better results than the other 2 rations. Sodium fluoride when added to the milk, Cu and Fe ration gave results which surpassed those on $KAl(SO_4)_2$ for the first 4 months; then the fluoride exhibited toxicity. Rats on milk, $FeCl_3$, and $CuSO_4$ and the Daniels and Hutton salt mixture, exclusive of NaI, grew satisfactorily and performed equally well as rats on our stock ration. Rats on milk, $FeCl_3$, $CuSO_4$, and $MnSO_4$ performed better than any of the previous lots and even surpassed those on the stock ration. Reproduction on milk, $FeCl_3$, $CuSO_4$, and the Daniels and Hutton salt mixture was very good, during 3 generations studied. The rats on milk, copper, and iron in the first generation reproduced equally well with the animals on the Daniels and Hutton salts, but the mortality of the young was considerably greater in the former case. Furthermore, the weight of the young at birth and weaning time was considerably less in the former case than on the Daniels and Hutton salts. Male and female rats of the first generation on milk, copper, and iron proved fertile for at least 20 months and are still active in this respect. However, rats on this same diet do not reproduce in the second generation. The animals of the second generation failed to reproduce in 8 months; but with the addition of the Daniels and Hutton salt mixture reproduction occurred, and the per cent mortality of the young was low. Infection of the middle ear, *otitis media,* was very prevalent in the second generation rats receiving milk, iron, and copper; but it was not nearly so common in the first generation. Only 2 of the 88 animals on milk, Cu, Fe, and Mn as $MnSO_4$ showed this pathological condition. 6 to 12 rats were employed in each experiment.

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Cardiovascular System in Acute Experimental Peritonitis.

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The purpose of this work is to determine the reaction of the cardiovascular system to experimental peritonitis. Holzbach[1] in his review on circulatory failure in infectious conditions states that functional heart failure appears early in peritonitis, but this opinion

[1] Holzbach, E., *Wurzb. Abhandl. a. d. Gesamtgeb. d. Med.*, 1931, **27**, 1.

is apparently based on clinical observations since no experimental work of this condition is reviewed.

Two types of peritonitis were induced in dogs. Fecal peritonitis was produced in 5 dogs by the intraperitoneal introduction of 5 gm. of small and large bowel feces suspended in 25 cc. of saline. Colon bacillus peritonitis was induced in another set of 5 dogs by the intraperitoneal injection of 50 cc. of a 2% gum tragacanth in saline containing 200 million *B. coli* per cc. In each of the animals blood pressure was obtained prior to and continuously after the onset of peritonitis by insertion into the carotid artery of a cannula which was connected to a mercury manometer and recorded on a kymograph. The animals were under pento-barbital sodium anesthesia. With each animal with peritonitis a normal control dog was run simultaneously. In another set of 4 dogs, 2 with fecal peritonitis and 2 with *B. coli* peritonitis, electrocardiograms were taken prior to and at intervals after the onset of peritonitis.

There was a gradual drop of blood pressure beginning within 15 to 20 minutes after the onset of peritonitis. In one hour the carotid pressure dropped 2 mm. of Hg. The pulse pressure became progressively smaller. The fall was of the systolic pressure, the diastolic being maintained. In 6 hours the carotid pressure dropped 5 mm. of Hg. and an arhythmia appeared. In *B. coli* peritonitis the carotid pressure drop was more rapid but seldom exceeded 4 mm. of Hg. The electrocardiographic study revealed an increase in pulse rate (from 90 to 220 in 4 hours) with an auriculo-ventricular response of 1:1. The ST interval was depressed and the T wave was diphasic. In 6 hours the pulse rate had further increased (to 264) and there was a greater T wave deformity. The changes in the electrocardiograms in fecal and *B. coli* peritonitis were identical and indicated myocardial damage. Histological sections of the peritoneum revealed dilated capillaries engorged with blood. This marked dilatation and engorgement became apparent within one hour after the onset of the infection.

In a former article one of us (B.S.)[2] demonstrated that the cause of death in peritonitis is due to liberation of soluble bacterial toxic substances within the peritoneum. With the observation of dilated and engorged peritoneal capillaries, the cause of the cardiovascular collapse is suggested to be due both to the toxic action of the soluble toxins on the myocardium and a local peritoneal stagnation of blood.

[2] Steinberg, B., PROC. SOC. EXP. BIOL. AND MED., 1930, **28**, 59.