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Effect of a 0.5-T static magnetic field on conduction in guinea pig spinal cord

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Abstract

Compound-evoked potentials were recorded from excised adult guinea pig spinal cords before, during, and following exposure to a 0.5-T static magnetic field (SMF). There was no change in response latency during exposure but there was a small, statistically significant, decrease in amplitude. Maximum effect was evident 1 to 2 min after the field was turned on with return to baseline within 1 min after the field was turned off. These results may be explained by a conduction block in the small axon subpopulation due to the effect of static magnetic fields on voltage-activated sodium channels. The relative selectivity of the field is believed to occur because of the relatively greater number of sodium channels present in smaller axons.

Keywords: Static magnetic field; Sodium channels; Spinal cord

1. Introduction

Moderate intensity static magnetic fields (SMF) have been shown to influence a variety of biological systems, particularly those whose function is closely linked to the function of membrane ion channels. Changes have been demonstrated in the somatosensory-evoked potential in rats [1], in spontaneous central nervous system neuronal activity in cats [2], in neurotransmitter release at the neuromuscular junction in mice [3], and in neuronal action potentials in dissociated cultures of dorsal root ganglia neurons in mice [4]. In all of these studies, it appears that SMFs exert their influence primarily at the synapse and it has been proposed [5] that these fields alter the function of membrane ion channels. The present study was carried out to determine if a moderate intensity static magnetic field is sufficient to influence nonsynaptic axonal excitability in mammalian spinal cord.

2. Methods

With the approval of the institutional animal utilization committee, studies were carried out on adult female guinea pigs. The spinal cord was removed following anesthetization with ketamine hydrochloride (60 mg/kg), acepromazine maleate (0.6 mg/kg), and xylazine (10 mg/kg). Following removal, a 35–38-mm segment of cord was bathed in oxygenated Krebs solution (in mM: 124 NaCl, 2 KCl, 1.2 KH_2PO_4, 1.3 $MgSO_4$, 1.2 $CaCl_2$, 10 dextrose, 26 $NaHCO_3$, and 10 Na-ascorbate) for 1 h before use. Recordings of compound action potentials were carried out in a double sucrose gap recording chamber [6,7] perfused with oxygenated Krebs solution. The temperature of the perfusate was maintained at 36.8–37.0 °C. The chamber was centered between the poles of a water cooled electromagnet with a horizontally oriented field, perpendicular to the long axis of the cord. This magnet, with its 10.2 diameter poles separated by 5.5 cm, generated a 0.5-T field that, in the area of the cord, had a nonhomogeneity of less than 0.1%. Cord segments were stimulated at 2 per second with 150-μs square waves, 9–15 V in amplitude. Fifteen consecutive responses were digitally averaged and stored. Readings were made at 1-min intervals for 20 min. The initial 5 min served as the

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control. During the second 5-min interval, the cord was exposed to the magnetic field. Post-exposure responses were recorded for 10 min.

3. Results

Data were collected from the spinal cords of 10 animals, in 5 of which stability was sufficient to allow detailed analysis. A typical compound action potential is shown in Fig. 1. The mean latency for all control responses was 0.28 ms with a standard deviation of 0.022. There were no changes in latencies during exposure to the magnetic field. The amplitude of the responses varied from 400 to 600 μV. In each experiment, the mean amplitude during the control period was calculated and the percent change from that value for each response was determined. Percent changes from all experiments were averaged and subjected to a non-parametric Wilcoxon analysis with a null hypothesis of zero. The results, summarized in Fig. 2, demonstrate a significant decrease in the amplitude of the response 1–2 min after onset of magnetic field exposure with return to baseline in the post-exposure period.

4. Discussion

A theoretical analysis [8] predicted that magnetic fields of at least 24 T would be required to produce sufficient Lorentz forces to slow axonal conduction. This model, however, did not consider the possibility that lower intensity fields might alter the function of ion channels in excitable membranes. It has been shown that the activation kinetics of both calcium [9] and sodium [10] channels are transiently slowed during exposure to static magnetic fields with intensities of only 125 mT. The mechanism proposed [5] to explain this phenomenon is based on the diamagnetic anisotropic properties of membrane phospholipids. Reorientation of these molecules during SMF exposure results in deformation of imbedded ion channels, thereby altering their activation kinetics. Channel inactivation will not be influenced by these fields because that mechanism is not located within the intramembraneous portion of the channel. Sodium channels, essential for the propagation of action potentials, are not uniformly distributed in mylenated axons. Rather, they are concentrated at the nodes of Ranvier [11,12], where their density is nearly 500 times greater than in the internodal membrane. In the central nervous system, it has been estimated that the internodal distance is 1–2 mm [13,14]. In our preparation, this translates to 10 to 20 nodes between the stimulating and recording sites. In the presence of a moderate intensity static magnetic field, there is a reduced probability of sodium channel opening [5]. Smaller fibers have shorter internodal distances [13] and therefore more sodium channels exposed to the field. A conduction block in this subpopulation of axons would not be expected to influence the latency of the compound action potential, that latency being determined by the larger and faster conducting axons. It would, however, explain the observed decrease in its amplitude.

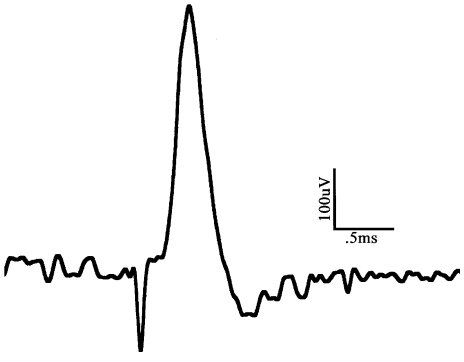

Fig. 1. Typical compound action potential recorded from guinea pig spinal cord. Digital average of 15 responses following 150-μs duration square wave stimulus. Negative deflection is stimulus artifact.

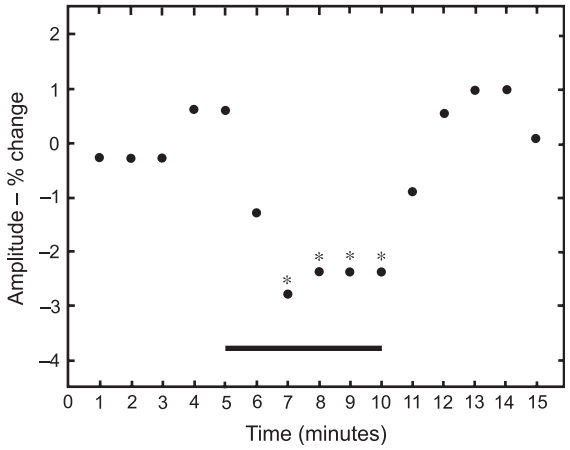

Fig. 2. Percent change in amplitude of recorded potentials when compared to control values. Means for all responses. Horizontal bar indicates exposure period to a 0.5-T static magnetic field. Points with * are those where $P < 0.05$ in Wilcoxon analysis for a null hypothesis of zero.

5. Conclusion

The results of this study are consistent with the previous findings that moderate intensity static magnetic fields affect Na^+ channels. The potential use of these fields for selective small fiber block in both the central and peripheral nervous system needs to be explored further.

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