INTRODUCTION AND AIM OF THE WORK

INTRODUCTION

Elevated levels of 1.25- dihydroxy Vitamin D and PTH shift intracellular cation levels and stimulate cellular calcium uptake from the extracellular space. As such, these calcium regulating hormones may be responsible, at least in part, for salt – sensitive hypertension ***(Resnick et al., 1991).***

Diez et al. (1991) suggested that an excess parathyroid hormone may be involved in the increase of Ca^{++}- dependent K^{+} efflux present in some essential hypertensive patients.

Elevated levels of PTH are characteristic of the low –renin or salt sensitive state, or both, and may contribute to the hypertensive process. Elevated PTH activity found in normotensive subjects may pressage the development of low renin, salt sensitive hypertension ***(Resnick et al., 1993).***

Abnormalities of Ca^{++} metabolism independent of changes in intracellular Ca^{++} have been described in patients with essential hypertension. These include increased urinary excretion for a given salt intake, a raised PTH level, an increase in urinary cyclic AMP, a tendency for a low serum ionized Ca^{++} level, a raised 1.25 Dihydroxy Vitamin D and an increased intestinal Ca^{++} absorption ***(MacGregor and Cappuccio, 1993).***

Takagi et al., (1991) Suggested that supplementation of dietary calcium may contribute to a reduction of blood pressure in elderly patients with essential hypertension.

AIM OF THE WORK

The aim of this work is to study the calcium homeostasis in essential hypertension through evaluation of calciotropic hormones (PTH and 1.25 (oH)2 D) and their relations to renin level in a trial to shed some lights on the mechanisms played by these hormones in the pathogenesis of essential hypertension.