THE INFLUENCE OF THE NERVOUS SYSTEM ON THE COMPOSITION OF THE BLOOD

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At the basis of the contacts formed by an organism with its environment a considerable role is played by residual reactions of the nervous system. Academician A. D. Speranskii has demonstrated the possibility of reproducing pathological processes using the principle of trace reactions. He showed [9] that organs of the hemapoietic system are well innervated. This made possible a very important hypothesis concerning the influence of the nervous system on the blood-forming organs. The studies of A. D. Speranskii and others made it important to analyze the residual reactions in the nervous system during some forms of anemia.

The present study is an attempt to clarify the role played by the nervous system in the development and changes produced in the blood by phenylhydrazine poisoning.

We based this study on the statement that this chemical blood-destroying agent has the capacity to "produce a special form of nervous stimulation." Residual reactions during regeneration of the red blood have been observed by a whole series of workers [1, 2, 4, 8].

EXPERIMENTAL METHODS

In our studies the basic pathological process was produced by the introduction of an acid salt of phenylhydrazine, 0.5-1 ml of a 2% solution per 1 kg wt of animal being used. The phenylhydrazine was continued until the appearance of a pronounced anemia after which the blood was studied until the moment of normalization.

It became important to determine whether the blood picture characteristic for phenylhydrazine poisoning could be reproduced by using a new stimulant of the nervous system. As such a factor, we chose "towing." As is known, this method was suggested by A. D. Speranskii in 1935; with the aid of this procedure he was able to produce relapses of previously existing nerve dystrophic processes.

In our studies the first changes in the blood we investigated were those occurring after the infliction of trauma to the nervous system. To do this, we took 5 dogs and subjected them to "towing." When such experiments were performed, changes were observed both in the white and the red blood cell counts.

In the majority of instances, within 30 minutes of beginning the "towing," there was observed a small but constant rise in the hemoglobin, fluctuating between 3 and 10% and averaging 5%. There was observed a corresponding change in the r.b.c. count. By the end of the first day the hemoglobin and the erythrocyte number returned to original levels.

Similar alterations were seen in the leucocytes. Within 15 minutes the leucocyte count rose to 14,420, while within 15 more minutes it reached 20,800 per cc. By the 2nd day the count fell. The leucocytic reaction ended by the 3rd to 5th day, the original levels being restored. "Towing" also provoked changes in the number of reticulocytes (Fig. 1).

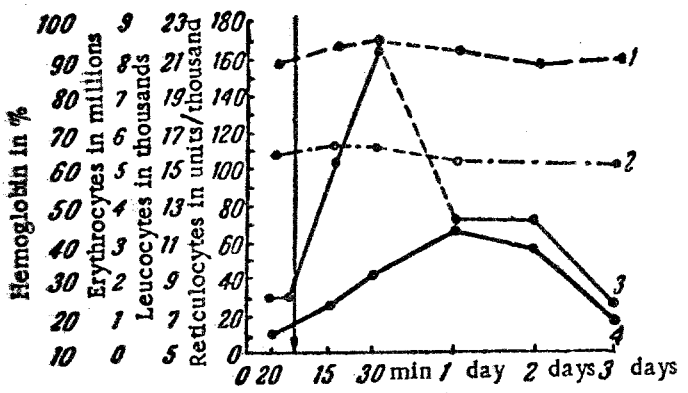

Fig. 1. Changes in some of the hematological indicators produced by "towing" (average of 5 experiments).
1) Hemoglobin; 2) erythrocytes; 3) leucocytes; 4) reticulocytes.
↑ "towing."

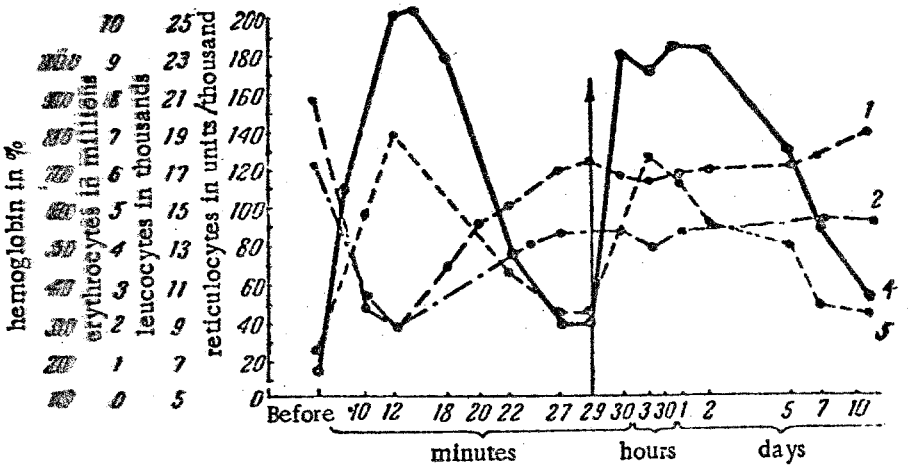

Fig. 2. Changes in some of the hematological indicators after "towing" (average of 8 experiments).
1) Hemoglobin; 2) erythrocytes; 3) leucocytes; 4) reticulocytes. ↑ "towing."

When examining blood smears, we observed that within 30 minutes after "towing," in spite of the leucocytosis, the leucocytic picture remained unchanged. This led to the conclusion that the number of leucocytes grew now because of new formation but as a result of blood redistribution [3, 10, 7].

The results obtained after "towing" gave us a basis of comparison for our second basic series of experiments.

In our experiments the hemotoxic anemia resulting from the phenylhydrazine poisoning usually developed by the 28th to 35th day. By that time the hemoglobin usually dropped to 27-29% (Fig. 2).

In all instances the greatest rapidity in the decrease in hemoglobin was noted in the first 5-7 days, after which the rapidity of the fall of the hemoglobin slackened somewhat.

Corresponding changes in the r.b.c. counts paralleled the hemoglobin fluctuations (Fig. 2). It is important to note that by the second or third day after the poisoning the regenerative capacity of the bone marrow became increased. In the peripheral blood there appeared normoblasts, which then regularly increased in number. At the same time there was a sharp rise in the reticulocyte count.

With such an experimental procedure there is observed a gradual increase in hemoglobin and the erythrocyte count. Usually, 10-12 days and more often not until 15-17 days after stopping the administration of the phenyl-

hydrazine, the animals emerged from their anemic state. By that time in some instances, the indicators have not all returned to base level and the hemoglobin content may be 10-12% below the original reading. However, the reticulocyte count dropped quidkly and remained but little above base level (Fig. 2).

As far as the leucocyte counts were concerned, phenylhydrazine poisoning produced a pronounced leucocytosis with a shift to the left. In the peripheral blood, starting with 3rd to 9th days, there was a rise of up to 7-9% in the polys and up to 3-4% in juvenile neutrophils.

After the cessation of the poisoning, the leucocyte count dropped, reaching about base level.

Thus, after a lapse of 39 to 46 days following the poisoning of the animals with phenylhydrazine, there ensued a complete restoration of the hematological indicators. This gave us the opportunity to administer the "second blow" by means of "towing." After the "towing" we observed the entire course of the changes previously noted after the phenylhydrazine poisoning. There was a difference in that the maximal reticulocyte count did not reach the dimensions attained after the phenylhydrazine poisoning. However, when compared with the control "towing," there were both qualitative and quantitative differences. "Towing" alone raised the reticulocyte count 4.1-5.8% while "towing" after a preliminary background of phenylhydrazine poisoning caused a reticulocytosis of 17.8-18.2%. Thus, "towing" is shown to be a stimulant which, to a certain extent, can reproduce the blood picture of phenylhydrazine poisoning.

The experiments just described demonstrate that blood changes can be produced which resemble the picture of phenylhydrazine poisoning. The results we have obtained are interpreted by us as being the consequence of the nervous system retaining traces of previous stimuli.

Our experimental data seem to help explain up to a certain point some of the relapses seen during blood ailments as observed in the clinic. Not infrequently, a nonspecific stimulant such as grippe or psychic trauma is capable of causing a relapse in such pathological processes as pernicious anemia or chlorosis.

SUMMARY

"Followed-up - after-discharge reactions" were studied in the nervous system of laboratory animals in experimental anemia induced by phenylhydrazine. The method of the "second blow" with "towing" (Speranskii) was employed. It was possible to reproduce, to a certain degree, the blood picture characteristic of phenylhydrazine poisoning at the time when the blood picture almost reached its initial stage. It could particularly be reproduced in the line of the changes of processes of regeneration.

The data which was thus obtained gave theoretical foundation to these phenomena, which are sometimes met in clinical practice as relapses of certain diseases of the blood system.

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* In Russian.