Editorial

European Neurology

Eur Neurol 2004;51:189–190
DOI: 10.1159/000078483

Published online: May 17, 2004

Brain Plasticity in Multiple Sclerosis

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The last two decades have witnessed an increased effort in understanding the mechanisms responsible for the accumulation of irreversible disability in multiple sclerosis (MS). In this context, the clinical application of magnetic resonance imaging (MRI) techniques has progressively increased our ability to achieve an accurate quantification of the overall burden of the disease and to monitor its course objectively. More recently, the use of functional MRI in the assessment of MS patients has shown a modified recruitment of cortical areas during motor, cognitive and sensory tasks in MS patients [1].

Functional cortical changes have been demonstrated during the acute phase of the disease [2] and in clinically stable patients with different disease phenotype and clinical impairment [1]. It has also been shown that cortical adaptive changes might be elicited and/or modulated by different substrates, including the extent of T_2-visible lesions and the severity of their intrinsic damage, the amount of normal-appearing white matter and gray matter damage, the extent of axonal loss/dysfunction and the severity of spinal cord involvement [1]. Up to now, the role of brain atrophy on cortical functional changes has not been fully elucidated yet. Conversely, this variable has been carefully controlled in all the previous studies in order to avoid a possible bias in functional MRI results. The single case report of Roelcke et al. [3] presented in this issue may be considered as a preliminary view on this important topic. The progressive development of brain atrophy is a well-known feature of MS patients [4]. Using different measurement techniques, many studies have shown that atrophy of the brain is present from the earliest stages of the disease and tends to progress with disease evolution [4]. In their report, Roelke et al. [3] compared the movement-associated pattern of cortical activations between the atrophic and the normal hemisphere in an MS patient with unilateral brain atrophy and found no differences in the location, extent and significance of the cluster of activations between the two hemispheres.

In some longitudinal follow-up studies of MS patients with a subcortical lesion affecting the corticospinal tracts, we invariably found during self-paced movements an increased activation of the contralateral primary motor cortex associated with increased activation of the supplementary motor area and ipsilateral primary motor cortex. During the follow-up, the overactivation both in the contralateral and ipsilateral areas always decreased if a good recovery of motor function was achieved. However, also in patients with good recovery, some increased activation of cortical motor areas persisted, indicating that cortical reorganization contributed to the recovery. Interestingly enough, in the more advanced phases of the disease, in patients with severe motor deficits, the overactivation of cortical motor areas (both contralateral and ipsilateral) disappeared, suggesting that the decrease in motor ability

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0014–3022/04/0514–0189$21.00/0

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was associated with a failure of cortical reorganization probably because of an exhaustion of brain plasticity and/or a degeneration of pyramidal tracts.

Our observation is fully consistent with the 'apparently' unmodified motor cortex activity in a hemisphere with a clear relevant cortical-subcortical atrophy and a severe involvement of the pyramidal tracts as demonstrated by the reduced amplitude of the motor potential evoked by cortical stimulation.

Further longitudinal studies of larger samples of MS patients are now warranted to better define the dynamic changes of cortical reorganization during the evolution of MS.

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