Background and Rationale

Changes in interhemispheric connectivity in Huntington’s Disease (HD) have been only recently investigated and little is known about their temporal relation with clinical features or grey matter atrophy: callosal disruption could contribute both to cognitive dysfunction and impairment of associative functions (Rosas et al., 2010) and likely occurs many years before clinical onset, along a posterior-to-anterior direction (Phillips et al., 2013). Here, we evaluated changes in ipsilateral Silent Period (iSP: onset latency, iSPOL, and duration, iSPD) and Transcallosal Conduction Time (TCT) in HD patients and correlated electrophysiological findings with mutational load and motor score.

Materials and Methods

Seven drug-free patients were enrolled, a number in line with previous papers studying early HD patients (Schippling et al., 2009). They had undergone genetic testing, which was diagnostic in all (CAG number ≥ 40). The mean duration of symptoms was about one year (15.1 ± 5.8 months). Electromyographic recordings were made from the ipsilateral abductor pollicis brevis muscle. A Super Rapid Transcranial Magnetic Stimulator connected to a eight-shaped focal coil with wing diameters of 70 mm was used. iSP onset was defined as when the post-stimulus EMG fell continuously (for at least 10 ms) in a window 30–60 ms after the stimulus. Subjects had to maintain a slight, unilateral, voluntary muscle contraction by means of an EMG visual feedback system and a maximum EMG peak-to-peak amplitude of 1 mV; a biphasic pulse of about 160% of the individual Resting Motor Threshold (RMT) value was chosen to evoke the iSP. The transcallosal conduction time (TCT) was then calculated by subtracting the shortest contralateral MEP onset latency from the iSP onset latency (Pettijean and Ko, 2014).

Discussion and Conclusions

- Our data proved that interhemispheric processing is impaired in early HD and significantly correlates with clinical and genetic data.

- Our results could have implications for the disruption of both sensorimotor integration and voluntary motor control in HD. Immediately before voluntary movements, interhemispheric interactions are likely responsible both for the temporary inhibition of ipsilateral primary motor cortex (M1) and the increased excitability of contralateral one (Leocani et al., 2000). Concurrently, especially during non-dominant hand movements, enhanced interhemispheric inhibition from the ipsilateral hemisphere suppresses superfluous activation arising from the contralateral cortex (Kobayashi et al., 2003). These mutual interactions may be lost in symptomatic HD, thus contributing to early motor symptoms as hyperkinesias.

- No study exists to date about the use of TMS to explore interhemispheric function in the whole field of hyperkinetic movement disorders; this approach may lead to a better knowledge of disease mechanisms, also in pre-symptomatic patients and other hyperkinetic movement disorders.

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