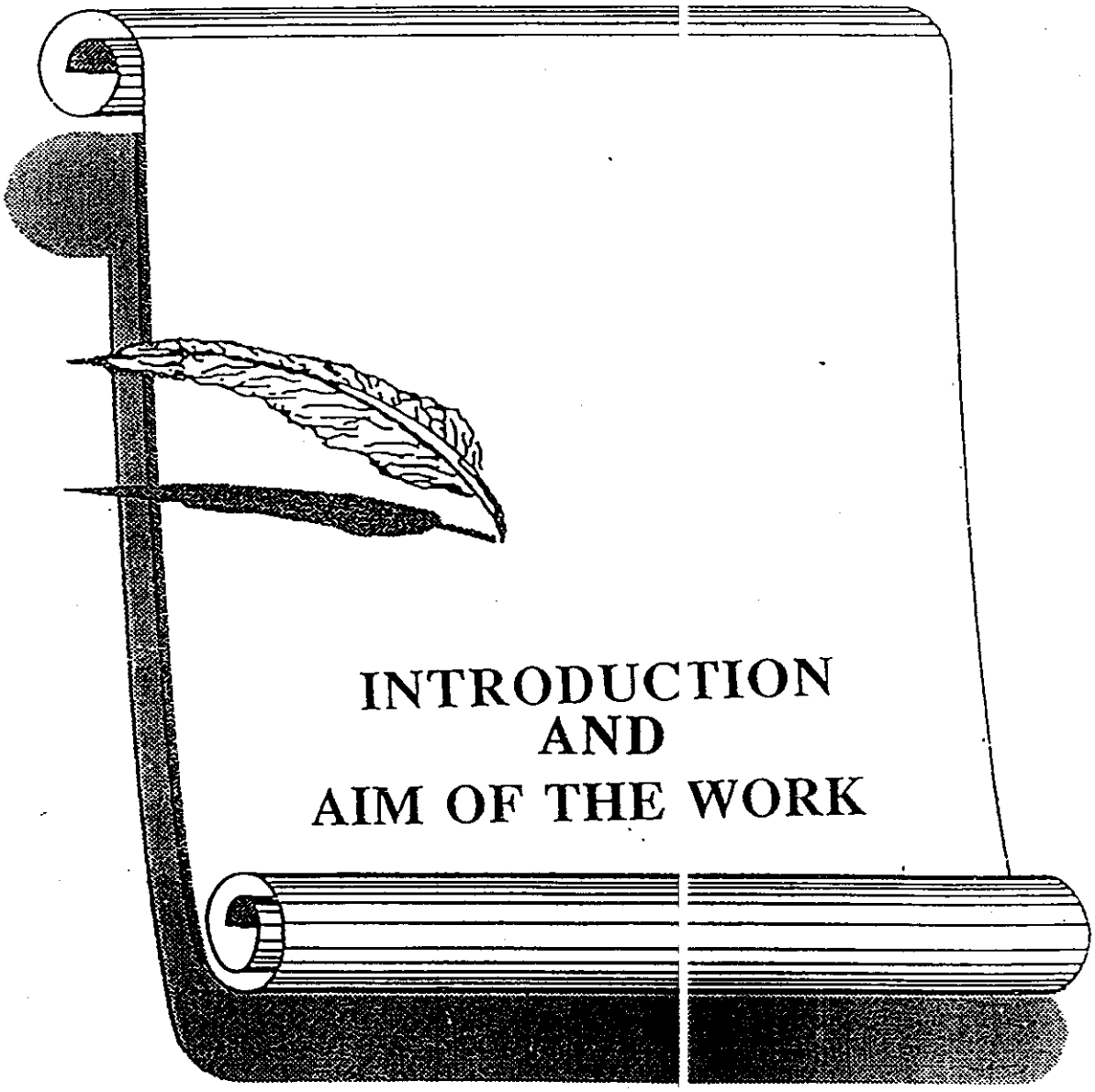

# INTRODUCTION AND AIM OF THE WORK

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Angiotensin II ( A II), as a vasoconstrictive and growth - promoting agent, appears to contribute to the development of atherosclerosis. A II is a mediator of hypertrophy in vascular smooth muscle cells, in addition, it enhances endothelial cell growth ( Geisterfer et al., 1988)..

Johnston et al., (1993) stated that all components of the renin- angiostensin system have been detected in the heart . Angiotensin Converting Enzyme (ACE) is located in discrete areas of the heart, including the cardiac valves, coronary vessels, atria and myocardium. After myocardial infarction, although plasma renin and aldosterone levels are not increased, ACE in the myocardium is markedly increased.

Laragh , (1992) reported that inappropriately high renin production may cause ischaemic vascular damage in the heart, kidney and brain, predisposing to infarction. Many clinical situations associated with high plasma renin levels are accompained by striking vascular damage, heart attack or stroke. A prospective study showed an equivocally positive relationship between myocardial infarction and high renin status regardless of other risk factors such as smoking, hypercholestremia or diabetes.

The aim of this work is to shed light on the possible role played by renin angiotensin system in the pathogenesis of ischaemic heart disease. Also to elucidate wheather or not this system can be considered as an independent risk factor for heart attack and stroke.