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Longitudinal changes of motor cortical excitability and transcallosal inhibition after subcortical stroke

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HIGHLIGHTS

- Continuing clinical improvement was observed over a 1-year period following subcortical stroke.
- Overall excitability in the unaffected hemisphere was increased at the post-acute period following stroke, which may have resulted in enhanced transcallosal inhibition to the affected hemisphere.
- It is unclear whether there was a causal relationship between the enhanced transcallosal inhibition and the extent of clinical recovery.

ABSTRACT

Objective: A general lack of longitudinal studies on interhemispheric interactions following stroke led us to use transcranial magnetic stimulation (TMS) to examine changes in corticospinal/intracortical excitability and transcallosal inhibition over a 1-year period following subcortical stroke.
Methods: We measured TMS parameters such as motor threshold (MT), short-interval intracortical inhibition (SICI), and ipsilateral silent period (iSP) and evaluated clinical scores at three time-points (T1, T2, and T3) in 24 patients and 25 age-matched healthy subjects.
Results: At T1, we observed reduced MTs and SICIs with prolonged iSPs in the unaffected hemisphere (UH). In contrast, increased MTs and reduced SICIs were observed in the affected hemisphere (AH). These abnormalities gradually reduced and no MEP response to TMS at T1 predicted a worse prognosis. The prolonged iSP at T1 was associated with more severe impairments, but it did not necessarily predict a worse prognosis after 1 year.
Conclusions: UH excitability was increased at the post-acute time-period, which may have resulted in enhanced transcallosal inhibition to the AH. However, it is unclear whether there was a causal relationship between the enhanced transcallosal inhibition and the extent of clinical recovery.
Significance: This is the first study to demonstrate changes in transcallosal inhibition over a longitudinal period following stroke.
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1. Introduction

Motor circuit reorganization in the cerebral cortex is known to contribute to recovery following stroke. This reorganization can be examined by transcranial magnetic stimulation (TMS) using

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measures of corticospinal and intracortical excitability (Liepert et al., 2000; Shimizu et al., 2002; Liepert et al., 2005; Talelli et al., 2006; Wittenberg et al., 2007; Bütefisch et al., 2008; Manganotti et al., 2008; Swayne et al., 2008; Takeuchi et al., 2010). TMS measures such as motor threshold (MT) and recruitment curve (RC) reflect corticospinal excitability, whereas intracortical excitability is reflected by cortical silent period (cSP), and short-interval intracortical inhibition (SICI) and intracortical facilitation (ICF), which is assessed by the paired-pulse TMS technique (Kujirai et al., 1993). Previous studies have demonstrated that reduced corticospinal excitability in the affected hemisphere could result not only from direct cortical damage but also from disruption of the corticospinal connection. In contrast, some studies have shown that decreased SICIs in both hemispheres might be associated with functional recovery after stroke (Liepert et al., 2000; Shimizu et al., 2002; Liepert et al., 2005; Wittenberg et al., 2007; Bütefisch et al., 2008; Manganotti et al., 2008). Most of these studies, however, were conducted over a short time-period following stroke (within 6 months).

It has been proposed that activity of the motor cortex (M1) in the unaffected hemisphere (UH) and/or the ipsilateral motor pathway of patients following stroke may play a role in functional recovery. This theory is based on an observation that the M1 in the UH of these patients was activated in association with movements of the paretic hand (Chollet et al., 1991; Cao et al., 1998; Marshall et al., 2000; Gerloff et al., 2006). However, there is some evidence that this is not the case. For example, the magnitude of M1 activation in the UH due to paretic hand movement does not correlate with functional recovery (Cramer et al., 1997; Ward et al., 2003). Additionally, ipsilateral motor pathways from the UH to the paretic hand are more commonly detected in patients with poor motor recovery (Netz et al., 1997; Gerloff et al., 2006). Lastly, a TMS study showed that disrupted M1 activation in the UH failed to delay simple reaction times in the paretic hand of patients with chronic stroke (Werhahn et al., 2003). Therefore, these findings support the hypothesis that a rescue of motor function in patients affected by chronic stroke relies predominantly on reorganization in the affected hemisphere (AH) (Werhahn et al., 2003; Ward et al., 2006). However, more recent imaging studies have demonstrated that the increased activation of the UH including the M1 was seen during movement of the affected hand in fully recovered chronic stroke patients (Butefisch et al., 2005; Schaechter and Perdue, 2008). Additionally, Lotze et al. (2006) demonstrated that rTMS over the dorsal premotor cortex, M1 and the superior parietal lobe of the UH during complex hand movements resulted in significant interference with recovered performance in chronic stroke patients. Thus, these studies support the idea that the UH is beneficial for some aspects of effectively recovered motor behavior after stroke (Lotze et al., 2006).

It has recently been suggested that interhemispheric imbalance is strongly related to motor function of the affected hand in patients with chronic stroke (Hummel et al., 2006; Nowak et al., 2009). Interhemispheric interactions between the two motor cortices can be assessed by paired-pulse TMS using measures such as interhemispheric inhibition (IHI) (Ferbert et al., 1992) or by ipsilateral silent period (iSP) (Meyer et al., 1995). Both IHI and iSP are believed to involve transcallosal pathways (Ferbert et al., 1992). Murase et al. (2004) studied IHI from the UH to the AH while patients with chronic subcortical stroke were told to generate a voluntary movement in their paretic hand. IHI at rest was comparable between patients and healthy controls, however, closer to movement onset, IHI turned into facilitation in controls but not in patients (Murase et al., 2004). These results suggest that an enhanced IHI from the UH to the AH might interfere with motor function of the affected hand. Another study demonstrated that 1-Hz rTMS (suppressive rTMS) over the UH can reduce the iSP (transcallosal inhibition from the UH to the AH) and improve motor function of the affected hand in patients with chronic stroke (Takeuchi et al., 2005). This group suggested that M1 in the UH inhibits M1 in the AH via an abnormally enhanced transcallosal inhibition, and that disruption of this abnormal transcallosal inhibition by 1-Hz rTMS causes paradoxical functional facilitation of the affected hand. Based on these findings, it has been suggested that enhanced transcallosal inhibition from the UH to the AH could adversely influence motor recovery in some patients with subcortical stroke (Murase et al., 2004). However, there have been no longitudinal studies on interhemispheric interactions following stroke.

One question addressed in the current study is how reorganization of bilateral motor circuits and interhemispheric connections occurs over the long course of recovery following stroke. A related question is how neurophysiological measures correlate with clinical scores. We hypothesize that there is excessive transcallosal inhibition from the UH to the AH associated with the disinhibited M1 of the UH, which could hamper recovery of motor symptoms, especially in the post-acute phase. Furthermore, we hypothesize that this interhemispheric imbalance gradually improves with clinical recovery. Therefore, in the current study we assessed longitudinal changes of transcallosal inhibition (using iSP) as well as corticospinal (using MT and RC) and intracortical (using cSP, SICI, and ICF) excitability by TMS and their clinical correlations from post-acute to chronic time-periods (up to 1 year) following subcortical stroke. Patients with subcortical stroke were chosen on the grounds that their cerebral cortex and transcallosal pathways were preserved, and we considered that dynamic functional changes could occur in these patients.

2. Subjects and methods

2.1. Subjects

A total of 24 patients (16 men and eight women) were recruited from the recovery rehabilitation ward. Ages of the patients ranged

Table 1
Patient characteristics.

Pt	Age	Sex	Affected hand	Lesion	Etiology	T1	T2	T3
1	64	M	R	P	Hemorrhage	30	163	367
2	76	M	R	CR	Lacunar	28	91	372
3	69	F	L	CR	Lacunar	18	81	360
4	81	F	R	CR∼DWM	Atheroma	23	58	365
5	50	F	R	T	Hemorrhage	24	135	359
6	72	M	L	P	Hemorrhage	28	69	365
7	75	F	R	IC	Lacunar	33	104	363
8	56	M	L	P	Hemorrhage	23	93	367
9	52	M	R	P	Hemorrhage	22	71	366
10	55	M	R	CR∼DWM	Atheroma	40	172	368
11	51	M	R	CR	Lacunar	18	53	362
12	39	M	R	CR	Lacunar	18	123	369
13	73	M	R	CR	Lacunar	18	54	365
14	72	M	R	CR	Lacunar	31	59	367
15	77	F	R	P	Hemorrhage	28	104	363
16	66	F	R	T	Hemorrhage	31	116	367
17	50	M	R	T	Hemorrhage	23	93	366
18	58	F	R	T	Hemorrhage	13	97	378
19	62	M	L	P	Hemorrhage	18	116	362
20	60	M	R	T	Hemorrhage	35	140	371
21	72	M	R	P	Hemorrhage	40	89	369
22	72	F	L	T	Hemorrhage	34	118	370
23	67	M	R	CR	Lacunar	32	137	361
24	58	M	L	T	Hemorrhage	33	117	361

Pt indicates patient; M, male; F, female; Rt, right; Lt, left; P, putamen; CR, corona radiata; T, thalamus; IC, internal capsule; DWM, deep white matter, T1, T2, T3, days post-stroke.

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