SUMMARY AND CONCLUSION

Serum levels of FSH, PRL and testosterone increases after 14 days and 21 days of ventral prostatectomy but no change is observed after 3 days and 7 days of removal of the ventral prostate in rats. Testicular $\Delta^5 -3\beta$-HSD and $17\beta$-HSD activities increase after 14 days and 21 days of prostatectomy but after 3 days and 7 days, no change is observed. Prostatectomy increases both acid and alkaline phosphatase activities in testis following 14 days and 21 days of prostatectomy but no change is noticed following 3 days and 7 days of surgery. On the other hand, prostatectomy also stimulates the adrenal $\Delta^5 -3\beta$ -HSD activity along with the rise of serum corticosterone after 14 days and 21 days of prostatectomy. So these studies lead to the conclusion that ventral prostate may contain some principle which has an effect on testicular and adrenal steroidogenesis by inducing FSH, PRL and corticosterone secretion. The stimulation of testicular steroidogenesis following ventral prostatectomy is possibly secondary to the increased serum FSH and PRL or may be primary due to the existence of some testicular inhibitory factors.

For exploring the effect of prostatic principles on testicular and adrenocortical function, aqueous ventral prostatic extracts were used. It appears that supplementation of aqueous prostatic extracts to prostatectomized rats prevents the prostatectomy induced increase in testicular $\Delta^5 -3\beta$-HSD and $17\beta$-HSD activities. Serum levels of FSH, and PRL and testosterone
decrease following supplementation of aqueous prostatic extracts in prostatectomized rats. Testicular acid phosphatase activity is restored following supplementation of aqueous extract. It indicates that aqueous prostatic extracts contain some principles which exert regulatory influences on testicular steroidogenesis either directly or through modulating FSH and PRL secretion. The restoration of testicular steroidogenesis may be secondary to the decrease in FSH and PRL release. Fall of serum corticosterone along with decreased adrenal $\Delta^{5}-3\beta$ -HSD activity after supplementation of aqueous extract of prostate gland indicates that prostatic principles can inhibit adrenal corticosterone production possibly by modulating PRL secretion. Testicular acid phosphatase activity is decreased following supplementation of aqueous prostatic extract to prostatectomized rats. This restoration of acid phosphatase activity is possibly due to the restoration of germ cells within the seminiferous tubules.

To ascertain the effects of solvent extract of ventral prostate on testicular functions, ether extract of homogenized ventral prostate was supplemented to prostatectomized rats. It appears that solvent extract of prostate gland has no effect on recovery of prostatectomy induced testicular and adrenocortical stimulation, indicating that prostatic principles involved in the regulation of testicular and adrenocortical functions are non-lipid substances.

From the present studies a major problem arises ——
whether the ventral prostatic principles have any direct effect on testicular and adrenal steroidogenesis. For further understanding of this problem, in vitro studies were performed using testicular and adrenal slices taken from normal rats. It appears that ammonium sulphate fractionated ventral prostatic peptide (60-90% saturation; fraction III) at lower dose is without effect on testicular steroidogenic enzymes. But, when the concentrations were increased, inhibition of testicular steroidogenic enzymes are observed. But, other two fractions (0-30% saturation; Fraction I and 30-60% saturation; Fraction II) have no effect on testicular steroidogenic enzymes. On the other hand, all the three fractions of prostatic peptides have no effects on adrenal steroidogenesis in in vitro. Thus, it may be concluded that Fraction III of ventral prostatic peptides probably exerts a direct inhibitory influence on testicular steroidogenesis.

For confirming the effects of Fraction III of ventral prostatic peptides, in vivo experiments were carried out after supplementation of this fraction in prostatectomized rats. It appears that Fraction III can prevent the prostatectomy induced stimulation of testicular and adrenal steroidogenesis and fall in serum levels of FSH and PRL. This experiment leads to the conclusion that Fraction III has inhibitory effect on
secretion of PRL and FSH which in turn may inhibit the testicular androgenesis. Thus, Fraction III of ventral prostatic peptide may exert its inhibitory effect either by direct (at testicular site) or by indirect (involving pituitary FSH and PRL secretion) manner or by both the ways.

Studies on immature rats reveal that prostatectomy at 35 days of age causes inhibition of testicular steroidogenesis in association with fall in serum levels of FSH, LH and PRL at 50, 55 and 60 days of age. It indicates that in immature state ventral prostate is necessary for maintaining the normal testicular functions. Thus ventral prostate exerts its biphasic action on testicular steroidogenesis depending on the age of the rat.

In order to correlate the influence of prostate gland on steroidogenesis and spermatogenesis, quantitative studies of gametogenesis were carried out in absence of prostate gland. The overall effects of prostatectomy following 14 days and 21 days are essentially same — the spermatocyte to spermatid conversion process is decreased. In absence of ventral prostate, increased FSH and PRL secretions are possibly responsible for germ cell degeneration specially at spermatid level. Spermatogenic inhibition, caused by prostatectomy can be reversed by supplementation of aqueous extract of ventral prostate or by administration of Fraction III of ventral prostatic peptides. The
restoration of spermatogenesis following supplementation is possibly the effects of restoration of FSH and PRL secretion. Ventral prostate removal in immature condition causes inhibition of spermatogonia, spermatocytes and spermatids at 50, 55 and 60 days of age. This gametogenic inhibition is possibly due to the inhibition of pituitary gonadotrophin and testicular testosterone formation. Thus, the inhibition of spermatogenesis resulting from removal of ventral prostate in immature and mature state may be due to two different mechanisms; in the former case it may be a consequence of pituitary gonadotrophin inhibition while the later may be due to the hypersecretion of FSH and PRL.