

Cognitive training in MS: Effects and relation to brain atrophy

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Abstract. *Purpose:* Cognitive disorders are common in MS patients without any generally recommended treatment. Recent brain imaging studies show considerable neuroplasticity for cognitive tasks in MS patients, but also brain atrophy already early in the disease progression. We explored the benefits of a home-based cognitive training program for memory and working memory functions in relapsing-remitting MS patients controlling for whole brain and central brain atrophy as covariates. *Methods:* Using a single-blinded controlled study design, 42 patients were randomised into a treatment group and a control group. Home based computer training focusing on memory and working memory was started at least 4 weeks after the discontinuation of methylprednisolone treatment and lasted for 6 weeks. Two weeks later the patients were re-investigated for their clinical and cognitive performance. We assessed also quality of life (QoL), depression and fatigue using self-rating scales. *Results:* Training had no effect on the neurological status and on QoL or fatigue. However, the treatment group showed better verbal learning, long-delay verbal memory performance, and working memory performance. The impact of treatment on long-delay verbal memory performance was independent from the extent of brain atrophy, whereas for the other findings brain atrophy played a significant role. *Conclusions:* An intensive home-based cognitive training program is suitable to improve the cognitive performance of MS patients. The impact of brain atrophy on rehabilitation outcome may differ for cognitive functions.

Keywords: Multiple sclerosis, cognitive training, memory, working memory, brain atrophy

1. Introduction

Cognitive impairments are common in multiple sclerosis (MS) and often influence patients' quality of life and job (Brassington & Marsh, 1998; Hildebrandt & Schwendemann, 2004; Prosiegel & Michael, 1993; Wishart & Sharpe, 1997; Zakzanis, 2000). Treatment with interferon beta may reduce disease progression

as well as the development of cognitive impairments (Barak & Achiron, 2002; Fischer, Priore, Jacobs, Cook-fair & Rudick, 2000; Pliskin, Hamer & Goldstein, 1996). However, this has not been replicated in all studies (Selby, Ling & Williams, 1998), and there is not such a finding for glatirameracetate (Weinstein, Schwid & Schiffer, 1999). In addition, immunomodulatory treatment does not improve already existing cognitive deficits of MS patients. There are several attempts of symptomatic treatment of cognitive impairment in MS using pharmacological agents such as amantadine and modafinil, focusing mainly on fatigue (Amato & Zipoli, 2003; Rosenberg & Shafor, 2005). Recent-

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