INTRODUCTION

Substantial and convinding evidence from recent investigation indicates that tissue injury after ischemia and reperfusion in acute myocardial infarction is not a consequence of simple tissue anoxia but a sequence of cellular interactions involving several agents including leukocytes ***(Huber et al., 1991),*** oxygen free radicals ***(Zweier, et al., 1987)*** and calcium ions ***(Hearse, 1977)***. Among these, neutrophils are believed to be one of the most important cell types causing damage to myocardial tissues by generating and releasing reactive oxygen metabolities ***(Bell et al., 1990),*** protoelytic enzymes ***(Dinerman et al.,1990),*** and arachidonate derivatives ***(Mullane et al., 1987).***

Therefore, it is very important to elucidate the mechanism of neutrophil recruitment from the blood stream to the myocardium during and after ischemia and reperfusion ***(Yasunori et al., 1993)***.

Infiltration of the myocardium by neutrophils and myocytes/macrophages is observed in patients with coronary artery diseases. Interleukin-8 (IL-8) regulates and activates neutrophils in acute inflammation ***(Huber et al., 1991).*** Moreover, IL-8 has a regulatory role in ischemic and reperfused myocardium, and has been hypothesized to participate in nuetrophil-mediated myocardial injury ***(Kukielka et al., 1995).*** Recently, a highly sensitive enzyme immunoassay was developd for IL-8 ***(Tsugiyasu et al., 1996)***.