CHAPTER 1

Introduction

It is well recognized that amino acid loading can cause acute rise of the glomerular filtration rate (GFR) and the renal plasma flow (RPF) in animals (Johannesen et al., 1977 ; Meyer et al., 1983 ; Woods et al., 1986 ; Woods et al., 1987) and human (Appiani et al., 1988 ; Castellino et al., 1986 ; Castellino et al., 1987 ; Castellino et al., 1988 ; Hirschberg et al., 1987 ; Hirschberg et al., 1988 ; Ruilope et al., 1987 ; Ter Wee et al., 1985), through renal vasodilatation. The mechanism of an amino acid induced renal vasodilatation was suggested to be the same mechanism of an oral protein induced renal vasodilatation (Brenner et al., 1982). Despite numerous reports of these effects, little is known about the mechanism responsible for renal vasodilatation Mechanisms operated through hormones (Castellino et al., 1986 ; Castellino et al., 1987 ; Castellino et al., 1988 ; Hirschberg et al., 1987 ; Ruilope et al., 1987), the liver (Alvestrand et al., 1984), macula densa and tubuloglomerular feedback (Appiani et al., 1988 ; Woods et al., 1986) have been proposed. Because of conflicting results the role of prostaglandin, as the cause of renal vasodilatation, is questionable (Herrera et al., 1988 ; Hirschberg et al., 1988, ; Hostetter, 1986 ; Ruilope et al., 1987). In previous reports the role of systemic

hemodynamics had not been studied. Amino acids might have the effects on the cardiac output or peripheral vascular resistance , which could in turn affect renal hemodynamics. Furthermore , amino acids may have effects on both systemicand renal hemodynamics. The cardiac output and peripheral vascular resistance may be affected leading to renal hemodynamic changes. This investigation was therefore conducted to explore the effects of arginine (ARG) on both systemic and renal hemodynamics and to study the effect of indomethacin on arginine induced hemodynamic changes.