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Intracortical Inhibition Assessed with Paired-Pulse Transcranial Magnetic Stimulation is Modulated during Shortening and Lengthening Contractions in Young and Old Adults

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ABSTRACT

Background: The modulation of intracortical inhibition is thought to be impaired in older adults, which may contribute to their reduced fine motor control, particularly during lengthening muscle contractions.
Objective: To quantify the magnitude of intracortical inhibition and movement performance during postural, shortening and lengthening contractions of a hand muscle in young and old adults.
Methods: In 18 young (23.2 ± 4.2) and 16 old (70.6 ± 6.5) subjects, paired-pulse transcranial magnetic stimulation (TMS) was used to assess short-interval intracortical inhibition (SICI) and long-interval intracortical inhibition (LICI) during a movement task involving the first dorsal interosseous muscle. The task required a constant load (50 g) to be slowly lifted and lowered using the index finger while single- or paired-pulse TMS was delivered during the shortening or lengthening contraction.
Results: Relative to postural contractions, SICI during shortening contractions was reduced by 29% in young subjects ($P < 0.0001$) and 43% in old subjects ($P < 0.0001$), whereas SICI during lengthening contractions was reduced by 11% in young subjects ($P = 0.0004$) and 33% in old subjects ($P < 0.0001$). Furthermore, SICI was significantly less in older adults during lengthening contractions (P-values < 0.01). For LICI, inhibition was not influenced by contraction type in old subjects, but was increased by 11% during shortening contractions ($P < 0.0001$) and by 9% during lengthening contractions in young subjects ($P = 0.0008$). In addition, old subjects showed significantly less LICI than young subjects in each movement phase (both P-values < 0.05).
Conclusions: Shortening and lengthening contractions with a constant load are associated with a modulation of GABAergic inhibition that is altered by healthy ageing.
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Introduction

A growing body of evidence suggests that the neural control of lengthening contractions represents a unique component of movement control. This includes observations that voluntary activation, electromyography (EMG), force generation and spinal motoneuron excitability are all different during lengthening contractions [1,2]. Furthermore, recent evidence from studies using a range of neurophysiological and neuroimaging techniques have provided compelling support for distinct patterns of cortical activity during lengthening contractions [3–11]. In addition, lengthening contractions are also associated with reduced motor performance [12–14]. Interestingly, the magnitude of this deficit is thought to be increased by advancing age, with greater impairments in performance observed in old adults during lengthening movements [13,15–18], which may contribute to the increased incidence of falls in the elderly [19].

Although age-related differences in neuromuscular function are well established [20] our understanding of the CNS mechanisms contributing to this movement deficit in old adults is limited. One factor that may contribute to this impaired motor performance is changes in inhibitory neurotransmission within primary motor cortex (M1) mediated by the neurotransmitter gamma amino-butyric acid (GABA). In young subjects, transcranial magnetic stimulation (TMS) has been used to show that the modulation of local GABAergic

Abbreviations: AMT, active motor threshold; SP, silent period; FDI, first dorsal interosseous muscle; fMRI, functional magnetic resonance imaging; GABA, gamma aminobutyric acid; LICI, long-interval intracortical inhibition; M1, primary motor cortex; MCP, metacarpophalangeal joint; MEP, motor evoked potential; MSO, maximum stimulator output; MVC, maximum voluntary contraction; RMT, resting motor threshold; SICI, short-interval intracortical inhibition; TMS, transcranial magnetic stimulation.

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inhibition is important for motor performance during isometric contractions [21–24]. Furthermore, variations in GABAergic inhibition during shortening and lengthening muscle contractions have also been proposed in young subjects [7,8,25], suggesting that these circuits may contribute to the accurate performance of slow movements. However, these previous studies have relied on the assessment of GABAergic inhibition from measures of the EMG silent period (SP) duration following TMS during shortening and lengthening contractions [7,8,25], which is difficult to interpret and highly sensitive to changes in spinal excitability (for review, see reference 26). In addition, there have been no assessments of GABAergic inhibition during movements in older adults. As previous studies with paired-pulse TMS have shown that a reduced ability to modulate GABAergic inhibition prior to contraction is associated with impaired motor performance in older adults [27], it is possible that age-related changes in the modulation of M1 GABAergic inhibition during movements (particularly lengthening contractions) may contribute to the movement performance deficits commonly observed in the elderly.

The main aim of the current study was therefore to investigate variations in GABAergic inhibition within contralateral M1 of young and old subjects during functional movements that involve shortening and lengthening contractions. We used paired-pulse TMS to assess $GABA_A$-mediated short-interval intracortical inhibition (SICI) and $GABA_B$-mediated long-interval intracortical inhibition (LICI), which provides a more robust assessment of M1 GABAergic inhibition compared with previous studies involving the SP [28]. As previous findings suggest that lengthening contractions are associated with disinhibition of contralateral M1 [7,8,25], we expected that lengthening movements would also be associated with a reduction in SICI and LICI. In addition, as the activity-dependent modulation of inhibitory tone is thought to be reduced in old adults [27,29,30], and this has been related to impaired motor performance in the elderly [27], we expected that old individuals would demonstrate less modulation of cortical inhibition during movement, and that this would be associated with greater motor deficits in older adults.

Methods

Eighteen young (mean ± SD: 23.3 ± 4.2 years; 9 females) and 16 old (70.6 ± 6.5 years, 9 females) healthy subjects were recruited from the university and wider community to participate in the current study. Exclusion criteria included a history of neurological or psychiatric disease, or current use of psychoactive medication (sedatives, antipsychotics, antidepressants, etc.). Hand preference and laterality were assessed using the Edinburgh Handedness Inventory [31]. All experimentation was approved by the University of Adelaide Human Research Ethics Committee and conducted in accordance with the Declaration of Helsinki. Each subject provided written, informed consent prior to participation.

Experimental arrangement

Subjects were seated in a chair with their right arm abducted approximately 45° at the shoulder. The right hand and forearm were pronated on a purpose-built manipulandum, similar to that described previously [32], which was located on a table in front of the subject. The index finger was extended over a cavity within the manipulandum, while the third, fourth and fifth fingers were flexed around the edge of the cavity at the level of the metacarpophalangeal (MCP) joint. The thumb was extended against a padded support on the manipulandum and the forearm was strapped to an adjustable rest. A strap was also placed across the hand to minimise movement. This position allowed abduction–adduction of the index finger that was isolated to activation of the first dorsal interosseous (FDI) muscle. A circular plastic cast placed around the distal end of the index finger was attached to a 50 g load via a length of low compliance line. The line ran over a pulley attached to the edge of the manipulandum, suspending the load in mid-air. Within this setup, abduction movements corresponded to raising the load (shortening contraction), while adduction movements corresponded to lowering the load (lengthening contractions), with the combined movements against the load defined hereafter as an anisometric contraction.

Surface EMG was used to record responses from the FDI muscle of the right hand. Two Ag–AgCl electrodes (1.5 cm diameter) were attached to the skin over the muscle in a belly-tendon montage, with a strap around the wrist grounding the electrodes. Acceleration of the index finger in the abduction–adduction plane was measured using a uniaxial accelerometer (V94-41, Coulbourn Instruments, Whitehall, PA) that was placed on the medial surface of the plastic cast attached to the index finger. Position of the index finger was assessed via a potentiometer where the rotational axis was aligned with the MCP joint and securely attached along the length of the index finger. EMG was amplified (300×) and band-pass filtered (20 Hz high pass, 1 kHz low pass) using a CED1902 (Cambridge Electronic Design, Cambridge, UK). EMG, position and acceleration signals were digitized at 2 kHz using a CED1401 interface (Cambridge Electronic Design), before being recorded and stored offline for analysis. To facilitate muscle relaxation when required, real-time EMG signals were displayed under high gain (50 μV/division) on an oscilloscope placed in front of the subject.

Experimental procedures

Maximal voluntary contraction

Index finger abduction force during maximum voluntary contraction (MVC) was assessed for each subject. MVCs were conducted with the hand positioned on the manipulandum as described above and with 0° of index finger abduction. When instructed, subjects abducted the lateral surface of the index finger against a force transducer (LC1205-K020; A&D Mercury Pty Ltd, Australia) placed in-line with the distal phalanx. Subjects were required to produce maximum force for 3 s in several repetitions, separated by 30 s rest, until the maximal force of three trials were within a 10% margin. The largest force recorded during these trials was chosen as the subject's MVC. To optimise force production, feedback was displayed on a computer monitor placed at eye level in front of the subject, and verbal encouragement was provided by the experimenter.

Postural, shortening and lengthening contractions

Subjects performed two types of low-intensity contractions against a 50 g inertial load: 1) postural contractions, during which the index finger was held abducted at a constant position of 10° from the index fingers neutral position; and 2) anisometric contractions, during which the subject performed abduction–adduction movements of the index finger over a 20° range of motion. For both contraction types, a display screen showing two cursors was placed at eye level in front of the subject. One cursor represented the position of the index finger, while the second represented a target position. Subjects performed the required movement by matching the position cursor to the target cursor. During postural contractions, the target cursor was static, representing the required abduction angle, whereas during anisometric contractions, the target cursor formed a triangular template representing a constant velocity contraction of 4 degs/s. The assessment of intracortical inhibition during postural contractions required subjects to maintain index finger abduction for approximately 4 minutes, while the assessment of intracortical inhibition during movement required

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