# LESIONS OF THE GASTRO-INTESTINAL MUCOUS MEMBRANE IN EXPERIMENTAL LEAD POISONING

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Dogs with lead poisoning develop necrotic gastroenterocolitis with involvement of the lining epithelium and the superficial part of the mucous membrane. If the lesion is severe, the deformation and necrosis spread to the gland structures and to their stroma, sometimes reaching to the submucosa. The gland cells which remain intact actively secrete a mucous material.

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In lead poisoning many systems of the body are injured [2-5, 7]. One of the most vulnerable is the digestive system. Morphological data characterizing changes in this system are incomplete and inadequate [1, 5-7]. In previous investigations by one of the authors (A. A. Mambeeva [3]), changes in the function of the intramural nervous apparatus of the gastro-intestinal tract were studied.

The object of the present morphological investigation was to study the course and character of the lesion in the mucous membrane of the gastro-intestinal tract in lead poisoning.

## EXPERIMENTAL METHOD

Lead acetate was given by mouth to dogs as 1, 3, and 5% solutions of $Pb(CH_3COO)_2 \cdot 3H_2O$ in doses of 1 ml/kg body weight daily. The solution was given with milk. The histological investigation was carried out on ten dogs (one control and nine experimental). The stomach (fundus, body, pyloric region) and the proximal and distal portions of the small and large intestine were investigated. The material was fixed in formalin. Pieces of tissue were embedded in celloidin–paraffin wax. Sections were stained, with toluidine blue and orange by the Dominici-Kedrovskii method, with carmine by Best's method, mucicarmine by Mayer's method, and picrofuchsin by Van Gieson's method.

## EXPERIMENTAL RESULTS

Depending on the doses received the experimental animals developed a picture of chronic, subacute, and acute poisoning. The dogs receiving the 1% solution gradually developed chronic lead poisoning. These animals survived for between 5 and 9 months. The poisoning was shown by anemia, loss of weight, vomiting, and convulsions. More concentrated solutions of lead acetate (3-5%) caused acute and subacute poisoning. The poisoning developed more rapidly and violently. The experimental animals receiving this dose died 1.5-3 months after the beginning of poisoning. The criterion of poisoning in this case was the general state of the animals, their behavior, and their blood picture.

Microscopic investigations showed considerable necrotic changes in the mucous membrane of the gastro-intestinal tract (Fig. 1). In every case the lining epithelium and superficial layer of the mucous membrane were involved. In more severe lesions the process spread to gland structures and to their stroma. Necrosis in some cases reached the submucosa. Later, not only the dead epithelium lining the mucous membrane and crypts but also the necrotic areas of the gland structures in both the stomach and intestine became detached. Erosion and ulcers appeared. The gastric and intestinal mucous membrane became uneven in thickness, and in some places it was considerably thinned. The changes in the stomach were most marked in the pyloric part, and those in the large intestine were more marked than in the small intestine, for the structure of the crypts was completely disorganized and the wall was considerably thin-

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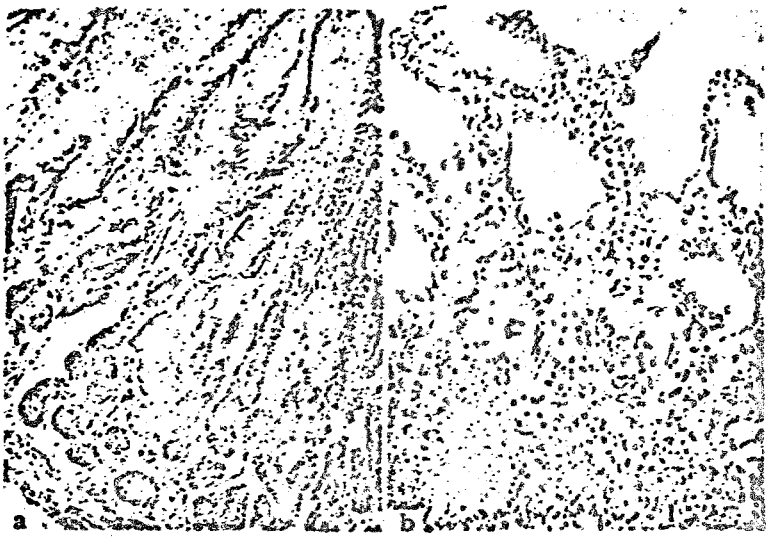


Fig. 1. Necrosis spreading to acinar cells of pyloric glands (a), deformation, death and desquamation of epithelium of crypts in mucous membrane of large intestine (b). a) Stained with Best's carmine, 80 x; b) stained with Mayer's mucicarmine 140 x.

ner. In chronic lead poisoning the necrotic changes appeared much less frequently. They were more superficial than in acute and subacute poisoning and were focal in character. Necrosis was accompanied by deformation of all types of gland cells in the stomach and intestine. The surface epithelium of the stomach was flattened, the reticular structure of the cytoplasm of the chief cells was disturbed, and the granules were less densely distributed.

A considerable decrease in the density of the secretory granules was also found in the cytoplasm of the parietal cells, while their nuclei appeared more enlarged. The prismatic cells of the intestinal epithelium were narrowed, while the goblet cells had become lengthened, increased or decreased in size, and modified in shape. Deformation and compression of the glandular cells and of the glands as a whole were not due entirely to the toxic action of the lead compounds but also to swelling and edema of the stroma of the mucous membrane, which were observed along with necrotic processes. At the same time, areas of considerable dilatation of capillaries, filled with blood cells, could be seen. Hemorrhages were also present.

Despite the fact that necrosis was taking place in the mucous membrane, with thining of its wall, edema, and deformation of the glandular cells, the cells remaining alive in the mucous membrane of the stomach and intestine still continued to secrete actively. The amount of mucous substance secreted from the intact surface and crypt epithelium of the stomach and the goblet cells of the intestine was particularly large. In some cases the dilated lumen of glands could be seen in the form of cystic cavities.

After administration of lead acetate solution to the animals had been discontinued, and the course of the poisoning had become chronic, the mucous membrane recovered. The necrotic areas sloughed. The surface and crypt epithelium and the chief and parietal cells regenerated. However, 79 days after the last dose of lead, complete recovery of the structure of the glands and cells of the mucous membrane of the stomach and intestine had not yet taken place.

The results of this histological investigation demonstrate the considerable development of necrotic gastroenterocolitis [4]. Its appearance is connected with involvement of the intramural nervous apparatus of the wall of the gastro-intestinal tract. The morphological picture of lead poisoning helps to elucidate its clinical manifestations.

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