INTRODUCTION

The cost of the renal replacement therapy to the community and the patient, and the quality of life of patients receiving this therapy argue for prevention rather than treatment of end stage renal failure.

The amount and quality of diet seem to be a very important element in progression of renal failure. Several experiments have shown that the deterioration of renal parenchyma after reduction of functional mass is affected by the protein content of the diet. The respective role of protein and that of other nutrients that vary with proteins were never clearly separated (Laouari et al., 1983).

Schrier (1983), hypothesized that ischemic insult in acute renal failure will decrease the mitochondrial energy production and lead to an increase in cytosolic and mitochondrial calcium ion which could be responsible for renal vasoconstriction and callular necrosis. Verapamil is one of calcium antagonist that prevent the influx of calcium ions into the cells. This action on the vascular smooth muscle cells could be benificial in preventing the vascular spasm caused by anoxia (Burke et al., 1981).

The mechanism of this protective effect of verapamil is still controversial : Lekuni et al., (1979) suggested the reversal of action of angiotensin II on the glomerular microcirculation. Van Neuten et al., (1980) suggested that verapamil prevents Ca influx into the red blood corpuscles,

thus preventing its malformation and thus keeping microcirculation and less tissue hypoxemia.

In addition verapamil has been demonstrated to decrease functional deterioration and mortality in the partially nephrectomized model of chronic renal failure (Harris et al., (1987).

AIM OF THE WORK :

The aim of the present work is to study the effects of protein diets and also to study the effects of verapamil on progression of experimental chronic renal failure in rats.