After intravenous injections, physalaemin was only one to six times more active than eledoisin, but after intra-arterial administration it had a much stronger local vasodilating action, particularly when the basal vascular tone was high.

Eledoisin had a powerful vasodilating action on the innervated and acutely denervated vessels supplying the gastrocnemius-plantaris muscle of the dog. Denervation enhanced the peripheral vasodilating activity of vasodilatory drugs.

In the rabbit both physalaemin and eledoisin reduced systemic blood pressure and contracted the uterus *in situ*. Physalaemin was more active on blood pressure and less active than eledoisin on the uterine muscle. Moreover, its oxytocic effect was generally shorter and sometimes irregular.

On the isolated vas deferens of the guinea pig, physalaemin and eledoisin potentiated contractions caused by electrical stimulation of the hypogastric nerve, both showing a direct action on the muscle when higher concentrations were used in the bath.

14. Pharmacologically Active Peptides in Trypanosome Infections. L. G. GOODWIN (*Nuffield Institute of Comparative Medicine, London, England*).

Mice inoculated with a strain of *Trypanosoma brucei*, which causes an acute infection, excrete histamine and pharmacologically active peptide in the urine. Increased histamine and kinin activity is also found in plasma and tissues, especially skin, ears, and feet.[1, 2] Treatment with a trypanocidal drug causes a reduction in active peptide output: if the infection relapses the peptide reappears. Studies are now in progress on chronic trypanosomiasis. Rabbits infected with *T. brucei* show few organisms in the circulating blood but usually die in 30 or more days. Active peptide excretion in the urine occurs in a series of diminishing peaks, which may perhaps be related to the emergence of antigenic variants of the parasite during the course of the infection (Boreham, unpublished). Studies are also being made on the mast cells in tissues which show increased histamine and peptide content during infection.

1. L. G. GOODWIN and W. A. G. RICHARDS, *Brit. J. Pharmacol.* **15**, 152 (1960).
2. W. H. G. RICHARDS, *Brit. J. Pharmacol.* **24**, 124 (1965).

15. The Role of Cathepsins in the Iniactivaton of Plasma Kinins. LOWELL M. GREENBAUM and KEIKO YAMAFUJI (*Dept. of Pharmacology, College of Physicians and Surgeons, Columbia Univ., New York, N.Y., U.S.A.*).

The possibility that intracellular proteinases (cathepsins) may play a role in the inactivation of kallidin, bradykinin, and related kinins is being investigated. Evidence has been presented (*Life Sci.* **4**, 625, 1965; *Fed. Proc.* **24**, 1965) that catheptic carboxypeptidase B from spleen inactivates bradykinin *in vitro* by cleaving the COOH-terminal arginine from the polypeptide. This enzyme has now been found in liver and kidney. The enzyme has an absolute requirement for SH activators such as cysteine and mercaptoethanol. The enzyme is active optimally at acid pH. Iodoacetic acid inhibits the reaction. Free phenylalanine is also found in the reaction mixture and results from the action of catheptic carboxypeptidase A (*J. biol. Chem.* **237**, 1082, 1962) on the COOH-terminal phenylalanine produced after cleavage of the arginine residue from bradykinin by catheptic carboxypeptidase B.

The catheptic carboxypeptidase B enzyme differs from carboxypeptidase N of plasma (Erdös *et al.*, *Biochem. Pharmacol.* **11**, 585, 1962) and the carboxypeptidases of brain tissue (Krivoy and Kroeger, *Brit. J. Pharmacol.* **22**, 329, 1964) in its pH optima and requirement for SH activators. Since the possibility exists that intracellular proteinases play a role after cellular injury in the production and degradation of plasma kinins, the degradative role of the carboxypeptidases is of interest. The possible role of other catheptic enzymes in producing kinins will be discussed. (Supported by Grants AM-09393, and General Research Support, U.S. Public Health Service; and a grant from the Life Insurance Medical Research Fund.)

16. Vasoconstriction Induced by Bradykinin in the Intact Rabbit Ear (cinematographic presentation). P. S. GUTH, R. BOBBIN, G. CANO and J. AMARO (*Dept. of Pharmacology, Tulane Univ., New Orleans, La., U.S.A.*).

In a previous article (*Ann. N.Y. Acad. Sci.* **104**, 69, 1963) it was reported that bradykinin induced a constriction of veins in the intact rabbit ear as well as a decrease in outflow in preparations of the isolated rabbit ear, dog and cat hind limbs, and rat hindquarters perfused with appropriate saline solutions. The present report confirms that work and extends it.

The bradykinin-induced venoconstriction may be demonstrated in the ears of rabbits lightly anesthetized with urethane (1 g/kg i.p.). Bradykinin in doses of 2·4–4 μg, injected i.v. (via cannula in a primary branch of the marginal ear vein) in an orthodromic direction in the ear being photographed, causes constriction of the marginal vein. The constriction occurs in less than 10 sec after injection and reaches a maximum in 25–35 sec. The maximal effect thus elicited is a 50% reduction in vessel diameter.

The venoconstrictive effect of bradykinin is still present in animals with greater auricular nerve