CHAPTER I

Introduction and Aims

Acute renal failure (ARF) occurs in many clinical situations such as hemorrhagic shock, septicemia, severe hemoglobinuria etc. Many reports showed that Russell's viper envenomation are followed by acute renal failure (Sitprija and Boonpucknavig, 1974 ; Harris et al, 1976; Shastry et al, 1977). Pathophysiology of venom on renal functions in man was studied by the observation from the clinical, the biochemical tests and renal histopathology in the patient who suffered from ARF following snake bites (Chugh et al, 1975). Previously,the attempts to design the experiments in animals were failed because of the high mortality (Benyajati et al, 1974).

It has been known that an intravenous infusion of epinephrine causes of severe acute tubular necrosis with a marked increase in packed cell volume (PCV) (Mandal et al, 1978) but the 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 protection might be mediated by prostaglandins, an effective vasodilator and without PCV elevation. The hypothesis was attested by reversal of the renal protection after pretreatment with indomethacin, a potent prostaglandin inhibitor before epinephrine infusion (Mandal, 1982; Tongvongchai, 1984).

Intravenous administration of Russell's viper venom in dogs has been shown a marked changes of both renal functions and general

circulations. The association in the decrease in renal blood flow and cardiac output during the initial period after envenomation was observed (Tungthanathanich, 1983; Chaiyabutr et al., 1984). It has been shown by Vick et al (1967) that Russell's viper venom produced a pooling of blood in hepato-splanchnic bed in the dog, following by a marked reduction of arterial blood pressure. These supporting evidences are incomplete, since there was an increase in blood pressure following the transient decrease and the reduction of plasma volume was not remarkable during envenomation (Chaiyabutr et al., 1984). An intrarenal mechanism, particularly renin-angiotensin system, has also been reported to be responsible for the reduction of renal blood flow and filtration rate after envenomation (Chaiyabutr, 1985). However, the mechanism for renin release may also be involved by acute plasma volume expansion (Roy et al, 1985). Therefore, the mechanisms of pathophysiological changes between renal functions and general circulation are still opened to question.

The present study aimed to elucidate the possible role which might take part in the development of ARF whether changes in renal functions after envenomation were due to the direct action of venom toxin or secondary to the circulatory hemodynamic changes.