mortality rate was similar in both groups (see Fig. 1). Anaphylactic shock is not lethal 5 days after sensitisation so that the toxic effect of bradykinin in both groups at this time is probably due to an action of the adjuvant itself. Forty days after treatment (when anaphylactic shock is minimal), bradykinin was not toxic to either group. Konzett (1962) has already reported that the polypeptide was well tolerated by non-sensitised rats in doses of up to 10 mg/kg.

As the jejunum and heart are the tissues most damaged in both bradykinin shock and anaphylactic shock, the evidence suggests that bradykinin plays an important role in anaphylaxis in the rat.

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Modification of anaphylactic shock by mepyramine and ethanolamine

SIR,—It is evident that under different experimental circumstances the modification of anaphylactic shock by mepyramine and ethanolamine gives different answers. Herxheimer & Streseman (1965) found that ethanolamine did not improve the protection afforded by mepyramine to guinea-pigs exposed to an aerosol of antigen solution, whereas in this laboratory ethanolamine substantially improves the protective effect of mepyramine under these circumstances as originally reported by Smith (1961). It has recently been reported by Dawson, Hemsworth, & Stockham (1965) that the sensitivity of guinea-pig ileum to histamine can be influenced by dietary ascorbic acid. Since all guinea-pigs used in these laboratories receive approximately 50 mg of ascorbic acid per day in their drinking water, the discrepancy between my own findings and those of Herxheimer & Streseman might be due to this. Ascorbic acid is known to influence the metabolism and methyl donating capacity of folic acid, and the possibility that ethanolamine is dependent for its anti-anaphylactic activity upon *N*-methylation *in vivo* followed by incorporation into glycerophosphatide has been the subject of experimental investigation here for some time.

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