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Abstract Title: Optic Nerve involvement in Experimental Autoimmune Encephalomyelitis to homologous Spinal Cord Homogenate immunization in the Dark Agouti rat
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Abstract

BACKGROUND AND OBJECTIVE: We performed Visual Evoked Potentials (VEPs) on Dark Agouti (DA) rats immunized with homologous spinal cord homogenate (SCH). This strain develop a protracted and relapsing Experimental Autoimmune Encephalomyelitis (EAE), an animal model of multiple sclerosis (MS). Although motor symptoms have already been studied in this MS model, little is known about central nervous system damage, in particular what happens in visual pathways. We will focus our investigation in this district, since visual symptoms are common in MS patients. In particular, we explored the involvement of visual pathways in SCH-EAE using VEPs, which are reliable biomarkers of neurophysiological dysfunctions affecting optic nerves (ONs).

METHODS: Baseline VEPs were recorded through epidural screw or epidermal cup electrodes in 27 DA rats. EAE was induced in 12 rats through injection of rat SCH at the base of the tail. EAE motor symptoms were assessed daily, while VEPs were recorded once a week for five or eight weeks with epidural (n = 14, 6 immunized) and epidermal (n = 13, 6 immunized) electrodes, respectively. At the end of the study, ON histopathology was performed.

RESULTS: All the immunized rats showed motor signs a week after SCH injection. VEPs were physiologically normal until the second week of recording, then a significant delay was detected, persisting until the fifth week, when a recovery appeared in the epidermal-recorded rats and lasted until the sacrifice. Histopathologic results showed morphological damage in EAE ONs dissected from epidural-recorded rats, consistent with VEP alterations. In epidermal-recorded rats, the presence of lesions in ONs was not significant, congruent with physiological VEP measurements.

CONCLUSIONS: In SCH-EAE model, VEPs were useful for detecting alterations in the visual pathway and could be exploited as a *bona-fide* neurophysiological marker to test the efficacy of novel treatments directed against neurodegenerative processes involving the ONs.