facts have an important bearing on any theory of acid causation of edema.

Our experiments do not permit us to deny that acids may be influential factors in the causation of edematous processes. Our results emphasize the fact, however, that the acids which may be produced in, or that are carried into, tissues tend to unite there with non-colloidal basic radicals and with dissolved colloids before combining with suspended colloids. The chemical means and excretory processes by which *living* protoplasm maintains a state of reaction-constancy cannot easily be overcome. In Fischer's published experiments on the bloating effects of acids, *large* excesses of *free* acid were present in all but a few cases. Would Fischer contend that edematous tissues contain *free* acid?

We feel that acids are not the only causes of colloidal water absorption in edema. Results obtained by Berg and Gies[1] several years ago indicate that *enzymes* facilitate any such influence that acids, whether free or combined, may exert; and vice versa. Fischer himself alludes, "in passing" (p. 109), to a result in harmony with that view. The italicized portion of the foregoing quotation from Fischer's book is broad enough to include enzyme influences and all other contributory factors. Experiments along these lines are still in progress.

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The relation of the toxic dose of horse serum to the protective dose of atropin in anaphylaxis.

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This study was prompted by the publications of Auer and Lewis,[2] and of Auer,[3] which definitely demonstrated the prophylactic action of atropin sulphat in the asphyxia of immediate anaphylaxis. The results of these writers have been confirmed repeatedly.

[1] Berg and Gies, *Journal of Biological Chemistry*, 1907, ii, pp. 508 and 522.

[2] Auer and Lewis, *Jour. A. M. A.*, 1909, viii, 458; *Jour. Exp. Med.*, 1910, xii, 153; *ibid.*, p. 165.

[3] Auer, *Amer. Jour. of Physiology*, 1910, xxvi, 439.

In our own studies we used guinea pigs averaging about 400 grams in weight and sensitized by subcutaneous administration of 0.05 c.c. horse serum. The atropin was injected intravenously five minutes before the toxic dose of horse serum which also was administered into the jugular vein.

The study shows that as the toxic dose of horse serum is increased the protecting dose of atropin must also be increased, but the increase in protecting dose is not proportionate to that of the horse serum. The curve of protecting dose rises much more sharply than that of horse serum and finally a point is reached where the animal succumbs to the dose of atropin. A 400-gram guinea pig is killed almost instantly by a dose of 0.060 gram atropin.

That the effect of the atropin is physiological and not due to any alkaloidal combination with the toxic fraction of the horse serum is shown by the fact that a mixture of atropin and horse serum incubated at 37° C. and dialyzed for four days killed sensitized animals whereas a control in the same proportions but not dialyzed saved the animals from anaphylactic death.

66 (591)

The effect of specific vaccines in the typhoid of rats and mice.

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Owing to the impossibility of infecting small laboratory animals by feeding with typhoid bacilli, the immunity produced by specific vaccines has always been tested by subcutaneous or intra-peritoneal inoculations of the living culture.

These methods do not produce a disease comparable to human typhoid, but when rats and mice are fed with certain of the paratyphoid group they contract a disease whose pathology does closely resemble it; therefore these were used for the comparative study of the vaccines. The Danysz virus (one of the Gaertner group) was the test organism.

White mice were used in the first series and the vaccines tested were killed cultures, Vaughan's residue, sensitized bacilli (Bes-