INTRODUCTION

The past decade has shown that emotional painful stress induced by environmental factors (1,2), in the form of "anxiety neurosis" (3) or caused by an endogenous factor like myocardial infarction (4,5) involves the activation of lipid peroxidation (LP) in the myocardium. Preliminary administration of antioxidants limits or eliminates the LP activation (6,7,8) and at the same time decreases or completely eliminates disturbances of the heart metabolism, structure, contractile function (9) and electric stability (10) in emotional painful stress. These data create definite prerequisites for therapeutic and prophylactic application of antioxidants both in the primary stress damage and in the ischemic heart disease. Thus the role of LP in the pathogenesis of stress-induced damage of the heart and possibilities of its antioxidant protection constitute one of essential problems of current cardiology. This problem has been discussed in recent works (9,11), however, a number of questions remains still open. In particular, the place is unclear which the stress activation of LP takes in the complex chain of the heart stress damage; the role of the organism's natural antioxidant system in antistress protection and possible ways of its activation need investigation. These problems constitute the subject of this chapter.
THE LP ACTIVATION AND HEART DAMAGE IN EMOTIONAL PAINFUL STRESS

Stress reaction is a necessary link of the organism adaptation to different factors of the environment and adrenergic mobilization of the heart function and metabolism is a necessary component of this reaction. Even following a short-term stress action which causes no injuries, a trace remains in the heart muscle. This trace manifests in the increased resistance to hypoxia of isolated hearts both of animals exposed to such a stress action and of those treated with small doses of catecholamines (12,13). At the same time, it is known that in stress action with increased duration and intensity or after administration of large doses of catecholamines, the adaptive effect regularly turns into the adverse one.

For this reason, the principal below-set results have been obtained on rats exposed to 6 hs emotional painful stress after the Desiderato et al. method (3). The essence of this model designed as "anxiety neurosis" is in the induced conflict between the developed conditioned reflex of the avoidance of pain by escaping to a "safe" platform and unconditioned pain reflex induced by electric footshocks inflicted on this platform. The footshocks are inflicted with occasional intervals to create in animals fear of expectation. It was found that, in the heart, such a stress action regularly resulted in a significant LP activation which showed in 2-3fold increase in lipid hydroperoxides and Schiff bases (6). This effect was accompanied by the labilization of myocardial lyzosomes and accelerated release of creatine phosphokinase, lactate dehydrogenase and other enzymes from the myocardium of isolated hearts of stress-exposed animals into the perfusate (14,15). Simultaneously the reduced activity and accelerated thermal inactivation of sarcolemmal Na,K-ATPase (16) were observed in the myocardium along with the decreased activity of Ca-