CONCLUSION
CONCLUSION

In view of the controversies regarding the implication and association of various vasoactive principles liberated from perivascular mast cells with hypertensive disorders the present study was undertaken to investigate whether mast cells exert any influence over the blood pressure level. It has been demonstrated that histamine, serotonin and heparin released from perivascular mast cells in kidney, lung, spleen and mesentery in the experimental groups (Goldblatt preparation) showed elevation in their contents in respect to the control groups. Moreover, analysis of nor-adrenaline content in tissues and serum of those experimental animals revealed a significant rise when compared with the control groups. As it is more or less well established that serotonin and noradrenaline are potent vasoconstrictors and histamine and heparin are potent vasodilators it is expected that there should have been no change in blood pressure level. As obtained from the observations made from the present study the observed hypertension in the experimental group may be attributed to:

(a) the biphasic action, i.e. both pressor and depressor action, of histamine liberated from perivascular mast cells.
(b) Besides, as there is a rise in DAO activity and
DAO being the enzyme responsible for degradation
of histamine it is quiet justified to suggest
that rise in DAO has some influence in elevating
blood pressure level by diminishing the depressor
action of histamine. Moreover, evidences suggest
that heparin can stimulate the release of DAO. Thus
it can be assumed that increase in heparin
concentration causes an increase in DAO release
that catabolises histamine (Vasodilator) and
thereby causes an indirect reflection in the blood
pressure level i.e. an elevation.

(d) However, evidences exist to support that histamine
and serotonin can potentiate noradrenaline. Thus it
can be surmised that increase in noradrenaline
concentration may be due to increase in histamine
and serotonin level. NA by its vasoconstrictor action
increases peripheral resistance and there by increases blood pressure.

(e) The increased triglyceride and cholesterol contents
in serum may also responsible for elevating
blood pressure level since deposition of triglyce-
ride and cholesterol can produce atherosclerotic
changes which inturn indirectly cause hypertension.
However, the role played by renin angiotensin system in creating hypertension cannot be eliminated though there are interesting reports which suggest that serotonin and histamine released from mast cells can potentiate the vasoconstrictor effects of angiotensin II.

Thus, it can be concluded that some of the vasoactive and chemotactic principles liberated from perivascular mast cells are directly or indirectly involved in the alteration of blood pressure. However, well-drawn future experiments only can confirm the cause and effect relationship between hypertension and the different chemotactic principles released from the perivascular mast cells.