Functional evaluation of damage to chromatic pathways in Multiple Sclerosis

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Multiple Sclerosis (MS) is the most frequent acquired disease of the central nervous system in young adults, frequently associated with Optic Neuritis (ON), that is generally manifested as blurring, graying, or loss of vision, most often unilaterally. However, subclinical cases may occur, in the absence of visual symptoms. The goal of this work was to evaluate and quantify subtle dysfunction of chromatic visual pathways in MS, with or without previous ON, and its correlation with clinical parameters of disease progression.

Forty-four subjects (12 males, 32 females; mean age= 42,29 $\pm$ 10,89) with MS, with or without previous history of ON, with no other ophthalmologic disease was compared with age-matched controls (12 males, 16 females; mean age= 49,71 $\pm$ 11,54 years). Patients were submitted to a standard neurological examination, including clinical staging (EDSS - Expanded Disability Status Scale). Standard and novel computed psychophysical tests were used to evaluate chromatic visual function (Ishihara plates, Anomaloscopy and the Cambridge Colour Test). Visual Evoked Potentials was also applied in order to evaluate objectively the effect of demyelination on bioelectrical conduction along the visual pathway.

Using computerized evaluation of chromatic function, we have found evidence for damage of all cone pathways in MS, even in the absence of ON and in cases of preserved visual acuity. Interestingly, evidence for axonal damage in the optic nerve dominates over functional evidence for demyelination in MS without ON. Correlation analysis between EDSS and visual dysfunction confirms that damage in MS without previous ON is distinct from MS with previous ON (significant only in the former).

Novel computerized quantitative psychophysical methods for chromatic function evaluation can detect subtle visual dysfunction, revealing impaired optic nerve function in MS even in the absence of ON and provide new insights into distinct stages of the pathophysiology of this disease.