cholamines from the adrenal glands of guinea pigs.[4] Release of catecholamines by bradykinin should be taken into account in interpreting the actions of bradykinin on respiration and the features of anaphylactic bronchoconstriction in the guinea-pig. For example, this effect probably explains the incompleteness of the bronchoconstriction induced by bradykinin compared with that induced by 5-hydroxytryptamine.[5,6] Likewise, it may explain the bronchodilatation sometimes induced by bradykinin after suppression of its bronchoconstrictor action by nonsteroidal anti-inflammatory drugs.[7] Again, the release by bradykinin of endogenous catecholamines must introduce uncertainty into interpretation of the effects of administered catecholamines on responses to bradykinin.

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8. Role of Bradykinin and Bradykinin-like Substances in the Genesis of Some Types of Cardiovascular Shock. A. P. Corrado (*Dept. of Pharmacology, Faculty of Medicine of Ribeirão Preto, Univ. of São Paulo, Brazil*).

Beraldo[1,2] was the first to suggest an activation of the enzymatic system *in vivo* which releases bradykinin in peptone or anaphylactic shock in dogs where an increase in blood bradykinin was observed. These results were corroborated by Brocklehurst and Lahiri[3] in guinea pig, rabbit, and rat anaphylaxis. A bradykinin-like substance was also detected in the blood of dogs during cardiovascular shock produced by the injection of proteolytic enzymes from bacteria or mammals.[4,5]

Bradykinin (or a bradykinin-like substance) is rapidly inactivated[6] by plasma and tissue kininases.[7–9] Therefore, the values of the plasma levels of the polypeptides released during cardiovascular shock are preferably determined in terms of the precursor bradykininogen.[4,10–15]

By measuring the kininogen under these conditions we have found a very good relationship between the amount of released polypeptide and the severity of the cardiovascular shock produced in dogs in experimental anaphylaxis, acute pancreatitis, and endotoxin shock.

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9. Possible Modes of Kinin Formation in Some Pathological States in Man. V. Eisen and C. A. Keele (*Dept. of Pharmacology, Middlesex Hospital Medical School, London, England*).

We shall discuss three conditions in which plasma kinin formation appears to be of clinical importance.

1. Transfusions of human plasma fractions containing fibrinogen and factor VIII produce in some patients adverse reactions which resemble the effects of bradykinin.[1] Analyses of such fractions[2,3] have shown that they contain plasminogen, preactive factor XII, and a small amount of kininogen. The observed clinical reactions, however, appear to be due to the presence of active plasma kallikrein; 100 Frey units or more may be introduced with some clinical transfusions. Moreover, the fractions contain an enzyme resembling C'1-esterase, which may also cause reactions.

2. Seegmiller[4] has reported that in gout the phagocytosis of urate microcrystals by the leukocytes in synovial fluid leads to kinin forma-