or to dialysed cat saliva injected close-arterially, leaves an equivalent vasodilatation caused by stimulation of the chorda-lingual nerve or by injection of ACh practically unaffected. (c) During prolonged perfusion of the gland with horse serum, from which cat saliva releases no kallidin, stimulation of the chorda-lingual nerve still produces marked vasodilatation, although intra-arterial injections of dialysed cat saliva are ineffective. (d) It is possible to deplete the submaxillary gland of kallikrein by ligation of the duct for 3 days and subsequent stimulation of the sympathetic nerve. Chorda-lingual nerve stimulation still produces a normal vasodilatation in such a kallikrein-depleted gland.

In the rabbit. The secretion and vasodilatation in the submaxillary gland produced by chorda-lingual nerve stimulation are reduced or blocked in parallel by atropine.

36. Recent Developments in the Chemistry of Gastrin. R. C. SHEPPARD (*The Robert Robinson Laboratories, Liverpool Univ., Liverpool, England*).

The natural hormone gastrin appears to play an important role in digestive processes, particularly in the control of gastric acid secretion. The recent isolation[1] of two gastrins from hog antral mucosa has been followed by their structural elucidation[2] and total synthesis.[3] Both hog gastrins are heptadecapeptide amides, and differ only in the sulphation of a tyrosyl residue in one hormone. Further synthetic work has enabled the minimal structural requirements for gastrin-like activity to be established, and has yielded a number of analogues with interesting biological properties.

1. R. A. GREGORY and H. J. TRACY, *Gut*, **5**, 103 (1964).
2. H. GREGORY *et al.*, *Nature* (*Lond.*) **204**, 931 (1965).
3. J. C. ANDERSON *et al.*, *ibid.*, p. 933.

37. Prevention of the Edematous Arterial Reaction and the Atherosclerosis of Cholesterol-Fed Rabbits by Anti-Bradykinin Agents. TAKIO SHIMAMOTO, FUJIO NUMANO, MASAYOSHI KUBOTA and TSUTOMU FUJITA (*Institute for Cardiovascular Diseases, Tokyo Medical and Dental Univ., Tokyo, Japan*).

Edematous arterial reaction (*Jap. Heart J.* **3**, 581, 1962) is an immediate and general effect in arterial walls of animals after such stresses as administration of atherogenic substances or traumatization. The reaction consists of a microscopic swelling of arterial wall, owing to the accumulation of serous material in the amorphous extracellular spaces of intimal and medial layers, which shows a close similarity to *das initiale fettfreie Ödem*, the hypothetical initial stage of human atherosclerosis of the German school.

In an attempt to prevent the reaction, ergotamine tartrate, atropin, pyribenzamine, Dexamethasone, etc. were without effect in relatively large doses, whereas the following substances were effective. (1) Trasylol (300 U/kg i.v.): a bradykinin-forming (polyvalent proteolytic) enzyme-inhibiting substance. (2) "Anti-bradykinin agents in veins"; acetylsalycilic acid (50 mg/kg p.o.), nialamide (50 mg/kg p.o.), cyproheptadine (5 mg/kg p.o.), and pyridinolcarbamate (1 mg/kg p.o.).

The atheromatous changes in cholesterol-fed rabbits were slightly inhibited by a nialamide (10 mg/kg/day p.o.) and cyproheptadine (5 mg/kg/day p.o.) and markedly inhibited by pyridinolcarbamate (5 mg/kg/day p.o.), showing a characteristic tendency to fibrous healing. Pyridinolcarbamate not only minimized very noticeably the atheromatous changes and accumulation of cholesterol in arterial walls ($P < 0.01$) but also prevented remarkably the formation of atheroma and its fatty degeneration and necrosis formation ($P < 0.01$) in animals kept on a cholesterol diet for 15 weeks, which had a high serum cholesterol level amounting to 1,735 ± 169 mg/kg during the last 5 weeks of experiment.

38. Some Physiological and Pathological Roles of Kininogen and Kinins. F. SICUTERI, G. FRANCHI, M. FRANCIULLACCI and P. L. DEL BIANCO (*Centro Cefalée, Clinica Medica dell'Università, Florence, Italy*).

The kininogen–kinin system was suggested to be of physiological importance in the control of microcirculation. According to our experiments, kininogen is higher in arterial than in venous blood.[1] The capillary bed exhibits a strong kinin releasing and clearing power.[2] Hydrolysis of kinins is higher in venous than in arterial blood.

Bradykinin and kallidin may also act as mediators in some experimental and vascular pains.

Evidence of strong sensitization to the pain-producing properties of bradykinin, kallidin, and kallikrein by 5-hydroxytryptamine on nociceptors has been shown in man. The importance of this potentiation in man during myocardial infarction and other thromboembolic accidents is emphasized.[3]

Experimental and laboratory findings support our hypothesis concerning the pathogenic importance of the presence of kinins in cerebrospinal fluid during subarachnoid hemorrhage. Kinins are released largely by dilution when blood is mixed with cerebrospinal fluid in subarachnoid spaces. Headache and meningeal syndrome of subarachnoid hemorrhage may be provoked by the pain-producing and inflammatory properties of kinins.[1]