

CHANGES IN ELECTRICAL ACTIVITY AND REFLEX EXCITABILITY OF THE SPINAL CORD UNDER THE INFLUENCE OF MANGANESE

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Injection of manganese chloride (0.01–1 mg/kg) into the general circulation of anesthetized cats causes changes in the electrical activity of the spinal cord recorded from the center for the flexor muscles.

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Significant morphological and functional changes arising in the central nervous system in manganese poisoning have been described in the literature [4, 5, 6]. Changes in excitability of peripheral and central nervous structures have been reported following administration of manganese compounds to animals in "biological concentrations" [7, 8, 11], and a definite correlation has been established between the course of the fundamental nervous processes and the variations in the content of manganese and other trace elements in the central nervous system [1, 2, 9, 10].

The object of this investigation was to study the effect of manganese on the electrical activity and reflex excitability of the spinal cord.

EXPERIMENTAL METHOD

Experiments were conducted on 52 cats anesthetized with chloralose (40 mg/kg) and Nembutal (15 mg/kg) injected intraperitoneally. To study the electrical activity of the spinal cord potentials were picked up from its dorsal surface by means of platinum needle electrodes and from deep inside the cord (center of the flexor muscles) by means of a focal microelectrode (Nichrome wire, 30 μ in diameter, with glass insulation). Mono- and polysynaptic responses were recorded from the central part of the divided deep peroneal nerve. Test stimulation, in the form of single square pulses of current, was applied to the central end of the divided dorsal root of L_6 from an electronic stimulator with radiofrequency output. An ac amplifier and CRO were used for recording.

Solutions of manganese chloride (0.0001–0.01 M) were injected into the femoral vein of the cats in doses of 0.01–1 mg of the pure metal per kg body weight [3], and were also applied locally to the place where the potentials were detected.

EXPERIMENTAL RESULTS

After intravenous injection of manganese chloride in a dose of 0.1–1 mg/kg, no visible changes in amplitude or frequency of the potentials recorded from the dorsal surface of the spinal cord were observed (Fig. 1).

When the microelectrode was buried into the gray matter of the ventral horn of the spinal cord to a depth of 2.0–2.8 mm, in the first 2–3 min after injection of manganese (0.1 mg/kg) the amplitude of the spikes increased appreciably from their background level (Fig. 1, A, B). By the 5th minute the initial amplitude of the potentials was restored (Fig. 1, C); starting with the 15th–20th minute the amplitude of the potentials again increased and remained high for 20–30 min (Fig. 1, D, E). By the 60th–90th minute the electrical activity had gradually returned to its initial background level (Fig. 1, F). The amplitude of the potentials recorded from the center for the flexor muscles was increased after administration of manganese in a dose of 1 mg/kg for the first 1–3 min, but later (after the 5th minute) it fell below the background level. By the 20th–30th minute the electrical activity had returned to its initial level.

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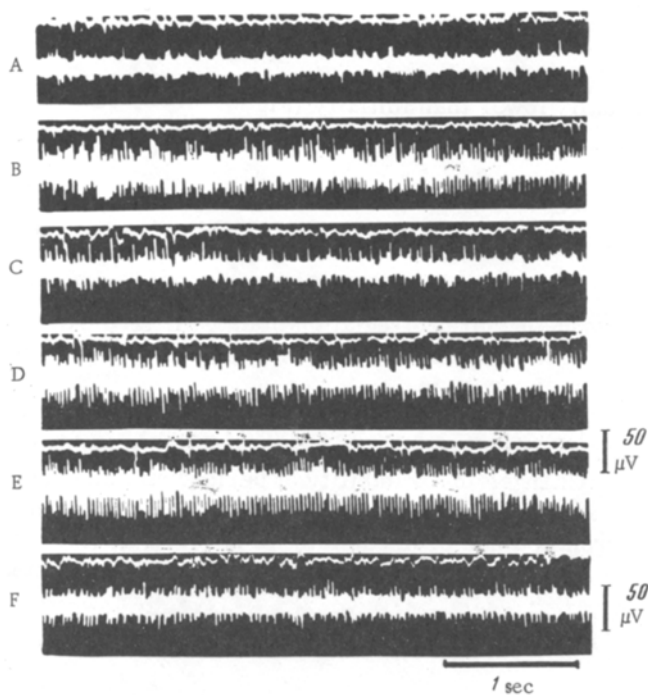


Fig. 1. Effect of manganese (0.1 mg/kg) on spontaneous electrical activity of the cat's spinal cord. A) Initial background; B-F) 30 sec and 5, 15, 30, and 60 min respectively after intravenous injection of manganese. Significance of curves (from top to bottom): potentials recorded from dorsal surface of spinal cord; potentials recorded from micro-electrode buried in region of motor nuclei of tibialis muscle.

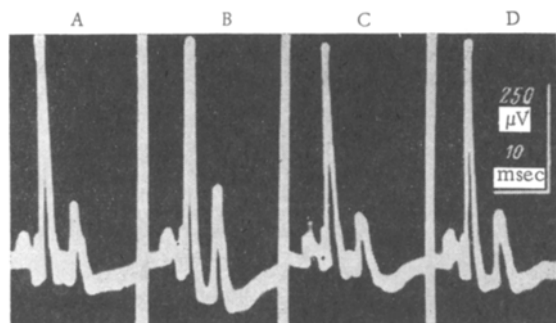


Fig. 3. Changes in mono- and polysynaptic reflex discharges after application of manganese chloride solution to dorsal surface of a cat's spinal cord. A) Initial background; B, C) 1 and 20 min respectively after application of manganese solution; D) 10 min after rinsing surface of spinal cord with physiological saline.

end of the experiment (Fig. 2, I, D, E). The polysynaptic reflex discharges (PRD) increased after the 30th second (Fig. 2, I, A, B, C, D), but from the 40th minute until the end of the experiment they differed only slightly from the initial indices, or were a little higher (Fig. 2, I, E). After injection of manganese in a

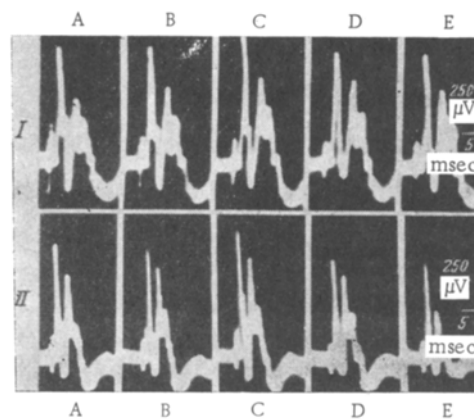


Fig. 2. Changes in mono- and polysynaptic discharges of the cat's spinal cord after injection of manganese (0.1-1 mg/kg). I) A: initial background B-E: 30 sec and 10, 30, and 60 min respectively after intravenous injection of manganese in a dose of 0.1 mg/kg. II) A: initial background; B-E: 30 sec and 10, 30, and 60 min respectively after intravenous injection of manganese in a dose of 1 mg/kg.

Experiments on spinal animals, in which the cord was preliminarily divided under deep ether anesthesia at the level T_1 - T_2 , showed that intravenous injection of manganese caused the same changes in electrical activity of the spinal cord as in animals with an intact nervous system.

After application of manganese chloride solution to the dorsal surface of the spinal cord (for a period of 30-45 min), in the first 2-3 min the amplitude of the potentials recorded from the flexor center increased by 30%. By the 5th minute the amplitude of the potentials had returned to its initial level, but after the 6th minute it increased again and remained high for 20-25 min. After the surface of the spinal cord had been washed with physiological saline, no appreciable changes in electrical activity were observed.

The state of the spinal centers after injection of magnesium chloride into the general circulation was judged from recordings of mono- and polysynaptic spinal cord reflexes.

Intravenous injection of manganese chloride in a dose of 0.01-0.1 mg/kg caused an initial increase in the amplitude of the monosynaptic reflex discharge (MRD) (Fig. 2, I, A, B, C). Later, starting with the 10th-20th minute, the amplitude of the MRD fell slightly and remained low until the

dose of 1 mg/kg the amplitude of the MRD at first fell slightly (Fig. 2, II, A, B), but by the 10th minute they were higher than initially (Fig. 2, II, C). After the 30th-40th minute the amplitude of the MRD again fell, and by the end of the experimental it was 60-70% of its initial value (Fig. 2, II, D, E). Under these conditions the polysynaptic responses were usually increased after the 30th second (Fig. 2, II, A-C) but by the end of the experiment the amplitude of the PRD was usually lower than initially (Fig. 2, II, D, E).

Experiments on spinal animals showed that the MRD increased in amplitude in the first 30 min after injection of manganese, but after the 30th-45th minute they decreased slightly. The amplitude of the PRD also increased after the 30th second to reach a maximum by the 20th-30th minute. After the 30th-40th min the increase in PRD was replaced by a decrease in their amplitude (by 30-40%).

The study of the local action of magnesium chloride solutions on the dorsal surface of the spinal cord showed that the MRD underwent no appreciable changes throughout the period of action of manganese (30-45 min) or they were very slightly depressed (Fig. 3, A, B). After application of manganese solution the PRD were increased after the 1st minute (Fig. 3, B), returning to their initial level only by the 20th minute (Fig. 3, C). After the surface of the cord had been washed with physiological saline no visible change took place in the amplitude of the MRD and PRD (Fig. 3, D).

In a series of control experiments (intravenous injection of 0.9% sodium chloride solution) no appreciable changes were observed in the background electrical activity or in the mono- and polysynaptic reflex responses of the spinal cord.

The results obtained demonstrate that injection of manganese chloride into the general circulation of an animal in a dose of 0.01-0.1 mg/kg evokes an initial increase in the background electrical activity of the spinal cord, recorded from the center for the flexor muscles, and an increase in the amplitude of the mono- and polysynaptic reflex responses, followed by a return to their initial level. It might be considered that these changes are due to the facilitatory influence of suprasegmental portions of the central nervous system on the spinal cord. However, the results of experiments on spinal animals showed that injection of manganese under such conditions caused changes in the electrical responses identical with those obtained in animals with an intact nervous system. In addition, no significant differences were found in the direction of the changes in the electrical responses when manganese solution was applied to the dorsal surface of the spinal cord. The changes in the electrical responses of the spinal cord are evidently dependent principally on the direct influence of manganese salts on its functional state, and they are presumably associated with stimulation of oxido-reductive processes taking place after injection of manganese [12].

At the same time it is worth noting that the polysynaptic reflexes were modified (increased in amplitude) to a more marked degree than monosynaptic. This indicates that manganese ions have a greater influence on the interneurons than on the motoneurons of the spinal cord. Injection of large doses of manganese (1 mg/kg) causes, after the initial stimulation of electrical activity and reflex responses of the spinal cord, a fairly prolonged depression of these functions. In this case manganese may evidently give rise to functional changes in spinal cord activity of a parabolic character.

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