section and cervical ganglionectomy. In preliminary experiments, administration of phentolamine (1–3 mg) antagonizes the venoconstrictive effects of both norepinephrine and bradykinin. It is suggested that the primary vascular event subsequent to bradykinin administration is venoconstriction. Secondarily, capillaries may be dilated passively by the pressure increase resulting from momentarily-impeded flow. (Supported by a grant from the Louisiana Heart Association.)

17. Enzymatic Kinin Release from Purified Kininogen and from Low Molecular Compounds. E. HABERMANN (*Institut für Pharmakologie und Toxikologie der Universität, Würzburg, Germany*).

The kinins released from bovine kininogen by highly purified enzymes (kallikreins, trypsin, crotalus venom) have been identified by a combination of column chromatography and biological testing. Two types can be distinguished: kinin-9-forming enzymes split kinin-11 (met-lys-bradykinin) and bradykinyl–serine bond besides kininogen. For the kinin-10-forming pancreatic kallikrein, only kininogen serves as substrate. Pig serum kallikrein belongs to the former group of enzymes. The purification and some of the pharmacological and biochemical characteristics of swine serum kallikrein will be described. For a closer approach to the portion of kininogen that can be activated, peptic fragments of the purified protein have been fractionated by a combination of chromatographic procedures. Two kinin-yielding peptides have been isolated, their structure determined, and their reaction products with kinin-forming enzyme identified. Both peptide fractions as well as further peptic fragments derived from kininogen are active on rat uterus, guinea pig ileum, rabbit blood pressure, and capillary permeability; therefore, 'pepsitocin' is a term which covers various kinins.

18. Effects of Bradykinin and Angiotensin on Ganglionic Transmission. W. HAEFELY, A. HÜRLIMANN and H. THOENEN (*Dept. of Experimental Medicine, Hoffmann-LaRoche, Inc., Basle, Switzerland*).

The effects of bradykinin and angiotensin on synaptic transmission were studied in the superior cervical ganglion of the cat. Both polypeptides produced an inhibition of ganglionic transmission in extremely low doses. For close-arterial injection to the ganglion, threshold doses were of the order of 10^{-18} moles for angiotensin. Bradykinin was slightly less potent on a molar basis and the inhibition of transmission less pronounced. A peculiar dose–effect relationship was observed regularly with both polypeptides, two ranges of effective doses being separated by a wide dose range within which no effect on ganglionic transmission occurred. Interaction of bradykinin with other drugs at the ganglionic level will be reported and possible mechanisms of the action on ganglionic transmission discussed.

19. Bradykinin and Pulmonary Vascular Permeability in Isolated Blood-Perfused Rabbit Lungs. A. HAUGE, P. K. M. LUNDE and B. A. WAALER (*Institute of Physiology, Univ. of Oslo, Norway*).

Bradykinin has been found to cause vasodilation in most areas of the systemic circulation, where another reported effect is increased capillary permeability. In the pulmonary vascular bed of various species, however, bradykinin seems to cause vasoconstriction.[1–4] We have tried to evaluate the possible effect of bradykinin on vascular permeability in isolated rabbit lungs, perfused with homologous heparinized blood at constant-volume inflow (average flow 234 ml/min). The weight of the preparation, the inflow pressure, and the tidal air were followed continuously.

When infusions into the pulmonary artery of 6 to 200 μg synthetic bradykinin per min were started or stopped, rapid weight changes were observed. These changes were apparently related to the vasoconstriction caused, and they were interpreted as being due to capacity changes in the vascular bed. No weight changes indicating alterations in net fluid movement across the vascular wall could be observed during infusions. Bradykinin infusions were also carried out during periods with elevated left atrial pressure and during reversed perfusion (left atrial inflow), both situations giving a high pulmonary capillary pressure with steady net outward filtration of fluid. Bradykinin infusions under such conditions did not influence the steady weight increase of the preparation.

It is concluded that intravascularly-infused bradykinin in doses large enough to give marked vasoconstrictor responses does not increase vascular permeability in the pulmonary vascular bed of rabbit lungs. The vasoconstriction caused must mainly occur at precapillary sites.

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20. Further Experiments on the Role of Plasma Kinins as Mediators of Functional Vasodilatation in Glandular Tissues. S. M. HILTON (*Dept. of*