INTRODUCTION
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Hypertension is a frequent, world wide health disorder and can be classified into primary and secondary hypertension. Essential or primary hypertension is the most prevalent form accounting for ninety percent of all cases of hypertension. Hypertension is essential when the causes are generally unknown while it is classified as secondary when some other disease process or abnormality is involved in its possession (Park & Park, 1991).

Blood pressure is a continuously distributed variable in population and there is no sharp dividing line between high and normal blood pressure. So there is ambiguity of what is normal blood pressure.

Hypertension is generally characterised by elevated blood pressure persistently exceeding 140/90 mmHg. Essential hypertension is characterised by the presence of sustained diastolic pressure in excess of 95 mmHg. Essential hypertension has no single identifiable cause but the risk of disorder is increased by obesity, high serum sodium level, hypercholesterolemia, family history of high blood
pressure etc. In essential hypertension cardiac output is close to normal but there is elevation of blood pressure due to increase in peripheral resistance (Frohlick et al, 1970).

Hypertension may be secondary to a variety of clinical conditions affecting specific organs:

1. Renal hypertension is produced by renal diseases. The main sub-division of renal hypertension are renovascular hypertension including pre-eclampsia and eclampsia and renal perenchymal hypertension. The possible cause for renal vascular hypertension is decreased perfusion of renal tissue due to stenosis of a main or branch of renal artery which activates renin-angiotensin system (Frohlick, 1971). Circulating angiotensin II elevates arterial blood pressure by direct vasoconstriction, by stimulation of aldosterone secretion with resultant sodium retention or by stimulating the adrenergic nervous system. In case of renal parenchymal hypertension, the decreased perfusion of renal tissue results from inflammatory and fibrotic changes involving multiple small intrarenal vessel, (Lüscher et al, 1985).
2. Endocrine hypertension a). In Cushing's syndrome hypertension is produced either due to sodium retaining effect caused by large amounts of glucocorticoids or due to increased level of mineralocorticoids (Bravo et al, 1973). b) Conn's syndrome or primary aldosteronism associated with overproduction of aldosterone by an adrenal tumour or hyperplasia (Conn, 1964). c) In pheochromocytoma, hypertension is caused due to excessive amount of catecholamines, produced by chromaffin cell tumour (Tarazi et al, 1970). d) Toxemia of pregnancy is a hypertensive state characterised by sodium retention, generalised vasoconstriction and glomerulonephritis (Guzick, 1987). e) Hypertension may be also due to use of estrogen containing oral contraceptives (Vessey et al, 1989).

3. Hypertension may occur due to aortic coarctation (Gross, 1964)

Malignant hypertension is characterised by a diastolic pressure higher than 120 mmHg, severe headache, blurred vision and confusion resulting to fatal uremia, myocardial and congestive heart failure (Frohlick, 1991). For a long time considerable interest has been focussed to understand and evaluate the role of different factors interplaying during hypertension.
It is well established that the areas of central nervous system involved in blood pressure control are mainly brainstem area, but it extends from hypothalamus to the thoracic spinal cord (Peiss, 1965). Though nervous control provides a background tone on arterial vessels, several reports also support that the tissue metabolites like histamine, serotonin, heparin, carbon-dioxide and various kinins also possess significant importance in controlling blood pressure. Moreover, the reflex responses arising from the baroreceptors, depend on the changes of the calibre of arteries controlled by tissue metabolites histamine, serotonin, etc. (Hilton, 1962). Following a complete arterial occlusion or following vigorous contraction a phenomenon of reactive hyperemia is observed which means that initially the blood flow increases markedly and then returns to normal level. Accumulation of local metabolites like histamine, serotonin etc. have been given more importance for causing this hyperemia (Gault et al, 1966).

Several evidences exist which suggest that mast cells in different perivascular tissues like lungs, stomach, spleen, kidney, mesentery etc. possess significant role in regulating hypertension by releasing several vasoactive and chemotactic principles like
histamine, serotonin, heparin, prostaglandins, bradykinin etc. (Purcell et al, 1989).

Cabanie and Godfraind (1988) reported that histamine present in all perivascular tissues, has a biphasic role on peripheral vessels - a constrictor action on large arteries and a vasodilator action on resistance vessels, arterioles and capillaries and its effect on blood pressure dependent on the balance between the constrictor actions of large vessels and dilator action of capillaries (Feldberg, 1927, Russell et al, 1994).

Serotonin also released from mast cells during their activation and taken up by blood platelets from which it is released was shown to exhibit a vascular effect. Though it is a potent vasoconstrictor, its vasodilator effect has also been noticed (Chandra and Chandra, 1993).

Besides histamine and serotonin, heparin is also known to be released from the mast cells and several investigators support the vasodilator effect of heparin (Theodore, 1986).
Since there are considerable controversies in the existing literature regarding the influence of mast cell population on hypertension, the present study was undertaken to evaluate and assess the role played by mast cells present in different perivascular tissues in experimental hypertension, a state stimulating secondary hypertension.

It is well known that granular contents of perivascular mast cells decrease with the release of different chemical mediators like histamine, serotonin etc. It has been suggested that these substances are closely related with the regulation of blood pressure possibly by influencing the peripheral resistance. Perivascular mast cell population therefore demands attention in view of the possibility of release of chemical mediators from them being involved in the genesis and maintenance of systemic arterial hypertension.

The present investigation has been planned to find out whether a good correlation can be drawn between the perivascular mast cell population and hypertension produced in experimental animals and to explore the role played by histamine, serotonin, heparin etc. on the cardiovascular system to investigate the mechanism involved.