Interhemispheric interactions are relevant to fundamental cognitive processes including perception, awareness (Gazzaniga 2000), and motor control (Geffen et al. 1994). In the motor domain, interhemispheric inhibitory interactions (IHI) operate between homonymous body part representations in the primary motor cortices (M1) (Ferbert et al. 1992). IHI appears to be mediated to a large extent through transcallosal excitatory connections that terminate on inhibitory interneurons (Daskalakis et al. 2002b; Di Lazzaro et al. 1999; Ferbert et al. 1992; Gerloff et al. 1998). Patients with unilateral brain lesions have abnormally persistent IHI from the intact to the affected hemisphere in the process of generating a voluntary paretic hand movement (Murase et al. 2004). On the other side of the spectrum, decreased IHI has been reported in patients with schizophrenia (Daskalakis et al. 2002a) and epilepsy (Hanjima et al. 2001). Patients with multiple sclerosis (Boroojerdi et al. 1998; Schmierer et al. 2002), schizophrenia (Bajbouj et al. 2004; Boroojerdi et al. 1999), focal hand dystonia (Niehaus et al. 2001), and parkinsonian syndromes (Wolters et al. 2004) have abnormal ipsilateral silent periods, another marker of transcallosal interactions. In some cases, the severity of clinical impairment correlated with the magnitude of IHI abnormality (Murase et al. 2004; Schmierer et al. 2002). These findings led to the hypothesis that correction of IHI abnormalities may result in clinical gains. In an interesting report in this issue of the Journal of Neurophysiology (p. 1668–1675), Pal and colleagues (2005) describe a possible strategy to manipulate IHI. Downregulation of activity in one motor cortex using 1-Hz repetitive transcranial magnetic stimulation (rTMS) (Chen et al. 2001) results in decreased IHI over the opposite motor cortex that outlasts the period of stimulation.

Pal et al. (2005) studied IHI at rest using a paired-pulse technique. A suprathreshold conditioning TMS stimulus (CS) applied to one M1 is followed a few milliseconds later by a second suprathreshold test stimulus (TS) delivered to the opposite M1 (Ferbert et al. 1992). IHI is calculated as the amplitude ratio of the motor-evoked potential (MEP) elicited by the combination of CS+TS to the MEP elicited by the unconditioned TS alone. This technique allows the noninvasive evaluation of interhemispheric inhibition in humans, providing complementary information to that obtained using functional neuroimaging, EEG and MEG in cognitive neuroscience. Pal et al. (2005) now demonstrate the possibility of modulating IHI in healthy human subjects using inhibitory 1-Hz rTMS. These findings provide a methodological tool to explore a variety of exciting hypotheses in human motor control and cognitive processing.

For example, this approach could be used in cognitive neurology and neurophysiology to modulate interhemispheric interactions in patients with hemispatial neglect (Brighina et al. 2003), tactile extinction (Oliveri et al. 1999), and aphasia (Martin et al. 2004). The mechanisms underlying these behavioral effects could be the re-establishment of a proper balance of IHI. A better understanding of the mechanisms underlying IHI, as explored in this paper, is a condition to facilitate the development of rationale strategies to purposefully modulate IHI in disease states.

REFERENCES


