INTRODUCTION &

AIM OF THE WORK

INTRODUCTION

Cigarette smoking is a strong independant risk factor for cardiovascular disease in adults. Arterial thrombosis has been shown to occur with increased frequency in cigarette smokers. There is an increased risk of myocardial infarction, cerebrovascular accident and sudden death. In the presence of established arterial disease, cessation of smoking has resulted in decreased cardiovascular morbidity and mortality rates Wilhelmsson et al., 1984).

Smokers also have an increased risk of developing occlusive peripheral vascular disease and persistant smoking after development of the disease, makes successful treatment difficult. The reasons, however, for this association are not always clear. Various suggestions link endothelial damage by constituents of tobacco smoke, increased carboxy Hb level, enhanced platelet aggregation, accumulation of fibrin and binding of low density lipoprotein with the development of astheroscterotic lesions (Dintenfass, 1975, Jenkins et al., 1978 and Tell et al., 1985).

Moreover, further studies in later years have postulated that smoking has an important pathological role and several deleterious effects in deterioration

of blood rheology and blood flow properties and this may be another contributing factor. It also seems that haemorheological response to harmful effects of smoking is dose related (Ernest, 1987).

AIM OF THE WORK

The aim of this work is to study the acute and chronic effects of smoking on viscosity and some of its major determinants in apparently healthy smokers, which might explain the increased incidence of peripheral vascular disorders among smokers. It is to be noted that this study will include not only the smoker himself (active smoker) but also those sitting with him and passively affected by smoke (passive smoker).