



Neuroimaging Assessment of Spasticity Developed after Acquired Brain Injuries and Multiple Sclerosis

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SUMMARY – *This study evaluated whether different imaging techniques correlate with specific variables routinely used to grade the types and complexities of patient conditions in neurorehabilitation services and their clinical outcomes, and if there are radiological patterns, topography or distribution of the lesions correlated to spasticity. The cohort studied included 75 patients, 38 patients with multiple sclerosis (MS) and 37 patients with acquired brain injuries (ABI) referred to the neurorehabilitation department from April 2009 to March 2010. Data included age, gender, diagnoses, complications, spasticity, length of stay, Rehabilitation Complexity Scale (RCS) and Northwick Park Dependency (NPDS) scores on admission and discharge. Forward stepwise multiple regressions were performed considering Spasticity as dependent and considering NPDS, RCS on admission and discharge, age, gender and length of stay as independent variables. Standardized intra-axial lesions in those with spasticity were fused with standardized axial DTIs from normal controls obtained under 3 T and 1.5 T MRI scanners. Spasticity was present in 36 patients. Regression was found between spasticity and all the other variables with $r = 0.42$, $r^2 = 0.17$, $_{adj}r^2 = 0.12$, $F_{(4,66)} = 3.53$, $p < 0.01$. Residuals were acceptable. If the same is calculated excluding MS patients the results are higher with: $r = 0.59$, $r^2 = 0.35$, $_{adj}r^2 = 0.30$, $F_{(2,27)} = 7.1885$, $p < 0.003$. In MS subjects, spasticity appeared if lesions affected pyramidal tracts, callosal radiations extensively or brainstem. Those with ABI did develop spasticity if the lesion involved the internal capsules, pericommissural areas, optochiasmatic cistern or brainstem. Findings suggest that NPDSa and discharge and RCSd are higher in those developing spasticity. No obvious correlation between spasticity and RCSa was found. Subjects with MS are more likely to develop spasticity especially if the lesions are numerous and affect the corticospinal tracts, callosal radiations extensively or brainstem. In patients with ABI lesions affecting pyramidal tracts, pericommissural areas and optochiasmatic cisterns seem more likely to develop spasticity.*

Introduction

Spasticity is defined as disordered sensorimotor control resulting from an upper motor neuron lesion and presenting as intermittent or sustained involuntary activation of muscles, a physiological consequence of an insult to the brain or spinal cord¹ which can lead to life-threatening disability and costly consequences².

Quantifiable clinically with simplicity following the Modified Ashworth Scale³ (Table 1) in general or specific muscular group cases⁴, spasticity remains a major clinical problem, which should be treated at the earliest opportunity to improve the chances of appropriate recuperation *ad integrum*, or the least handicapping degree if full recuperation is not reachable⁵.