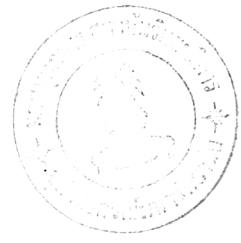

Chapter I

Introduction and Aims

Acute renal failure (ARF) occurs in many clinical situations such as septicemia, hemorrhagic shock, severe hemoglobinuria, myoglobinuria, etc. Several reports in the past showed acute renal failure following envenomation of Russell's viper (Sitprija and Boonpucknavig, 1974; Harris et al., 1976; Shastrey et al., 1977). The pathophysiology of ARF following viper envenomation remains uncertain despite a number of experiments and clinical investigations (Aung - Khin, 1978). No evidence is available so far to indicate if the changes in kidney functions are due to direct toxic action of the venom by toxin or if it occur indirectly as a consequence of shock, disseminated intravascular clotting, vasculopathies singly or in combination. Pathophysiology of the venom on renal functions in human was undertaken by studying the clinical, biochemical and renal histopathological profiles in patients who suffered from ARF following snake bites (Chugh et al., 1975). Previous attempts to design the experiments have failed because of high animal mortality (Benyajati et al., 1974).

It has been reported that intravenous infusion of epinephrine caused severe acute tubular necrosis with a marked increase in packed cell volume (PCV) (Mandal et al., 1978). Renal damage was infrequent and less severe in chronic splenectomized dogs with absence of PCV increment (Mandal et al., 1978; Bell et al., 1981). This renal protection

in splenectomized dogs might be mediated by prostaglandin, an effective vasodilator and lack of elevation of arterial PCV. This hypothesis was further attested by reversal of the renal protection after pretreatment with indomethacin, a potent prostaglandin inhibitor, before epinephrine infusion (Mandal, 1982).

It has been found in the dog that Russell's viper venom causes a decrease in renal function after envenomation. The changes were associated with increases in PCV and blood viscosity (Tungthanathanich, 1983). To our knowledge there has been no study of the effects of Russell's viper venom on the pathophysiology of ARF in intact or splenectomized dogs, including the protective mechanism of prostaglandin. The present study was conducted to gain deeper insight into the mechanisms responsible for the development of ARF following Russell's viper envenomation. Cardiovascular and renal functions were investigated in dogs to determine whether changes in renal functions during envenomation were due to the direct action of venom toxin or secondary from circulatory hemodynamic changes.