MOTILITY OF THE GUT AND EVACUATION IN EXPERIMENTAL LEAD POISONING

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A variety of interpretations have been made of the pathological changes in the gut caused by lead poisoning. Most of them are related to the secretory function of the gastric glands [4-7]. However, besides disturbance of secretion, more frequently there are changes in gastric and intestinal motility. The few investigations in this direction give a very incomplete account of the damage to the gut and its pathogenesis [1, 3]. They have been based on clinical observations, which has appreciably limited the scope of the study.

What is required, therefore, is a systematic experimental study of the various forms of activity of the gut in lead poisoning.

The present work is a study of the motility of the gut and evacuation in animals suffering from lead poisoning; use has been made of x-rays.

EXPERIMENTAL METHOD

Experiments were carried out on eight dogs with acute, subacute, or chronic lead poisoning. Altogether 102 experiments were performed.

The experiment was begun while the stomach was empty, 18-20 h after the last meal. As food stimulus some of the animals received milk jelly, and others meat paste; both were mixed with 30-50 g of barium sulfate. We measured the following quantitities: time at which the food left the stomach, time for complete emptying of the stomach, time for the passage of the first portion of the food along the small intestine (from the start of evacuation from the stomach to the entrance into the large intestine); time for the passage of the last portion of food along the small intestine (from the time at which the stomach had emptied completely to the time of the complete emptying of the small intestine). In some animals we were also able to determine the time for the passage of food through the large intestine. Observations were made in 5-6 experiments before poisoning, to establish the normal times, as well as after poisoning, which was usually carried out once per week. Observations were continued for from 1 to 5 months. The animals received 1, 2, or 3% lead acetate given as 1 mg/kg; the solution was added to the milk.

The extent of the poisoning was determined from the general condition and behavior (weight, activity, and appetite) and from a blood analysis. In all the animals, as poisoning progressed, the typical blood changes developed: first there was an increase in the number of reticulocytes (up to 12-40/1000 erythrocytes), then erythrocytes showing a granular basophilia appeared (up to 2-5/10,000 erythrocytes), there was a certain degree of leucocytosis with a shift to the left, etc. The reduction in hemoglobin was of the order of 1-2 g%, and the fall in the red cell count was also insignificant.

The clinical signs were reduction in appetite, exhaustion, and constipation alternating occasionally with diarrhea.

EXPERIMENTAL RESULTS

The earliest changes in motility and evacuation were shown in the delayed emptying of the stomach; there may be recovery if the poisoning is slight, or the condition may become more marked in more severe cases. Before poisoning, after milk jelly had been eaten, in most of the dogs the stomach emptied in 2-3 min, and only in a few animals

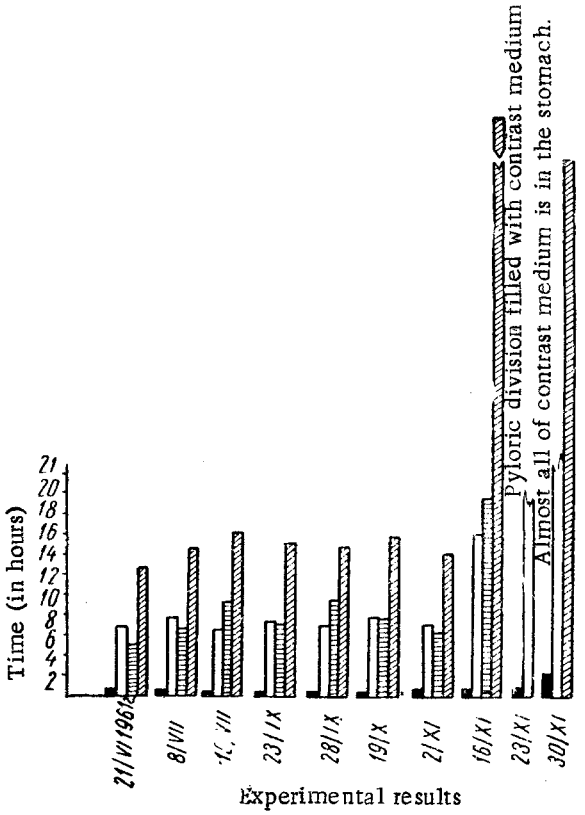

Fig. 1. Change of time for emptying the stomach and small intestine in subacute lead poisoning in the dog Mirnyi. 1) Time for the onset of evacuation of the contrast medium from the stomach; 2) time for the stomach to empty; 3) time for evacuation of the contrast medium from the small intestine; 4) total time for stomach and small intestine to empty. First five experiments before poisoning; remaining four show the effect of lead poisoning.

did it take 10-15 min; by the second week after poisoning, in all the animals the time was increased 3-4 fold. The greatest delay of the onset of evacuation from the stomach was observed in the first days after poisoning; next this varied, and later was again delayed (up to 1 h or more); Fig. 1 shows the considerable delay before complete evacuation occurred.

Before poisoning, and after the animals had eaten milk jelly, the time required for the stomach to empty was 2 h 20 min-3 h 40 min; after meat paste had been taken the time was 5 h 30 min - 7 h.

In the first group of animals (two dogs with acute lead poisoning) by the end of the second week there was a small increase in the time required for the stomach to empty. Later the time returned to within normal limits, but both dogs suddenly died in continuous convulsions.

In the second group of animals (four dogs with subacute lead poisoning) the degree and times of onset of disturbance of motility and evacuation were varied, but there was a common feature. For the first two weeks, after an initial delay, there was recovery to normal times, and not until the second month was there again a delay. The time for which food remained in the stomach was 8-10 times longer than normal. Peristalsis of the stomach was greatly weakened and slowed. X-rays showed that the stomach was dilated (Fig. 1).

In the third group (two dogs with chronic poisoning) for the first month there was no change of evacuation. A delay occurred at the $1\frac{1}{2}$-2nd month, when the time was increased three or more times. Subsequently, the time for complete emptying returned to normal, and though there were some fluctuations it remained essentially unchanged until death. In this group, at times the rate of evacuation was accelerated so that the time was reduced to 55-60% of the previous value.

Thus, in animals with chronic lead poisoning, normal function was restored after some initial suppression of gastric motility (Fig. 3).

As we have already pointed out, the motility of the small intestine was inferred from the time for the passage of the first and last portions of food through it; we attributed greatest significance to the second quantity, because in the opinion of many investigators [2] this time is the principal index describing motility of the small intestine.

Our observations made before poisoning and at various times afterwards showed that in severe lead poisoning there is a delay in the movement of food along the small intestine, but it occurs much later and is much less well shown than are the disturbances in gastric motility (Fig. 3).

Of the eight animals studied, in four with subacute and in two with chronic lead poisoning the time for which the food remained in the small intestine was increased. In the small intestine the functional disturbance took the form of stasis, and in the large intestine there was a spastic haustration. During the period (which occurred in some animals) of motility during temporary remission of the condition, the reverse situation was observed: intestinal motility became normal or even accelerated, while evacuation from the stomach was still long delayed.

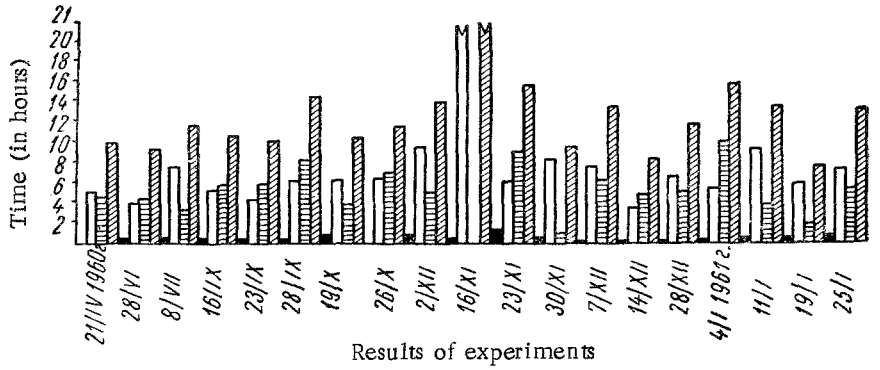

Fig. 2. Change in the time for the stomach and small intestine to empty; dog Jack, lead poisoning. The first four experiments show initial condition, the remainder the effect of lead poisoning. Indications are the same as in Fig. 1.

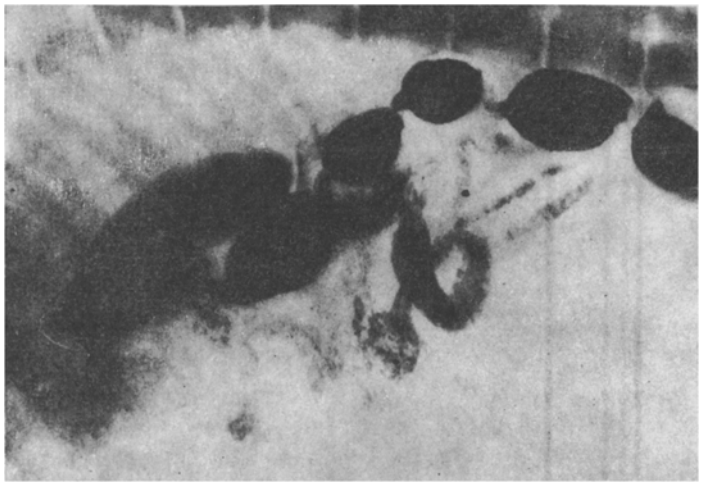

Fig. 3. X-ray picture of stomach of dog Mirnyi 30 h after it had taken a barium meal, on the 42nd day after receiving poison. Stomach is free from contrast medium, which fills the large intestine (strongly haustrated) and some of the small intestine.

SUMMARY

X-rays were used to study motility of the gut and evacuation in animals poisoned with lead. Experiments were made on eight dogs with acute, subacute, or chronic lead poisoning. It was found that the motility of the gut and evacuation was depressed. The first effect was a delay of evacuation from the stomach. In severe poisoning the time was greatly extended. Evacuation from the small intestine was also delayed, but the disturbance was here less marked, and developed later.

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