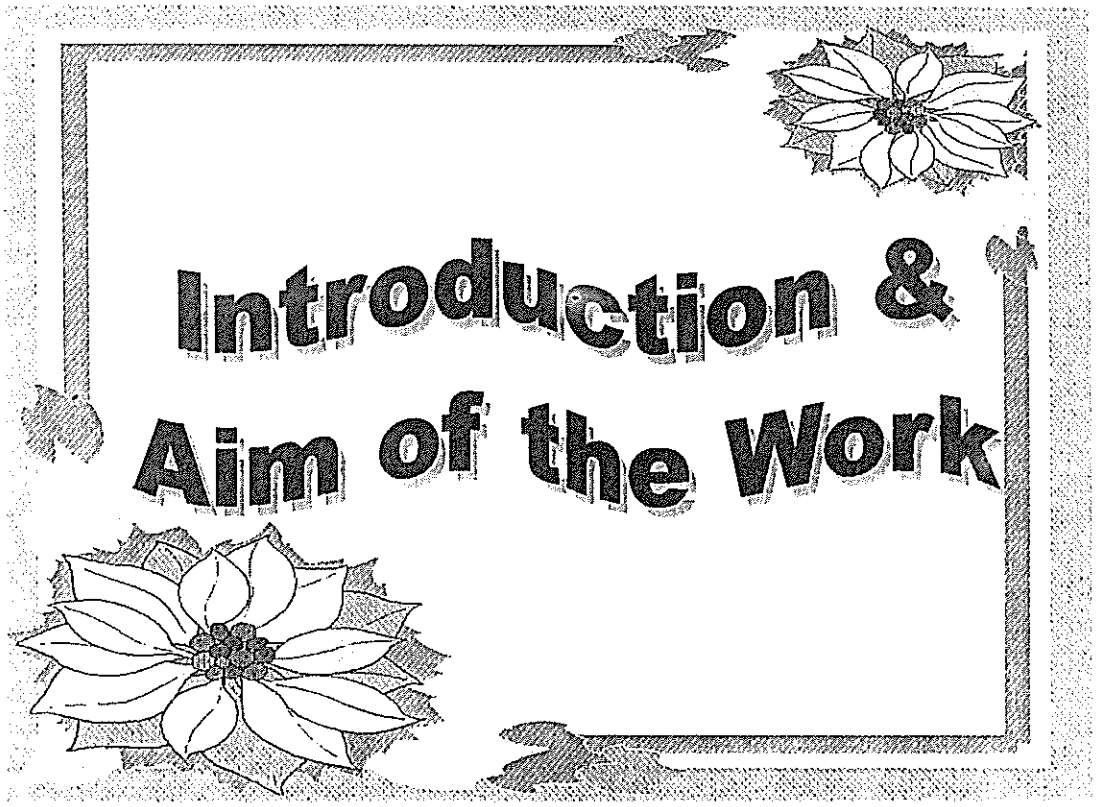
Introduction &
Aim of the Work

Introduction

Coronary heart disease is a major cause of morbidity and one of the leading causes of death. Coronary heart disease is most often the result of atherosclerosis in the epicardial coronary vessels.**(Brown, 1993).**

(John,1999)studied a consecutive group of patients who,after initial hospital discharge,underwent repeat coronary angiography for chest pain within 30 days of successful percutaneous coronary intervention(**PCI**).we hypothesized that the use of coronary stents,which inhibit elastic recoil and arterial remodeling(**Painter et al,1995**)would be associated with a lower incidence of early restenosis in these patients.

In the early 1970s, thrombolytic agents emerged as a major innovation in the pharmcologic treatment of intracoronary thrombi and acute coronary syndrmes. More over, because successful reperfusion in acute myocardial infarction often leaves salvaged mucle at future risk, large clinical trials have been organized to define the indications for angiongraphy and percutaenous transluminal coronary angioplasty **(PTCA)** after the use of thrombolytic agents **(kennedy JW, 1983).**

Angioplasty is found to lead to dissection of the arterial wall and subsequent vessel closure in a significant proportion of patients. Such acute occlusion has been said to occur in as many as 5% of patient. This finding, plus the problem of restenosis, which in some series is as high as 35% within the first few months after the procedure, has led to the development of intravascular stents to prevent restenosis after angioplasy **(Falke.,1983)**

In 1991 Schatz and Colleagues **(schatz RA. 1991)** reviewed the first 213 patients with successfully implanted stents in native coronary arteries. The restenosis rate at 4 to 6 months was 20% in patients with single stents and 50% in patients with multiple stents. Clinical restenosis (requiring repeat percutaenous transluminal angioplasty PTCA or By pass surgery) accured in approximately half of the patients with angiographic restenosis **(Braun wald, 1992)**.