Physiology, The Medical School, Birmingham, England).

In many glandular tissues which display intermittent secretory activity, this function is accompanied by a local dilatation of the arterioles, which permits a great increase of blood flow through the tissue or organ concerned. It is ten years since the hypothesis was advanced that the vasodilatation is directly caused by bradykinin, or some similar plasma kinin, formed in the interstitial fluid of the gland as a result of its activity.

The hypothesis was first formulated on the basis of experiments on the submandibular salivary gland of the cat. It has since been found to be applicable to the pancreas in the cat and to the sweat glands in man.

The recent experimental findings have enabled several possible arguments against the hypothesis to be overcome.

21. Studies of the Structure of Kininogen of Beef Serum; The Isolation of a Kinin-like Substance from the Sera of Birds. K. HOCHSTRASSER, H. SCHELLER and E. WERLE (*Klinish-Chemisches Institut an der Chirurgischen Klinik der Universität, Munich, Germany*).

The digestion of fractionated kininogen of beef serum with pepsin yields a mixture of polypeptides which contains peptides with the amino acid sequence of kinins. The peptides, which still react with kininogenases, are isolated and analyzed for amino acid composition.

Besides these polypeptides there are at least two more pharmacologically active peptides in the pepsin digest of beef serum. One lowers the blood pressure, the other raises it. Both polypeptides have been isolated, and their amino acid composition is currently under study.

Kallikrein from the pancreas of birds liberates kinin only from avian but not from mammalian serum. This kallikrein, the spontaneously formed kinin, and the kinin liberated by kallikrein from avian serum have been characterized.

22. The Action of Hypotensive Polypeptides on the Pulmonary Arterial Pressure. H. KONZETT (*Institute of Pharmacology, Univ. of Innsbruck, Austria*).

Bradykinin, kallidin, and eledoisin cause a rise of pressure in the pulmonary artery of some species. These polypeptides also contract the isolated pulmonary arteries of the same species. Under certain conditions, antiphlogistic substances antagonize the effect of bradykinin and kallidin on the pulmonary vessels *in vitro* and *in vivo*. The effect of eledoisin on these vessels is less easily antagonized by such compounds.

According to investigations on the left and right atrial pressure and on the heart–lung preparation, the increase in the pressure in the pulmonary artery after bradykinin, kallidin, and eledoisin is due, predominantly, to a direct action on the pulmonary vessels.

23. Effect of Bradykinin on Submandibular Salivary Gland Permeability. D. C. KROEGER and W. KRIVOY (*Dental Branch, Univ. of Texas and Baylor Univ. College of Medicine, Houston, Texas, U.S.A.*).

Previous investigations from these and other laboratories have demonstrated that the nerve-induced release of a plasma kinin in glandular tissue results in vasodilatation. Other studies on the central nervous system and muscles suggest that the plasma kinin, bradykinin, alters cellular permeability. The technique of Martin and Burgen (*J. gen. Physiol.* **46**, 225, 1962), using sucrose, was employed here to study the possibility that bradykinin might influence glandular permeability in addition to the aforementioned glandular vascularity. Intravenous doses of synthetic bradykinin (1–2 μg/kg) were equipotent to epinephrine (3–5 μg/kg) in causing an increase in the permeability of the dog's submandibular salivary gland to sucrose. The time courses of this response to both drugs at this dose level were similar. Phenoxybenzamine (5 mg/kg) was found to block the action of epinephrine on permeability, whereas it intensified the degree and duration of the effect of bradykinin upon the permeability to sucrose. In summary, bradykinin increases the permeability of the dog's submandibular salivary gland to a large carbohydrate molecule. This action of bradykinin appears to be independent of epinephrine. (This research was supported by funds from Grant DE01390 of the U.S. Public Health Service.)

24. Extraction of Substance P from Brain. F. LEMBECK, H. HEIZMANN and G. SEIDEL (*Pharmakologisches Institut der Universität, Tübingen, Germany*).

Substance P was extracted from pig brain by a new method. The tissue was homogenized with chloroform:methanol (2:1), centrifuged, and re-extracted; the chloroform–methanol extract, after washing with distilled water, was concentrated *in vacuo* and freeze-dried. The white powder obtained was boiled in 0·1 N HCl, and a large amount of sediment was separated. The supernatant fluid contained a substance that contracted smooth muscle. Further purification was achieved by acetic acid–ether precipitation.

This extract was compared with substance P obtained from the same tissue by the usual Gaddum-Euler method followed by acetic acid–ether precipitation. The activity ratio of both