INTRODUCTION AND AIM OF THE WORK

Introduction:

Beta-blockers are competitive inhibitors, their action depending on the ratio of beta-blocker concentration to catecholamine concentration at beta-adrenoreceptor sites, blockade of cardiac $beta_1$-receptors cause a decrease in heart rate, myocardial contractility, velocity of cardiac contraction and myocardial oxygen demands (Watanabe, 1983).

Beta-blockade is associated with impaired triglyceride clearance, this most probably reflected reduced endothelial lipoprotein lipase activity (Day, et al. 1979).

The rise in concentraction of serum free fatty acids (FFA) which occurs with stress may result in increased hepatic production of triglycerides (Havel et al., 1980).

The stress of acute myocardial ischaemia in the presence of effective beta-blockade, so impaired triglyceride clearance will result in rise of serum triglyceride which may be sufficient to provoke acute pancreatitis (Durrington and Cairns, 1982).

Beta-blockade is a known cause of hypertriglyceridemia. Severe hypertriglyceridemia is a well recognized cause of acute pancreatitis. The relationship between hyperlipidemia and acute pancreatitis is unclear (Dickson, et al. 1984).

Aim of the work:

Hence the study of pancreatitis in patients with coronary heart disease under beta-blockade is essential as both give chest and abdominal pain and misdiagnosis may be a possibility.

Again persistance of pain after acute coronary attack under beta-blockade may be due to complicating pancreatitis (Durrington and Cairns, 1982).