the cause seems to be the uterine involution and the trauma of the delivery.

The low BKG content of the fetal blood at birth and in the first days of extrauterine life could reflect the immaturity of the liver in the newborn.

33. The Purification and Some Properties of Human Plasma Kallidinogen. JACK V. PIERCE and MARION E. WEBSTER (*Laboratory of Metabolism, and Laboratory of Cardiovascular Physiology, National Heart Institute, Bethesda, Md., U.S.A.*).

Kallidinogen, a plasma α_2-globulin which produces kallidin or bradykinin when treated with kallikrein, trypsin, or snake venom, has been purified about 400-fold over the starting human plasma and in 10% yield. This was accomplished by the following four steps: (1) DEAE-cellulose chromatography and (2) rechromatography, (3) hydroxylapatite chromatography, and (4) Sephadex G-200 gel filtration. Linear gradients of phosphate buffer were used in steps (2) and (3). Step (2) gave two peaks of activity in a ratio of 3·9. Step (3) on the combined activity from (2) also gave two peaks, I and II in the order of their elution, but in a ratio of 0·7. Gel filtration of peak I gave a major activity peak with a K_d of 0·46 and a minor peak with a K_d of about 0·23. Peak II on the same G-200 column gave only one activity peak (K_d = 0·45), from which the purest material was obtained. Hydroxylapatite chromatography of peak II material, with a linear gradient of phosphate buffer in the presence of 1 M sodium chloride, also gave two peaks of activity in a ratio of about 1·0. Experiments are being done to clarify this confusing situation. Studies of the physicochemical and biochemical properties of kallidinogen will be made as soon as material satisfying several criteria of homogeneity has been obtained.

34. Characterization of Kinins in Wasp Venom. J. L. PRADO,* Z. TAMURA,† E. FURANO, J. J. PISANO and S. UDENFRIEND (*Laboratory of Clinical Biochemistry, National Heart Institute, Bethesda, Md., U.S.A.*).

At least six kinin fractions were observed when wasp venom preparations (genus *Polistes*) were chromatographed on columns of carboxymethyl cellulose and carboxymethyl Sephadex. The first two kinins had pharmacological and chemical properties similar to bradykinin and kallidin respectively. However, when the fluorescent dimethylnaphthylsulfonyl (dansyl) derivatives were examined by thin-layer chromatography (TLC) several biologically active fluorescent bands were observed, none of which corresponded exactly to dansyl bradykinin or dansyl kallidin. The fluorescent peptide derivatives had 1–2% the potency of the free peptide in the estrous rat uterus assay. Most of the kinin activity of venom (>50%) was in fraction 3 which consisted of a single peptide as revealed by TLC of the dansyl derivative. It had the amino acid composition: Arg_2, Asp, Gly_2, Glu, Leu, Lys_2, Phe_2 Pro_3, Ser, Thr. Chymotrypsin but not trypsin destroyed biological activity. An active peptide was isolated from the tryptic digest. It was composed of Arg_2, Gly_2, Phe_2, Pro_3, Ser, with glycine the N-terminal amino acid. The peptide was indistinguishable in chemical and biological tests from synthetic glycylbradykinin, also termed gly^1-kallidin (Schröder and Hempel, *Experientia, Basel* **20**, 529, 1964). To be determined are the amino acid sequence of undigested fraction 3 and the structures of the kinins in the other fractions. (Reference peptides were kindly supplied by E. Nicolaides and E. Schröder.)

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35. Kallikrein in the Submaxillary Gland. M. SCHACHTER (*Dept. of Physiology, Univ. of Alberta, Edmonton, Alberta, Canada*).

Shortly after salivary kallikrein was described (Werle and Roden, 1936), it was suggested that this substance was the mediator of chorda-tympani-evoked vasodilatation in the submaxillary gland and tongue (Ungar and Parrot, 1936). This suggestion was made to explain the fact that vasodilatation in the gland caused by stimulation of the chorda-tympani nerve is not blocked by doses of atropine, which readily block the secretory response. Further work led to the specific conclusion that vasodilatation in the active gland is secondary to secretion; i.e. it is caused by kallikrein passing from the secretory cells into the tissue spaces where it releases the vasodilator peptide, kallidin (Hilton and Lewis, 1955, 1956, 1958).

Our experimental results listed below, however, have led us to conclude that vasodilatation produced in the submaxillary gland by stimulation of the chorda-lingual nerve is *not* mediated by kallikrein, but that true vasodilator nerve fibres, probably cholinergic, are present in this nerve.

In the cat. (a) The vasodilatation resulting from close arterial injection of dialysed cat saliva into the salivary gland with intact blood circulation differs from that caused by stimulation of the chorda-lingual nerve or by acetylcholine (ACh) similarly injected: it is slower in onset, it is not so great, and it is generally more prolonged. (b) Desensitization of the blood vessels to the vasodilator action of a standard dose of bradykinin