Occurrence of limb movement during sleep in rats with spinal cord injury

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Abstract

Several studies have shown the occurrence of Periodic Leg Movement (PLM) in spinal cord injury patients. The aim of this study was to identify the occurrence of limb movements during sleep in spinal cord injury rats and the possible involvement of the spinal cord in causing these movements. The animals were allocated to spinal cord injury (SCI) and SHAM groups. The two groups were submitted to surgery and electrodes inserted to analyze sleep patterns (electroencephalogram—ECoG) and muscular activity patterns (electromyogram—EMG). After baseline sleep recording (24 h), the spinal cord injury surgery (level T9) was performed on the SCI group rats and sleep was recorded for seven consecutive days. After spinal cord injury, 10 of the 11 rats began to present limb movements during sleep, while the SHAM group showed no limb movements during the 8-day sleep-recording period. In relation to sleep efficiency, the SCI group presented alterations during the first few days after spinal cord injury but returned to normal values at the end of the 7-day experimental period. The data suggest that spinal cord injury rats may be used as models to study PLM in paraplegic patients, and that these movements may be generated in the spinal cord itself, without the involvement of the cortical structures.

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1. Introduction

Periodic Limb Movements (PLM) are stereotyped movements of lower limbs, that appear specially during light sleep stages 1 and 2 and usually take place at 5–90 s intervals \cite{1}. PLM affects 6\% of the population and may occur associated with the restless legs syndrome (RLS) \cite{3}. This disturbance may lead to reduced sleep efficiency and quality.

The precise origin and pathophysiological mechanisms for PLM are obscure. However, there are descriptions in the literature of the possible involvement of the spinal cord.

Though reduced cortical inhibition \cite{16} or impairment of cortical–sub cortical motor structures, in particular of motor inhibitory pathways, has been reported in RLS, a cortical origin for PLM is unlikely because back-averaging studies failed to disclose any cortical potentials preceding PLM. However, the occurrence of PLM in patients with spinal cord injury suggests that PLM may be directly generated in the spinal cord \cite{4,5,6,9,20}.

Having studied 10 physically disadvantaged subjects (two of them spinal cord injury patients) and observing leg movements during sleep, Yokota et al. \cite{20} suggested that PLM of spinal cord origin may be induced by interruption of a tract distinct from but close to the corticospinal tract. In another study, Lee et al. \cite{9} described abnormal lower limb movements during sleep in three patients with spinal cord injury at the thoracic level and related them to PLM. De Mello et al. \cite{4–6} have described treatment of PLM based on physical exercise and dopaminergic agonist drugs, and a positive correlation between K-complex and PLM.

The findings of Hains et al. \cite{8} demonstrated dynamic plasticity in properties of dorsal horn somatosensory neurons after SCI (rats).