Acta Biochimica Polonica
Vol. 40 No. 1 1993

Superoxide dismutase and catalase activity in patients with stable angina pectoris

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The superoxide dismutase anion and its transformation species have been implicated as an etiological factor underlying many clinical disorders, especially heart diseases [1, 2].

Three stages of ischemic cardiac injury are currently recognized, and a free radical mechanism has been proposed to be involved in all of the. The first detectable stage of injury is the generation of reperfusion arhythmia. Reperfusion after an ischemic period of only several minutes can result in ventricular tachycardia or fibrillation [3]. The second stage, an ischemic period lasting from 5 to 15 min, will result in a prolonged deficit in contractility following reperfusion. In this state, called "stunned" myocardium, all of the ischemic myocytes are still viable and the heart will completely recover its function but the recovery may require several days [4]. Finally, when the ischemic period extends to 20 min or more, some of the heart cells will be irreversibly injured resulting in the heart infarct [5].

The source of the free radicals in myocardium following reperfusion is currently a subject of discussion and several sources have been proposed among them xanthine oxidase [2, 6]; the others are the electron chain of mitochondria and the arachidonic acid pathway. Recently some investigators have proposed leukocytes, especially activated neutrophils, as the source of free radicals in the reperfused heart [7].

The aim of this study was to determine the activity of antioxidant enzymes: superoxide dismutase (SOD)[1] and catalase in erythrocytes from healthy persons and patients with stable angina pectoris.

Twenty six patients (aged 39 - 76 years, 14 men and 12 women) with stable angina pectoris were tested. The control group consisted of 86 persons (aged 29 - 65 years, 53 men and 33 women) clinically healthy. The methods used were the same as described in the previous communication [8].

The results were analyzed by Student's *t* - test and the means and standard deviations (S.D.) are given. The results of SOD activity in erythrocytes of patients and healthy persons are presented in Fig. 1 (A). The mean SOD activity in patients with stable angina pectoris was $(1.34 \pm 0.54) \times 10^3$ U/g Hb, and in the control group $(1.61 \pm 0.48) \times 10^3$ U/g Hb. The difference was statistically significant ($P < 0.02$). Figure 1 (B) presents the results for catalase in erythrocytes of 26 patients with stable angina pectoris and 86 healthy persons. The catalase activity in patients was $(5.11 \pm 1.06) \times 10^4$ IU/g Hb, while in control group $(5.72 \pm 1.17) \times 10^4$ IU/g Hb. Also in this case the decrease in the enzyme activity was statistically significant ($P < 0.02$).

In our study, the venous blood was taken from patients soon after the onset of pain (immediately after bringing the patient to hospi-

[1]Abbreviations: Hb, hemoglobin; SAP, stable angina pectoris; SOD, superoxide dismutase

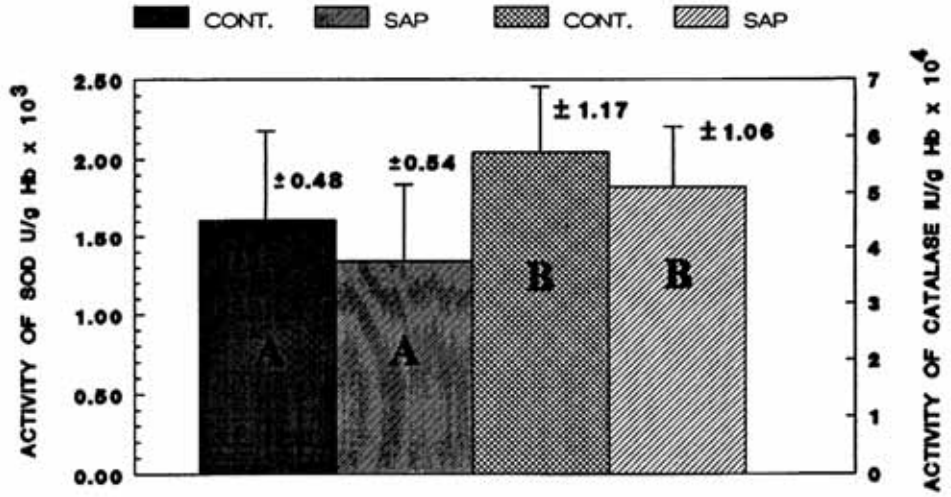

Fig. 1. Activity of superoxide dismutase (SOD)(A) and catalase (B) in erythrocytes of patients with stable angina pectoris (SAP) and in the control group.
Activity of catalase is expressed in international units per gram hemoglobin. One unit of SOD activity is defined as the amount of the enzyme which inhibits by 50% the superoxide radical-mediated conversion of adrenalin to adrenochrome

tal). Evidences in literature suggesting that radicals are generated during ischemia and reperfusion allow to suppose that the onset of pain is related to a burst of oxygen free radicals formation. Bolli *et al.* [9] using electron paramagnetic resonance spectroscopy and a spin trapping agent, investigated free radicals formation within the coronary venous effluent in dogs subjected to 15 min of coronary occlusion and reperfusion. Although the initial burst of oxygen free radicals peaked at 2 min after reflow, the production of oxygen radical continued for up to 3 h after reperfusion [9].

The decrease of SOD activity may be caused by its inhibition by an excess of substrate, that is by superoxide radicals.

The activity of catalase in erythrocytes of patients with stable angina pectoris was also decreased. This could be caused by inhibition of the enzyme by excess of H_2O_2.

The oxygen free radicals can be cytotoxic to cells in several ways. Reaction with protein includes oxidation and destruction of amino acids, oxidation of sulfhydryl groups, and polypeptide chain scission [10]. The reaction with fatty acids leads to lipid peroxidation in membranes. Many findings indicate that lipid peroxidation in heart cell membranes causes various electrophysiological and mechanical abnormalities [11]. Yagi *et al.* [12] showed that endothelial cells in coronary arteries are particularly susceptible to lipid peroxidation.

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