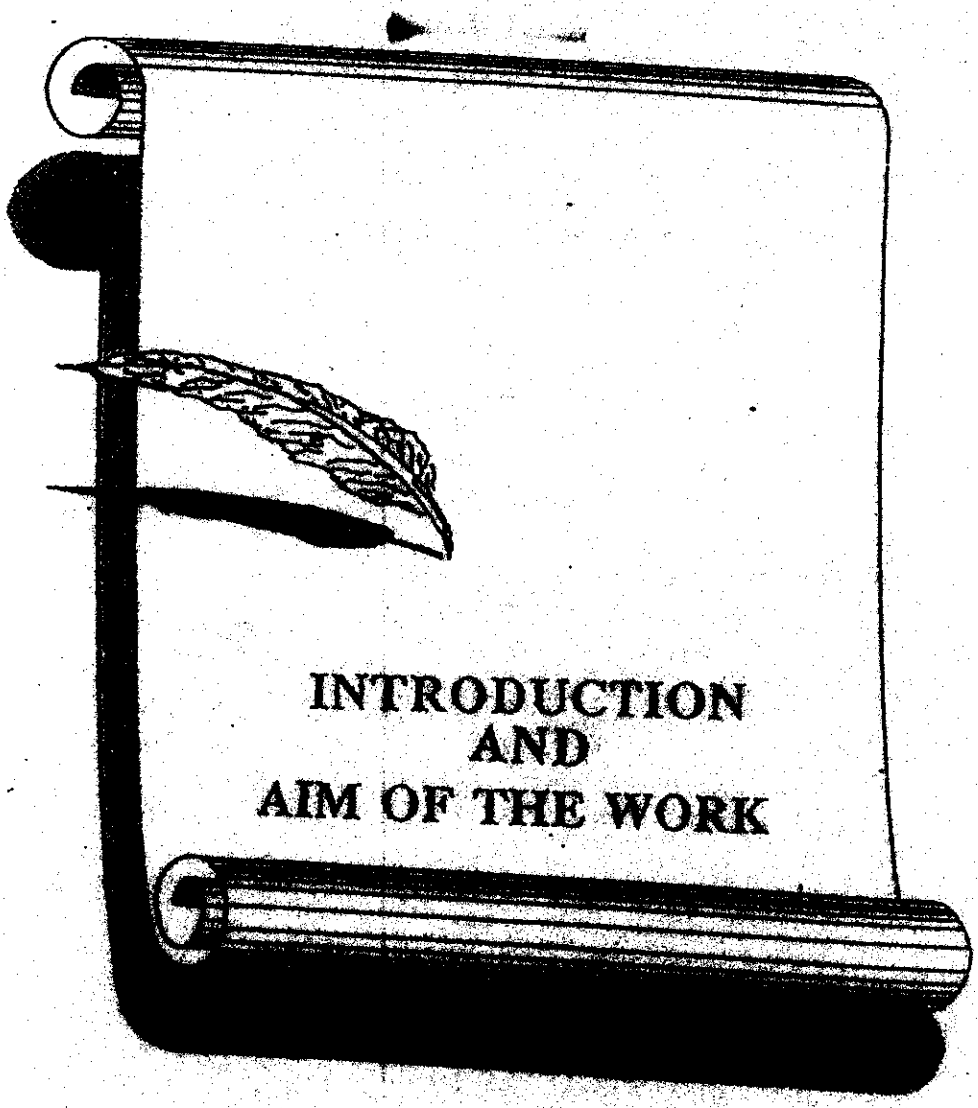

INTRODUCTION AND AIM OF THE WORK

INTRODUCTION

Portal hypertension is a major complication of cirrhosis and is responsible for many clinical manifestations observed in patients with advanced liver failure. 'When the liver is full of fluid and this over flows into the peritoneal cavity so that the belly becomes full of water death follows' ***(Hypocrates., ca. 400 B.C).***

While expecting portal pressure to be lower by development of collaterals deviating blood into systemic veins, the portal pressure is maintained by increasing the blood flow in the portal system which becomes hyperaemic ***(Bocsh et al., 1992).***

This increased blood flow is achieved by raising cardiac output and splanchnic vasodilatation, however, the actual portal flow reaching the liver is of course reduced ***(Corszman , 1994).***

The factors involved in the pathogenesis of this hyperdynamic state can be categorised into factors leading to enhanced vasodilatation and factors reducing the sensitivity to vasoconstrictors together with hypervoleamia ***(Mac Mathura et al., 1992).***

Portal hypertension may be complicated by ascites which may occur around the time of portal hypertension (PHT) or later on ***(Mowart , 1994).***

A significant controversy regarding the pathogenesis of ascites exists ,the results of all reports agree that there is abnormal renal sodium retention and increase in plasma volume which may even precede the appearance of ascites and

systemic heamodynamic changes which require the stimulation of several factors *(Longmire- cook ., 1993).*

Renin angiotensin aldosterone system is one of the principle and major regulatory factors in the maintenance of extracellular fluid, blood volume and arterial blood pressure. This results from the sodium retaining effect of aldosterone on the kidneys and the vasoconstrictor properties of angiotensin II. In addition, angiotensin II may also have a direct renal sodium retaining effect *(Wilkson & Williams., 1980).*

In contrast to the previous factors, the atrial natriuretic peptide decreases blood pressure, increases glomerular filtration rate, and sodium retention without increase in renal plasma flow, and exerts an inhibitory effect on renin secretory rate and serum aldosterone levels (*Brenner et al ., 1990).* However many factors affecting Renin - angiotensin aldosterone system in cirrhosis and these factors are affected by salt and water retention.

So, the aim of this work is to study the renin angiotensin aldosterone system in cirrhosis with portal hypertension in addition to studying changes in atrial natriuretic peptide level which may be involved in portal hypertension.