

Relationship between Bioelectric Activity of Neurons in the Gigantocellular Nucleus of the Medulla Oblongata and Spontaneous Movements of Chick Embryo

E. A. Tsitsurina

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Electrophysiological study revealed a correlation between changes in bioelectric activity of the reticular gigantocellular nucleus and movements of chick embryos during ontogeny (16-20 days). This relationship increased by the end of embryogenesis. The reticular gigantocellular nucleus is the major source of supraspinal influences on motor activity during ontogeny. Blockade of proprioceptive impulses with myorelaxin inhibited bioelectric activity of the regulatory gigantocellular nucleus, which attests to the activating effect of proprioception.

Key Words: *gigantocellular nucleus; ontogeny; myorelaxin; motor activity; proprioception*

Growth of supraspinal reticular fibers to motoneurons during ontogeny is a necessary and very important factor for the appearance of movement [1,2,4,6-13]. In 10-day-old chick embryos stimulation of the reticular gigantocellular nucleus (RGCN) initiates locomotor activity [12,15]. Electrical activity of RGCN and its relationship with spontaneous motor activity during ontogeny was not studied. Little is known about the role of proprioceptive impulses in these relationships. It should be emphasized that proprioceptive impulses serve as a feedback necessary to perform movements.

Here we studied the relationship between electrical activity of RGCN and motor activity of the embryo and evaluated the role of proprioceptive impulses in the induction of bioelectric activity in RGCN.

MATERIALS AND METHODS

Experiments were performed on White Leghorn chick embryos ($n=25$) on days 16-19 of incubation. Extracellular neuronal activity in RGCN was recorded using a stain-steel microelectrode (tip diameter $\leq 2 \mu$). The reference electrode was fixed in the embryo beak.

Recording was performed according to coordinates of A. Tienhoven and L. P. Juhas [14]. Localization of the electrode in RGCN was verified histologically. The zone of the introduced microelectrode was electrocoagulated. Electromyogram of the gastrocnemius muscle was recorded with stain-steel bipolar electrodes (inter-electrode distance 1 mm). Electromyogram and neuronal activity were recorded simultaneously using an oscillograph. Proprioceptive impulses were blocked with myorelaxin (0.1-0.2 ml, 1×10^{-4}) to study proper activity of RGCN neurons. Myorelaxin produced a rapidly developed and short-term blocking effect on nerve-muscle transmission (15-20 min).

RESULTS

Periodic fluctuations of bioelectric activity were observed in the reticular formation of the medulla oblongata during various periods. Bioelectric activity of RGCN and electromyogram reflect locomotor activity, which appears as periodic spindle bursts [8]. On day 16 of incubation they were arranged in trains, while on day 18 single bursts prevailed. Age-related changes occurred simultaneously in both structures (Fig. 1). Synchronization of activity increased with age. The percent of coincidence over the specified period in-

I. M. Sechenov Institute of Evolutional Physiology and Biochemistry, St. Petersburg

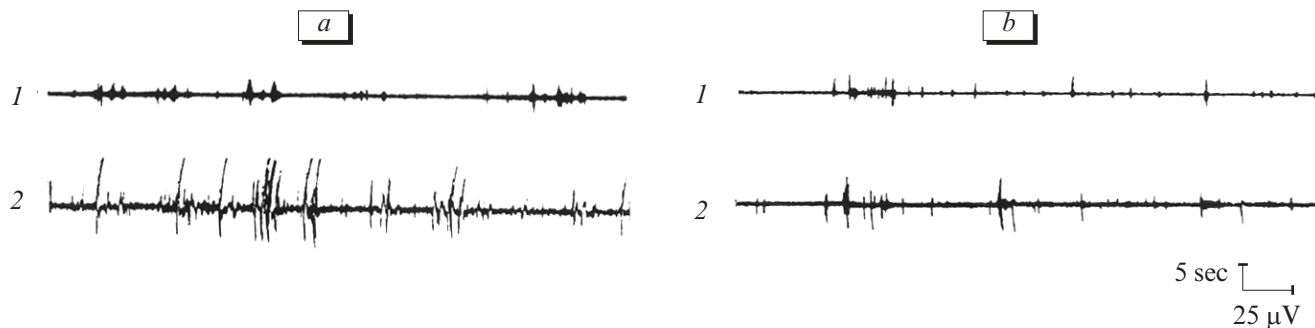


Fig 1. Parallel recording of bioelectric activity in the reticular gigantocellular nucleus (RGCN) and motor activity of chick embryo. Incubation for 16 (a) and 19 days (b). Activity of RGCN (1) and motor activity (2).



Fig. 2. Neuronal activity in RGCN before (a) and 12 min after administration of myorelaxin (b). Embryo: 17 days.

creased from 40 to 85% (correlation coefficient 0.6-0.8). RGCN is probably the major source of supraspinal influences regulating the appearance and progression of motor activity, which increases in the moment of hatching. Proprioceptive impulses from contracting muscles can be one of the factors that induce bioelectric activity in RGCN. Published data show that proprioceptive impulses pass from the cerebellum to RGCN starting from days 6-7 [1,10].

Myorelaxin produced a progressive decrease in motor activity. Movements were completely blocked 10-15 min after treatment. These changes were accompanied by a decrease in activity of RGCN. The total number of bursts decreased, while interspike intervals markedly increased (from 10 to 25 sec). The amplitude of brain potentials decreased (Fig. 2). Myorelaxin-produced changes in myoelectric activity of RGCN are consistent with published data that sensory afferent impulses from the locomotor apparatus play an important role in electrical activity of the brain, which is well developed in 14-day-old embryos [1,5]. In intact embryos the general spindle burst of bioelectric activity was often preceded by short bursts of low amplitude. Similar preceding bursts in activity of RGCN were observed during afferent stimulation of this structure [3]. After treatment with myorelaxin we revealed not only the decrease in bioelectric activity, but also disappearance of short preceding potentials. The baseline activity of RGCN was "exhausted" (Figs. 2 and 3). Preceding low-amplitude potentials were probably related to proprioceptive afferentation from RGCN neurons. A close relationship exists between motor and bioelectric activity of RGCN in chick embryo starting from days 18-19 of embryogenesis. Probably, RGCN

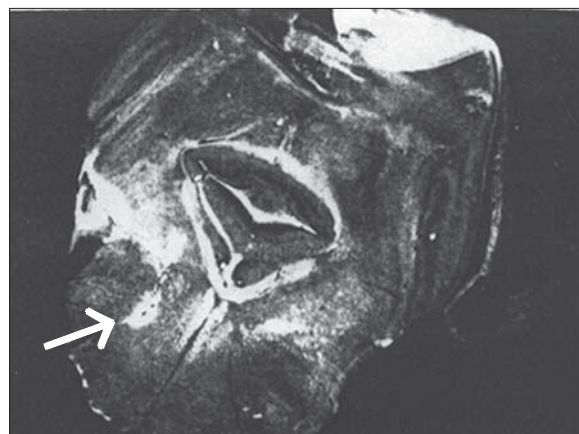


Fig. 3. Cross-section of the medulla oblongata from 16-day-old chick embryo. Arrow: localization of microelectrode. Frozen section, $\times 10$.

is the major source of supraspinal influences. The strength of impulses increases by the end of embryogenesis. Blockade of proprioceptive impulses with myorelaxin decreases bioelectric activity of RGCN. These results indicate that proprioception has an activating effect.

REFERENCES

1. O. V. Bogdanov, *Functional Embryogenesis of the Brain* [in Russian], Leningrad (1978).
2. N. G. Gladkovich, *Neuronal Mechanisms of the Developing Brain* [in Russian], Moscow (1979).
3. Yu. P. Limanskii, *Fiziol. Zh. SSSR*, **51**, No. 6, 671-676 (1961).
4. A. Bekoff, *Int. J. Dev. Neurosci.*, **19**, 155-160 (2001).
5. N. S. Bradley and C. Sebelksi, *J. Neurophysiol.*, **83**, No. 1, 431-440 (2000).
6. J. C. Glover and G. Petursclottir, *J. Comp. Neurol.*, **270**, No. 1, 25-38 (1988).

7. J. C. Glover and G. Petursclottir, *J. Neurobiol.*, **22**, No. 4, 28-35 (1991).
 8. S. Grillner, *Curr. Opin. Neurobiol.*, **9**, 663-669 (1999).
 9. R. Guglielmone and G. Corveti, *Cell Tissue Res.*, **300**, No. 1, 213-376 (2000).
 10. A. Lopez-Raman and J. A. Armengol, *Neurosci. Res.*, **25**, No. 1, 33-40 (1996).
 11. A. A. Sharp, E. Ma, and A. Bekoff, *J. Neurophysiol.*, **82**, No. 5, 2406-2414 (1999).
 12. G. N. Sholomenko and M. J. O'Donovan, *Ibid.*, **73**, No. 3, 1223-1233 (1995).
 13. K. Tan and N. M. Le Douarin, *Anat. Embryol.*, **183**, No. 4, 321-343 (1991).
 14. A. Van Tienhoven and L. P. Juhasz, *J. Comp. Neurol.*, **118**, 185-197 (1962).
 15. J. T. Valenzuela, S. J. Hasan, and J. D. Steeres, *Dev. Brain Res.*, **56**, 13-18 (1990).
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