SOME DATA ON INTESTINAL PARESIS
IN EXPERIMENTAL PERITONITIS

O. S. Kochnev

Department of Normal Physiology (Head — Prof. I. N. Volkova)
of the Kazan Medical Institute
(Presented by Active Member of the Akad. Med. Nauk SSSR A. V. Lebedinskii)
Translated from Byulleten' Eksperimental'noi Biologii i Meditsiny, Vol. 52, No. 10,
pp. 54-57, October, 1961.
Original article submitted September 24, 1960.

Until now investigators have held divergent opinions in regard to the reasons for intestinal paresis in peritonitis. A number of authors connect the drop in the tonus of the intestinal musculature, which is seen in this disease process, with a disturbance of the activity of the vegetative nervous system, innervating the intestine [1, 8, 11, 12]. However, in the opinion of some investigators, the parasympathetic innervation is depressed [1, 11], while others believe the reason for disruption of the intestinal motor activity lies in excitation of the sympathetic nervous system [6], or, on the contrary, in its depression [8, 10, 12].

In view of this, we decided to investigate the influence of the vegetative nervous system on the motor activity of the intestine, and to determine the effect of stimulating the posterior spinal roots at different intervals in the development of experimental peritonitis. In order to judge the degree and character of the disturbance in activity of the vegetative innervation, we also determined the concentration of neuromediators (acetylcholine, adrenalin) in the blood, and the serum cholinesterase activity, since it is known from the works of D. E. Al'pern [2] and other authors [3, 7], that the concentration of neurohumoral factors in the blood reflects the level of synthesis of chemical mediators in the organism, and thus, the functional state of different divisions of the vegetative nervous system.

EXPERIMENTAL METHOD

The experiments were carried out on dogs. For acetylcholine determinations we drew 5 ml of blood from the v. saphena into a syringe containing 5 ml of a proserine solution in a concentration of $5 \times 10^{-4}$. The blood was defibrinated. The concentration of acetylcholine in the blood was determined according to the method of Corsten [11], on an isolated frog lung, whose contraction occurs at a dilution of acetylcholine of $1 \times 10^{-20}$. Determination of the serum cholinesterase activity was performed by the titration method of T. V. Pravdich-Neminskaya [7]. To determine the amount of adrenalin in the blood we used the method of luminescence analysis, following the modification described by K. V. Lebedev and S. V. Senkevich [5]. The adrenalin was determined in the blood plasma, where it is contained in a greater quantity than in the serum. Blood for this determination was also drawn from the v. saphena, this time collecting 3 ml into a syringe containing 1 ml of a 2% sodium citrate solution. To bring out the fluorescence of the adrenalin, the plasma obtained from the citrated blood was radiated with a mercury-quartz lamp. The fluorescing intensity of the plasma was photometrically compared to the luminescence of a standard solution of pharmacological adrenalin (5 micrograms/ml).

Peritonitis was induced in the dogs by injecting 0.4-0.6 ml of feces (30% dilution) per kg of body weight into the peritoneal cavity.

Examination of the blood for acetylcholine, cholinesterase, and adrenalin was carried out before injection of the feces, and again 2 hours after its injection, and on the following 1-12 days if the animal survived.

In a series of experiments at different intervals in the development of the peritonitis, we recorded the motor activity of the intestine under morphine-hexanol narcosis by inserting a rubber balloon into the lumen of the ileum and connecting it to a recording system (water manometer, Marey capsule). The respiratory movements were simultaneously registered. The cervical portion of the vagus, the II-III spinal roots, and the splanchnic nerves were dissected out. Stimulation of the nerves was performed with an induction current from a sliding coil, fed by a storage cell (2.5 v).
**EXPERIMENTAL RESULTS**

The level of acetylcholine in the experimental dogs ranged from $2 \times 10^{-4}$ to $2 \times 10^{-6}$ and the concentration of serum cholinesterase was an average of 25.8%, ranging from 18.2 to 34.5%. The concentration of adrenalin in the blood was an average of 1.4 micrograms, ranging from 0.85 to 2.7 micrograms/ml.

Injection of the peritoneal irritant into the dogs caused an extremely marked excitation within 5-10 minutes, accompanied by violent movements and screaming. Then the animals became constrained, laid down, stood unwillingly, and moved slowly and carefully. Vomiting occurred in the majority of them. Respiration quickened and became more superficial; the pulse also increased in rate, and was less full. The abdomen was found to be markedly tense and tender to palpation. In the following days the dogs remained in a depressed state, and they accepted little food, doing so reluctantly. The