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that were detected by immunocytochemistry in proximity to the portal vessels in the rat (Tramu and Pillez[11] and our unpublished observations).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].

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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

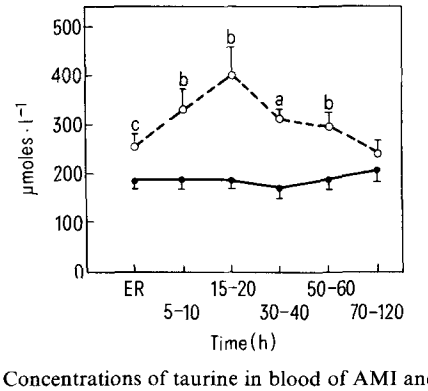

Figure 1. Concentrations of taurine in blood of AMI and non-AMI patients. Blood samples were analyzed from 8 AMI patient and from 9 non-AMI patients. Open circles denote AMI patients; closed circles denote non-AMI patients. Time periods were calculated by using the ER admittance time as zero h. Data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

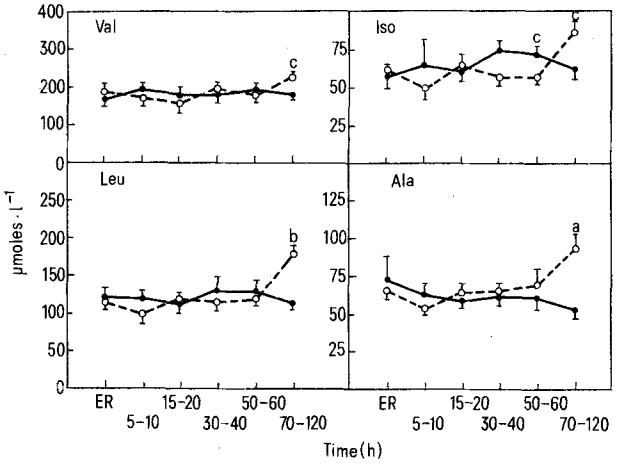

Figure 2. Concentrations of essential amino acids in blood of AMI and non-AMI patients. See legend to figure 1 for details. (Val, valine; Iso, isoleucine; Leu, leucine; Ala, phenylalanine) ($^{a}p < 0.001$; $^{b}p < 0.005$; $^{c}p < 0.05$).

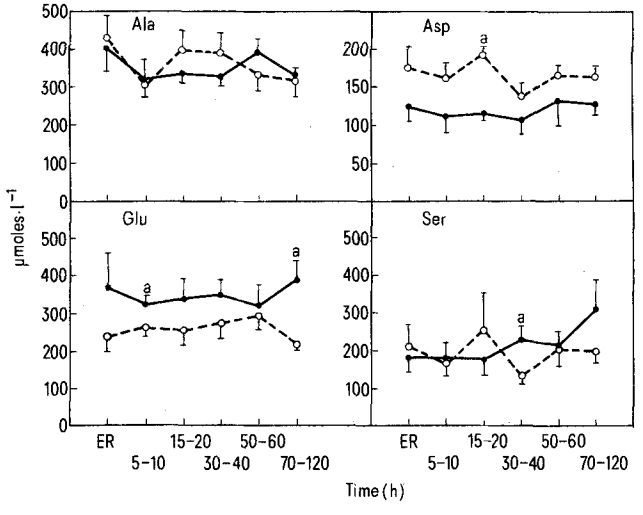

Figure 3. Concentrations of nonessential amino acids in blood of AMI and non-AMI patients. See legend to figure 1 for details. (Ala, alanine; Asp, aspartic acid; Glu, glutamic acid; Ser, serine) ($^{a}p < 0.05$).

supernatant was analyzed for amino acid content on a Beckman amino acid analyzer. Statistical significances were determined by using the Wilcoxon 2-sample test.

Results. Levels of taurine in blood samples which were obtained at the ER admittance from AMI patients were significantly elevated as compared to blood taurine levels of patients who had chest pain but who were later diagnosed as not having had an AMI (fig. 1). The taurine levels remained elevated for 50–60 h and returned to control values by the 70–120 time period. Significant differences in the essential amino acid levels in the blood of AMI and non-AMI patients were observed primarily at the 70–120-h time period (fig. 2). Of the nonessential amino acid levels (fig. 3) no differences between AMI and non-AMI patients were observed for alanine; fluctuations in levels were demonstrated for aspartic acid (15–20 h), glutamic acid (5–10; 70–120 h) and serine (30–40 h).

Discussion. In this study comparisons of the concentrations of essential and nonessential amino acids in the blood of AMI and non-AMI patients demonstrated only few differences at early time periods and thus differed from the pattern obtained for taurine. We thus conclude that the elevation of taurine levels is unique as denoted by its early rise after myocardial injury.

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Strain- and age-dependent change in carrier independent helper capacity

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Summary. Throughout life the immune system undergoes progressive changes. We report, here, that carrier-independent helper capacity increases during adult life and that the rate of the increase varies in different mouse strains, i.e. is polymorphic.

The relative capacity of regulatory and executive cells of the immune system changes from sexual maturity to old age; the changes are polymorphic. So far, age-related changes of immune regulation have been observed in terms of decline in specific suppressor or amplifier suppressor cells[2], increase in nonspecific non-T[3] and T[4] suppressor cells, and increase in anti-auto-idiotypic antibody[5]. Changes in effector functions have been observed in terms of B-cell subpopulations[6] and of T cells in delayed hypersensitivity[7].

Help for a secondary response to a determinant can be dependent on previous exposure to other determinants of the macromolecule; it can take place if in vitro stimulation occurs in the presence of the sensitizing macromolecule,