No uric acid was formed by incubating these bloods with the purines used with rat blood.

Rat blood then contains enzymes which convert guanine, xanthine, and hypoxanthine into uric acid. The nature of the action suggests the presence of guanase and a very active xanthine oxidase.

Dixon and Keilin[2] reported the inhibition of xanthine oxidase by cyanide. The production of uric acid on incubation of rat blood alone or with guanine or xanthine was inhibited by 0.01 M potassium cyanide. It was of interest to find that the quinimine form of para-amino-phenol reported by Bernheim and Bernheim[3] as an inhibitor for xanthine oxidase also inhibited the formation of uric acid from xanthine by rat blood.

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Vitamin C and Peptone Shock in Dogs.

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Hochwald[1] and others have reported that the administration of Vitamin C to sensitized guinea pigs exercises a protective effect against the anaphylactic reaction produced by the provocative injection of the antigen. Hochwald reported that Vitamin C administration had no protective effect against histamine shock and therefore postulated that the mechanism of the action of Vitamin C in anaphylaxis was to prevent the liberation of histamine rather than to inhibit or inactivate the action of histamine after its liberation. Ungar, Parrot, and Levillain[2] reported some *in vitro* experiments tending to confirm this hypothesis. We were unable to confirm the protective effect of Vitamin C against anaphylaxis in the dog. Since the anaphylactic experiment is, however, quite complex and it is not always possible to discriminate between those agents which may interfere with the antibody-antigen reactions and those which mod-

2 Dixon, M., and Keilin, D., *Proc. Roy. Soc. London*, 1936, **119**B, 159.

3 Bernheim, F., and Bernheim, M. L. C., *J. Biol. Chem.*, 1938, **123**, 307.

1 Hochwald, A., *Z. f. d. ges. exp. Med.*, 1935, **97**, 433.

2 Ungar, G., Parrot, J. L., and Levillain, A., *C. R. Soc. de biol.*, 1937, **125**, 1015.

ify the resultant reaction, it seemed desirable to test the above hypothesis of the mechanism of action of Vitamin C in a histamine liberating reaction that is apparently not dependent upon an antigen-antibody reaction. Such a reaction occurs in peptone shock. We have demonstrated that peptone shock in dogs is accompanied by an explosive liberation of histamine and that the degree of shock is proportional to the amount of histamine liberated.[3] We have, therefore, studied the effect of the prior administration of Vitamin C upon the severity of peptone shock in dogs.

Eleven dogs were anesthetized with ether and sodium barbital and the carotid blood pressure tracings recorded to provide an objective record of the severity of the shock reactions. Cevitamic acid* was injected intravenously in amounts varying from 25 to 100 mg per kilo and after varying intervals of from 15 to 45 minutes an injection of 2 cc per kilo of a 10% solution of a proteose peptone† was made. Definite shock reactions occurred in 10 animals, 2 being rapidly fatal, 5 quite severe, and 3 mild. The distribution of the varying grades of severity of shock was similar with that in a large number of controls.[3] Blood samples were drawn approximately 5 minutes after the peptone injection and the presence of histamine demonstrated in all instances in which the reaction was severe.[3]

It is concluded that the prior administration of Vitamin C to dogs does not protect against peptone shock and that correspondingly it does not prevent the liberation of histamine from the fixed cells of the body into the blood.

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Contradictory reports upon the effect of Vitamin C on the anaphylactic reaction of guinea pigs have appeared. Hochwald,[1]

[3] Dragstedt, C. A., and Mead, F. B., *J. Pharm. and Exp. Ther.*, 1937, **59**, 429.

* We are indebted to Merck and Co. for the Cebione used in these experiments.

† Bacto-Protone-Difco was used in these experiments.

[1] Hochwald, A., *Z. f. d. ges. Exp. Med.*, 1935, **97**, 433.