Changes in Spinal Motoneuron "Fastness" in Post-stroke Spastic Patients

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Abstract

Motor unit (MU) action potentials trains were recorded from brachial biceps of 11 post-stroke patients (Brunstrom stage 2–4) and 8 healthy volunteers. The variability analysis of interspike intervals (ISIs), aimed at the comparison of the duration of afterhyperpolarization (AHP) in motoneuron (MN), was performed on MUs of 39 controls and 64 patients. Our results concerning MU discharge characteristics conformed in general to the previous reports, showing positive serial correlation coefficients and lower MU firing rates in patients as compared with control subjects. We have found, however, that the positive correlation coefficients result from trends in MU firing rates related to less efficient control of the muscle force. ISI variability determined in short-interval range was the same in patients and control subjects. The transition intervals of variability-mean ISI characteristics, which were previously shown to correlate with AHP duration, were significantly longer in patients, but the prolongation decreased with patient's age and disease duration. Our results indicate that the spinal MNs respond to the cerebral stroke with prolongation of AHP duration, which tends to recover after the accident. These changes are less pronounced in older patients, presumably due to decreased MN plasticity. We conclude that the match between MN and muscle properties is preserved after stroke.

Keywords: Stroke, Age-dependence, Afterhyperpolarization (AHP), Human motoneuron (MN) firing patterns, Neuronal plasticity

1. Introduction

Spasticity is one of the consequences of central nervous system (CNS) lesions, which can result in serious problems for affected individuals and therefore is still a challenge to clinicians and scientists. Although considerable scientific and medical literature discusses the etiology and treatment of spasticity, the underlying mechanisms are largely unknown. In particular, motor unit (MU) firing patterns were often studied in spastic patients [1-7] but there is very little knowledge about the alterations which spastic disorders induced in motoneurons (MNs).

Spasticity after cerebral lesions, including stroke, is often thought to be connected with increased contraction of the skeletal muscle. Experimentally, such conditions may be reproduced with chronic electrical stimulation, which was investigated in several experiments [8-11]. The stimulation caused muscle phenotype shift from fast towards slow. In line with these results are reductions of firing rates observed in spastic post-stroke patients [2-6]. In contrast, spasticity after spinal cord injury (SCI) is more often associated with the chronic decreased use of the muscle, which leads to the muscle transformation towards faster phenotype, both in animals [12] and in humans [13]. In rat SCI model, this transformation in soleus muscle was shown to be accompanied by the decrease in motoneuronal afterhyperpolarization (AHP) duration [14]. However, MUs in other muscles and other species became slower after SCI [15,16]. Even single MUs in the given muscle could be differentially affected during long-term spastic hemiplegia [17].

The animal models of stroke are still under development. To our knowledge, studies on changes in motoneuron properties after stroke were not yet reported. It was suggested that the decreased MU firing rates could be due to the increased AHP duration of their MNs [18] and the match between motoneuron and muscle fiber characteristics in stroke patients is not preserved, which might reduce the efficiency of muscle contraction [4]. This issue thus calls for further investigations.

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