

MODULATION OF THE SPINAL CORD MOTOR EVOKED POTENTIALS AFTER SPINAL CONTUSION IN RAT DURING TREATMENT WITH LOCAL HYPOTHERMIA

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Introduction

Motor evoked potentials induced by epidural spinal cord stimulation can be used for evaluation of the spinal cord functional state after spinal cord injury (SCI) (Lavrov et al. 2006). In this study, the early response (ER) and the middle response (MR) of spinal cord evoked potentials were studied during a one-month period after applying of local hypothermia following contusion of spinal cord in rat.

Methods

The study was performed on laboratory Wistar rats. The contusion was made on the level of open T8 vertebra. There were two experimental groups: animals with SCI contusion and animals receiving hypothermia procedures after SCI contusion. The local hypothermia was applied immediately after contusion at the same site for 20 min. The motor evoked potentials induced by epidural stimulation of S1 spinal cord segment were recorded from m. gastrocnemius m. soleus, and m. tibialis anterior on 3, 7, 14, 21 and 30 days after contusion and hypothermia procedures. The responses from intact animals were control. All procedures were performed in accordance to bioethics norms and data were processed statistically with one-way ANOVA.

Results

We found that in m.gastrocnemius the maximum amplitude and threshold of ER and MR were not significantly changing during one-month period after contusion, but after applying a local hypothermia, the threshold of MR was not significantly increase to on 14 day and return to control values on 30 day.

In m. soleus the maximum amplitude of ER and MR stayed decreased on 30 day in group with SCI, and increased to 190,81±56,79% on 30 day in group treated with hypothermia. The value of the threshold of ER and MR responses did not significantly change.

In m. tibialis anterior the maximum amplitude of ER was significantly increased to 220,99±52,51% on 1 day after contusion and recovered back to control values on 21 day in first group. On the contrary, in a groupe treated with hypothermia the ER maximum amplitude significantly decreased to 25,88±16,32% on 1 day then

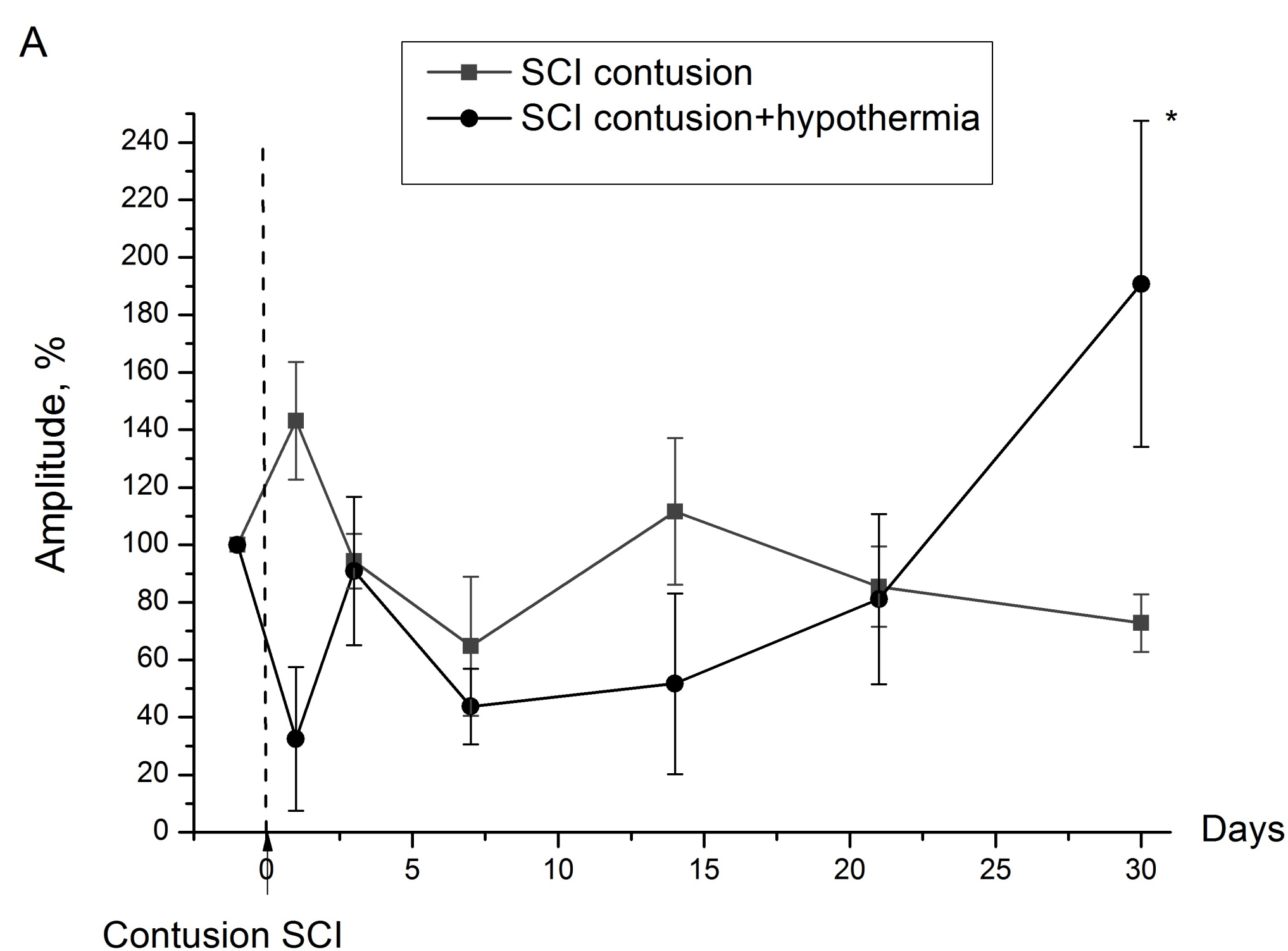


Fig.1 Amplitude of ER response (100% - the value of ER amplitude of intact animal) of m. soleus in a group of animals with SCI contusion and group received hypothermia treatment after SCI contusion. * - differed significantly from the group with SCI contusion (P<0,05).

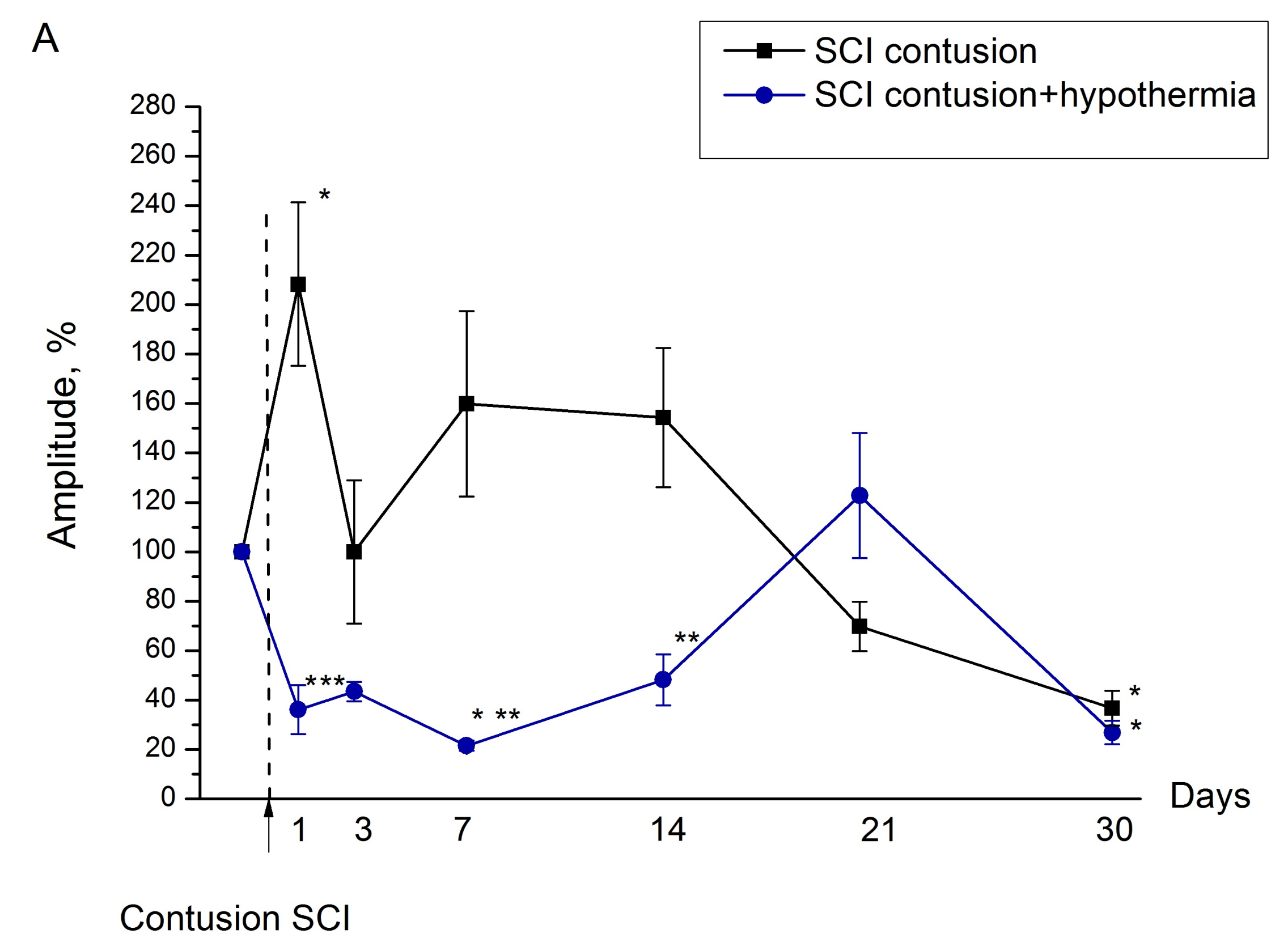


Fig.2 Amplitude of ER response (100% - the value of ER amplitude of intact animal) of m. tibialis anterior in a group of animals with SCI contusion and group received hypothermia treatment after SCI contusion. * - differed significantly from the group with SCI contusion (P<0,05). ** - differed significantly from the group of intact animal (P<0,05).

recovered back to control values on 21 day and again significantly decreased to 28,94±9,47% on 30 day. The MR amplitude did not significantly change in a first group. But in second group it was observed a significant decrease of MR amplitude (to 28,97±9,67%) on 7 day after SCI contusion and treatment with hypothermia. The threshold of ER and MR of m. tibialis anterior were significantly increased to 2,77±1,23V on 14 day and to 2±0,3 V on 21 day only after hypothermia treatment.

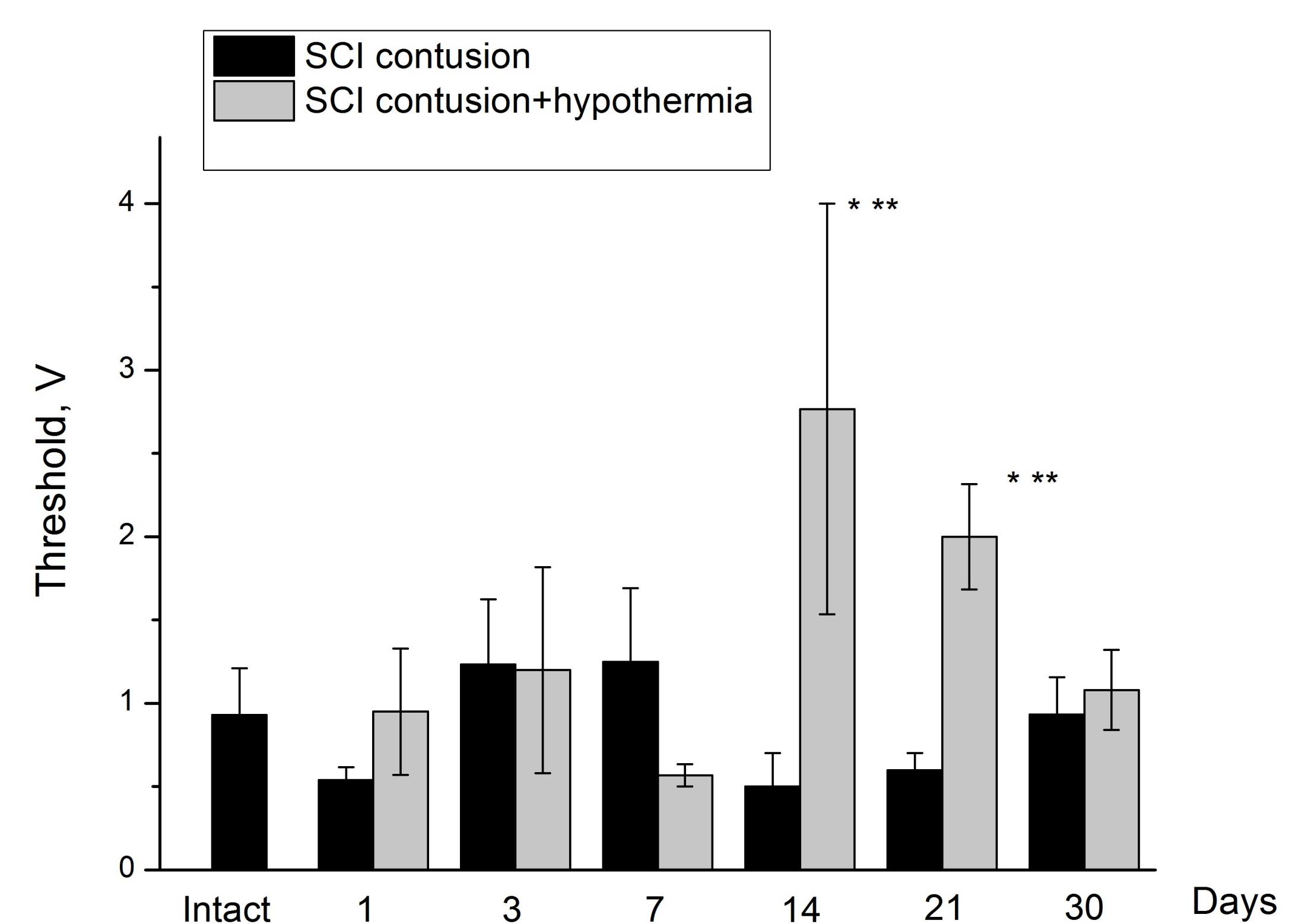


Fig.3 Threshold of ER response (in Volts) of m. tibialis anterior in a group of animals with SCI contusion and group received hypothermia treatment after SCI contusion. * - differed significantly from the group with SCI contusion (P<0,05). ** - differed significantly from the group of intact animal (P<0,05).

Previously it was shown that local hypothermia after spinal cord contusion might delay development of excitability of the motoneurons in dogs for a week (Iafarova et al. 2014). Our results suggested that hypothermia treatment decreased the excitability in the spinal circuitries of hindlimbs muscles in rat during first 3 weeks after SCI and recovering back on 30 day. Though hypothermia along with inhibitory effect on damage process occurring after SCI might caused delay recovery of neuromuscular system of hindlims in rat.

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