICI during locomotor output?

Sidhu et al. (2013) suggested that SICI in the knee extensors was weaker than that shown by Barthelemy and Nielsen (2010) in the posterior deltoid and suggested the presence of muscle-dependent differences in the amount of intracortical inhibition during locomotion. This is plausible given the task-, position-, intensity- and muscle-dependent differences in corticospinal excitability during locomotor output (Power et al. 2018). Interestingly, the studies by Sidhu et al. (2013) and Barthelemy and Nielsen (2010) examined extensors muscles while we examined a flexor muscle. There are well-known differences in extensor and flexor motoneurone properties that have been shown using both non-human animal (Cotel et al. 2009) and human models (Wilson et al. 2015) which suggest that cortical control may also be different and perhaps greater for flexor muscles (Power et al. 2018). If greater cortical input is required to activate flexor muscles during locomotor output, it may be that SICI would be lower for flexor than extensor muscles to allow greater descending excitatory input. Though an interesting possibility, muscle-dependent differences in SICI during locomotor output remains to be explored.

Methodological considerations

There are several factors that must be considered in the interpretation of the present results.

First, we state that SICI is not task dependent. It is noted, however, that this was at a single workload and set cadence. The extent of SICI has been shown to be intensity dependent during tonic tasks (Ortu et al. 2008) and that the amount of cortical activation may play a role in the amount of SICI observed (Opie et al. 2015). Because cycling is bilateral and likely involves greater overall cortical excitation due to the numerous active muscles, it is possible that task-dependent differences may become evident as the intensity of the motor outputs increases. This remains to be examined. Second, the sample size ($n = 12$) in the present study was not determined using a power calculation. With this in mind, although SICI was not task dependent, there was a trend for SICI to be higher during arm cycling, the opposite of our hypothesis. It is unclear whether a larger sample size would influence the present results. Finally, SICI is only one of the several cortical circuits which can be assessed using ppTMS. These excitatory and inhibitory circuits, however, are known to interact during tonic motor output (Rossini et al. 2015). If

or how these circuits interact during locomotor output and whether any interaction is different than a tonic contraction have not been examined and the implications for locomotor output are, thus, unclear.

Conclusion

In this study, we showed that SICI was present during arm cycling but was not different than a position- and intensity-matched tonic contraction. We also showed that 70% and 90% AMT CS intensities were both similarly effective in eliciting SICI to the biceps brachii during arm cycling. Further research is required to determine the influence of different cortical circuits on supraspinal excitability during locomotor activity.

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Author contributions LRA, DCB, and KEP conception and design of research; LRA, AS, and EJL performed experiments; all authors interpreted results of experiments; LRA analyzed data; LRA prepared figures; LRA and KEP drafted manuscript; All authors approved final version of manuscript.

Compliance with ethical standards

Conflict of interest No conflict of interest, either financial or otherwise, is declared by the authors.

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