CONCLUSION

1. Studies on isolation, toxicology and pharmacology of venoms have been made with Indian snakes, viz., cobra (Naja naja) and Russell's viper (Vipera russelli).

2. Toxicity studies on both the venoms of cobra and Russell's viper indicate that mortality of the animals due to crude cobra venom is highest in the calotes, followed in decreasing order in mice, rabbit, cat, rat and toad. In case of crude Russell's viper venom, mortality of animals is also highest in calotes, followed in decreasing order in mice, rabbit, cat, rat and toad. Toads of average body weight of about 100 g can tolerate 40 mg of cobra and 20 mg of Russell's viper venoms, if injected intramuscularly.

Toxicity studies in mice with venoms heated at different grades of temperature indicate that the cobra venom loses its toxicity if heated over 100°C, whereas Russell's viper venom loses its toxicity if heated at 80°C.

3. Pharmacological and physiological studies with heat-treated venoms demonstrate that cobra venom solution heated at 100°C for 30 minutes show toxicity presumably due to presence of neurotoxin, but not of cardiotoxin, as evidenced from the studies on isolated amphibian heart on blood pressure and respiration and also on electrocardiographic appearances in rabbits. Such studies with Russell's viper venom heated at 100°C for 30 minutes indicate no such toxicity, instead a sustained respiratory stimulation is observed. This respiratory stimulatory factor (R.S.F.) can permanently resuscitate the respiratory failure induced by cobra venom solution heated at 100°C for 30 minutes. This stimulatory factor cannot do so permanently to the respiratory failure, induced by crude cobra and Russell's viper venoms, only because of irreversible cardiac damage.

4. Isolation through Sephadex G-50 provides separation of three factors of low molecular weight protein from both the venoms heated at 100°C for 30 minutes.

Of the three factors isolated from cobra venom, Factor C-I possesses vaso-depressor property and acts through adrenergic β-receptors and Factor C-II shows transient respiratory inhibition, which is presumably due to presence of neurotoxin in lower concentration.

Among the factors isolated from Russell's viper venom, Factor V-I shows sustained vaso-depressor property, which is abolished after atropinization. Other two factors have got no actions on blood pressure. But the respiratory stimulatory factor (R.S.F.) detected in the venom solution heated at 100°C for 30 minutes is found only in the mixture of the three factors in equal volume. None of the factors, separately or two together, is effective in such respiratory stimulation. This respiratory stimulatory effect is abolished by β-blocker but remains unaffected by vagotomy and atropinization.

The above respiratory stimulation is unlikely to happen through reflexogenic pathways, but very likely through centrogenic pathways, which may have some role particularly for increase in the rate of respiration and β-receptors of bronchial muscles are presumably responsible for the increased depth of respiration by decreasing the airway resistance by bronchial relaxation. Although subject to further study, the respiratory stimulatory factor may possibly play a role in the management of bronchial asthma of β-receptor origin. Similarly Factor V-I having sustained vaso-depressor property may be a weapon in the management of hypertension.