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The Cause of Death in Experimental Acute Diffuse Peritonitis.*

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There are several current conceptions regarding the cause of death in acute diffuse peritonitis. Askanazy[1] considers the intestinal paralysis with dilatation of lymphatics and pressure upon the ganglion cells responsible for death. Lennander[2] agrees with the intestinal paralysis view but holds that the passage of toxins and bacteria through the paralyzed intestinal wall is the causative factor in fatal terminations. Whatever factor the intestinal obstruction plays in the fatal outcome (Orr and Haden[3] do not believe that sufficient evidence has been presented to justify the belief that intestinal obstruction is alone the cause of death) it is of secondary importance. The intestinal paralysis constitutes one of the several complications incident to an acute diffuse peritonitis. Steinberg and Ecker[4] presented evidence in experimental colon bacillus peritonitis that the soluble toxic substance of the bacillus produces death and possibly the coincident complications. Additional evidence that the bacterial toxin is the primary death producing factor has been furnished by Williams,[5] who demonstrated that *B. welchii* toxin is responsible for death in acute diffuse peritonitis from obstruction of the small intestine.

The experiments here reported have been undertaken to obtain further facts of the rôle of the bacterial toxin in acute peritonitis. Diphtheria bacillus was used because it is a definite toxin producer and the antitoxin is readily available. Diffuse peritonitis was pro-

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1 Quoted by Heineke, H., *Deutsches Arch. f. klin. Med.*, 1900-1901, **49**, 429.

2 Quoted in Kaufmann's Pathology by Reimann, **2**, 865.

3 Orr, T. G., and Haden, R. L., *Arch. Surg.*, 1929, **18**, 2159.

4 Steinberg, B., and Ecker, E. E., *J. Exp. Med.*, 1926, **43**, 443.

5 Williams, B. W., *Brit. J. Surg.*, 1926, **14**, 295.

duced by the intraperitoneal introduction into dogs of diphtheria bacilli suspended in a 2½% suspension of gum tragacanth in saline. It has been demonstrated[6] that bacteria suspended in gum tragacanth invariably produce a fatal peritonitis.

Nineteen dogs were injected. Each animal received intraperitoneally the washings of 3 slants of a 24-hour diphtheria bacillus culture suspended in 40 cc. of a 2½% suspension of gum tragacanth. The injection was made with syringe and needle through the abdominal wall. Ten of these 19 dogs were given 20,000 units of diphtheria antitoxin each by routes indicated on the chart. The antitoxin was given a few minutes after the production of peritonitis. The 9 dogs *without* antitoxin died in within 18 to 48 hours. All of the 9 dogs had a marked fibrino-hemorrhagic peritonitis. The 10 dogs that received diphtheria antitoxin survived. They were slightly ill during the first 18 hours but after that they were apparently well. One of the 10 dogs that survived was killed 56 hours after production of peritonitis. The animal had a moderate fibrinopurulent peritonitis. The result of further experiments with a detailed description of the differences in the type of peritonitis in the passively immunized and in the non-immunized dogs will be reported elsewhere at a later date.

TABLE I.
3 slants of diphtheria bacilli in 2½% gum tragacanth injected intraperitoneally.

No. of Dogs	Diphtheria Antitoxin Given	Route of Antitoxin Administered	Outcome
9	None	———	Died 18 to 48 hours with fibrino hemorrhagic peritonitis.
6	20,000 units	Intravenously	Survived
2	20,000 "	Intramuscularly	"
1	20,000 "	Intraperitoneally	"
1	20,000 "	Subcutaneously	"

Summary: Further evidence is submitted that death in acute diffuse peritonitis under the conditions of these experiments is due to passage of a bacterial toxin from the peritoneal cavity. The toxin is elaborated by the introduced bacteria within the peritoneal cavity. Death can be prevented by the administration of the corresponding antitoxin. The local inflammatory reaction *per se,* apparently, has no effect on the survival or death of the animal.

[6] Steinberg, B., and Goldblatt, H., *Arch. Int. Med.,* 1927, **39,** 446.