appears unlikely that PGE_1-induced salivary secretion is the result of neurotransmitter release from postganglionic autonomic nerve endings of parotid gland. However, PGE_1 might act directly on acinar cells of this gland. Although PGE_1 at lower doses did not evoke salivary secretion, it had a modulating effect on secretory responses evoked by parasympathetic nerve stimulation of the parotid[7].

1 Acknowledgment. This work was supported by NIDR grant DE05633. The authors wish to thank Ms Sonya Wynn for her technical assistance.

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Synthetic corticoliberin needs arginine vasopressin for full corticotropin releasing activity[1]

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Summary. The corticotropin-releasing activity of synthetic corticoliberin increases 75–90% when it is incubated in the presence of arginine vasopressin with rat anterior pituitary cell suspensions; this suggests a synergistic control of ACTH secretion by vasopressin and corticoliberin.

A synthetic sheep corticoliberin (sCRF) has recently been shown to evoke a marked release of corticotropin (ACTH) in vitro[2] an in vivo[3]. Because of our interest in the role of arginine vasopressin (AVP) in the control of ACTH secretion[4–7], we tested the hypothesis that sCRF and AVP may interact at the anterior pituitary level. Vitamin C has previously been implicated in CRF activity[6,8]; therefore we also asked the question whether vitamin C played a role in the biological activity of sCRF.

Methods and materials. CRF activity of various compounds was determined by the Sayers assay[9], as described previously[5,6], by measuring the amount of bioactive ACTH released during 1 h from dispersed anterior pituitary cells. The 1-ml medium was a Krebs-Ringer bicarbonate buffer supplemented with 2 mg/ml glucose and 1 mg/ml beef serum albumin. Medium from anterior pituitary cell suspensions was tested in duplicate on adrenocortical cells.

Secretagogues included sCRF (Bachem); AVP (Sigma); and basal hypothalamic extracts[6]. The sCRF was diluted under nitrogen in a degassed 0.1 N HCl solution, and one half of the solution was supplemented with 10^{-3} M vitamin C. The 2 types of solutions were distributed in 1 ml aliquots containing 2 µM sCRF, frozen at −20 °C, and neutralized before the assay.

Average CRF activities from 4 separate assays were determined for sCRF, AVP, hypothalamic extracts, vitamin C and combinations of the above at doses similar to those used previously[2,6]. Differences in means were analyzed for statistical significance by Student's t-test for small numbers.

Results and discussion. In contradistinction to the exponential dose-response curves for hypothalamic extracts[5,8,9], those for sCRF display a progressively lower slope at

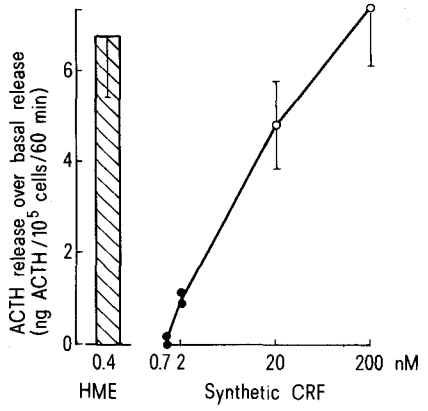

Figure 1. CRF activity of synthetic CRF (sCRF) and of 0.4 equivalents of medial basal hypothalamic extracts (HME). CRF activity is expressed by the amount of ACTH released per 100,000 cells during the 1-h incubation. Basal release, 0.8 ± 0.18 ng/h/10^5 cells, was subtracted to yield the net CRF effect. Averages and SEM are from 4 experiments (2 experiments for the lowest doses of sCRF). The sCRF was conserved in vitamin C.

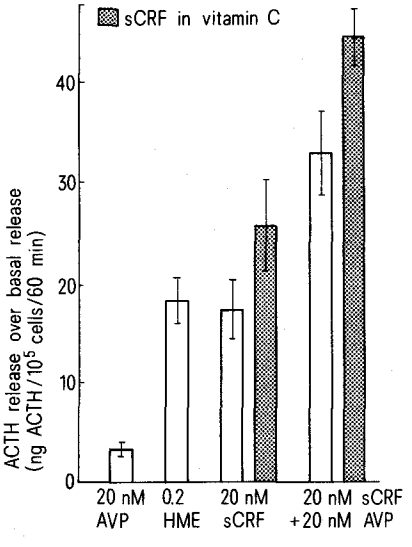

Figure 2. CRF activity of AVP, HME, sCRF and sCRF plus AVP. Means ± SEM are from 4 experiments each in a different (more sensitive) assay series than that in figure 1. CRF activity (see legend to fig. 1). Basal ACTH release was 0.72 ± 0.03 ng/h/10^5 cells. The sCRF was conserved (hatched column) or not (white column) in vitamin C.

increasing concentrations (fig. 1). At 200 nM, the CRF activity of sCRF is similar to that of 0.4 equivalents of a rat medial basal hypothalamic extract.

When 20 nM sCRF is applied to anterior pituitary cells in combination with 20 nM AVP, the total CRF effect is much larger than the sum of individual CRF activities (fig. 2). This is observed whether vitamin C is present or not within the medium (compare hatched columns, vitamin C, with white columns). However, the presence of vitamin C enhances the total CRF effect ($p < 0.1$ for sCRF, $p < 0.025$ for sCRF plus AVP). In a previous study[6], we have shown that the presence of vitamin C enhances the amount of CRF distinct from AVP found in medium bathing rat median eminences in vitro, but has no significant effects on the CRF bioassay. Thus vitamin C probably protects CRF from being rapidly oxidized and converted to a biologically less active[2] compound.

The AVP concentrations applied in this study correspond to those found in hypophysial portal plasma of the rat[10]. The concentrations of a sCRF-like substance in portal plasma is not yet known, but is anticipated to be considerable in view of the large number of nerve terminals containing a sCRF-like substance that were detected by immunocytochemistry in proximity to the portal vessels in the rat (Tramu and Pillez[11] and our unpublished observations). Thus, we propose that AVP and a sCRF-like substance exert a synergistic control of ACTH secretion at the anterior pituitary level. It remains to be seen whether this sCRF-like substance is identical to the 'potentiating-factor' described previously[8,12].

1 Acknowledgments. This study was supported by grant No. 3.581-0.79 of the Swiss National Science Foundation. We thank Ms C. Estoppey for technical assistance. Reprint requests should be addressed to A. B.

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Amino acid concentrations in blood of patients with an acute myocardial infarction

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Summary. Levels of essential and nonessential amino acids in blood of patients with an acute myocardial infarction (AMI) were found in general not to differ from values obtained from non-AMI patients. This is in contrast to blood taurine levels which are elevated in the AMI patients.

Taurine (2-aminoethanesulfonic acid) is present in high concentrations in cardiac tissue[1] and is reported to have pharmacologic effects of central origin on the cardiovascular system in cats[2]. However, while the physiologic function of taurine is not known it has been proposed that taurine may have effects on the electrical activity of the heart[3,4].

Studies with animal models have demonstrated that cardiac muscle is depleted of its taurine stores by such insults as coronary artery occlusion[5], hypoxia[6], and drug-induced myocardial necrosis[7].

Cardiac tissue appears to be the source of elevated concentrations of taurine observed in the blood from animals treated with sympathomimetic agents[7]. In this context we have recently reported[8] that the concentrations of taurine are increased in the blood of patients who had an AMI. Furthermore determination of taurine content of human cardiac tissue obtained at autopsy indicates that patients who died of an AMI had decreased levels of taurine in their cardiac tissue[9].

The purpose of this study was thus to determine whether the othe natural amino acids present in the blood were also increased after an AMI or if the increase in blood taurine levels was unique.

Methods. Patients admitted to the hospital for complaint of chest pain were classified as AMI or non-AMI according to standard diagnostic criteria such as changes in cardiac enzymes[10] and electrocardiogram[11]. Blood samples were obtained at various time periods beginning at the emergency room (ER) admittance and extending to 120 h. Blood samples were anticoagulated with EDTA and then deproteinized with an equal volume of 5% perchloric acid and centrifuged for 10 min at 10,000 × g. An aliquot of the

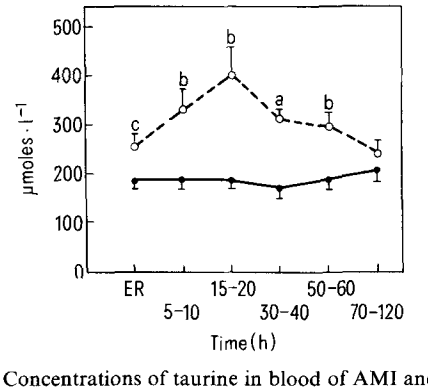

Figure 1. Concentrations of taurine in blood of AMI and non-AMI patients. Blood samples were analyzed from 8 AMI patient and from 9 non-AMI patients. Open circles denote AMI patients; closed circles denote non-AMI patients. Time periods were calculated by using the ER admittance time as zero h. Data are expressed as means ± SE. ($^a p < 0.001$; $^b p < 0.005$; $^c p < 0.05$).