Review Of Literature
REVIEW OF LITERATURE

The end of natural history of untreated hypertension is an increased likelihood of premature disability or death from cardiovascular diseases. The pathogenesis of hypertension involve structural changes in the resistance arterioles described under term remodeling and hypertrophy. Hypertrophic remodeling clearly develops in larger arteries as an early manifestations of essential hypertension (Tice et al, 1996), with close symmetry between vascular and cardiac hypertrophy (Romon et al, 2000; Vaudo et al, 2000).

Diastolic and systolic dysfunction have been observed early in the course of hypertension and either or both may lead to heart failure. Such diastolic dysfunction may reflect more vigorous atrial emptying (Ahmed et al) or abnormal diastolic relaxation (de Simone et al, 2000). The earliest function cardiac changes in hypertension are in left ventricular diastolic function, with lower E/A ratio and longer isovolemic relaxation time (Aeshbacher BC, Hutter D; Fuhrer J; et al: Am J Hypertension 14: 106, 2001).

Left ventricular hypertrophy (LVH) is detectable in 25%, 35% of all hypertensive patients and in 1% to 9% of normotensive
individuals. When present concomitant to hypertension, LVH is initially a useful compensatory process that represents an adaptation to increased ventricular stress. However, LVH is also the first step toward to development of overt clinical disease such as congestive heart failure, cardiac dysrhythmias and ischemic heart disease.

Current standard for defining and diagnosing hypertension rests on blood pressure levels which confers an increased risk of developing a morbid cardiovascular event and/or will clearly benefit from medial therapy.

In adults according to VIIth report of joint national committee for prevention, detection, evaluation and treatment of high blood pressure the following values are now considered.

**Systolic blood pressure :**

<table>
<thead>
<tr>
<th>Below 120 mmHg</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>120 mmHg - 139 mmHg</td>
<td>Prehypertension</td>
</tr>
<tr>
<td>140 mmHg – 159 mmHg</td>
<td>Hypertension, Stage 1</td>
</tr>
<tr>
<td>160 mmHg or above</td>
<td>Hypertension, Stage 2</td>
</tr>
</tbody>
</table>
Diastolic blood pressure:

<table>
<thead>
<tr>
<th>Blood Pressure Range</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Below 80 mmHg</td>
<td>Normal</td>
</tr>
<tr>
<td>80 mmHg - 89 mmHg</td>
<td>Prehypertension</td>
</tr>
<tr>
<td>90 mmHg - 99 mmHg</td>
<td>Hypertension, Stage 1</td>
</tr>
<tr>
<td>100 mmHg or above</td>
<td>Hypertension, Stage 2</td>
</tr>
</tbody>
</table>

Blood pressure was measured on two separate occasions under the proposed near ideal condition before labeling a patient as hypertensive.

Values of systolic or diastolic BP whichever was higher pressure was taken for staging.

**Diastolic dysfunction precedes systolic dysfunction in hypertensive patients**

Earliest functional cardiac changes in hypertension are in left ventricular diastolic dysfunction, with prolongation and in coordination of isovolumic relaxation time and lower E/A ratio (Braunwald 967, Dibello V. et al, 1999).

Arterial hypertension associated to LV concentric remodeling is the determinant of diastolic dysfunction but several other cardiac diseases, including myocardial ischaemia, and extra cardiac
pathologies involving the heart are other possible cause (Mausizio Cralderisi, Cardiovascular ultrasound 2005, 3:9).

Systolic cardiac function at rest is usually preserved in hypertension, however, diastolic function may be frequently altered (Nogureira JB, Acta Med Port, 1992 May; 5(5): 269-73.

**Mechanism of L.V.H. in systemic hypertension**

Pathogenesis of LVH involve a number of variables other than the pressure load, one of which is hemodynamic volume load.

When the heart faces a hemodynamic overload, the major compensation is an increase in muscle mass (Lorell and Carbello, 2000).

LV mass has been found to be more closely related to systolic than to diastolic BP; the opposite is true for LV wall thickness (Schmieder and Messerli 2000).

Neurohormonal responses involving both the sympathetic and rennin angiotensin systems may be recruited to increase contractility and participate in hypertrophic response. Aldosterone increases collagen content and thereby influences adaptation and structural remodeling, independent of BP levels (Weber et al, 1994). Evidence for a critical role of the rennin-angiotensin system
is the close correlation of their circulating levels of LV mass (Harrap et al, 1996; Schmieder et al, 1996).

Different patterns were found by echocardiography in hypertensive patients 19% had normal geometry; 11% concentric remodeling; 47%, eccentric hypertrophy; and 23%, concentric hypertrophy (Wachtel et al, 2001).

**Relationship between L.V.H. and L.V. dysfunction in patients of systemic hypertension**

Bonaduce et al (1989) studied 13 normotensive subjects (Group I) 12 hypertensive patients without LVH (Group II) 28 with LVH (Group III). In group III patients diastolic filling parameters were impaired while in group II they were intermediate between group I and III.

Systolic dysfunction, however does not correlate well with LVH in hypertensive cases. Toshima et al reported a normal echocardiographic ejection fraction in 11 patients with concentric LVH caused by hypertension.

**L.V. dysfunction in hypertensive patients with CHF**

Heart failure is an abnormality of cardiac function responsible for the inability of the heart to pump blood at a rate commensurate
with the requirements of the metabolizing tissue and/or can do only
from an abnormally elevated filling pressure.

Abnormalities during systole and/or diastole may be present in heart failure and/or diastole may be present in heart failure (Vasan RS et al, 1996).

In so called systolic heart failure i.e. classical heart failure, an impaired inotropic state causes weakened systolic contraction which leads, ultimately to a reduction in stroke volume, inadequate ventricular diastolic pressure. Traditionally, CHF consequent to hypertension and was considered to have only systolic dysfunction. In diastolic heart failure the principal abnormality involves impaired relaxation of the ventricle and or normal diastolic volume. Failure of relaxation can be caused by a stiffened thickened ventricle as in hypertension. So hypertension is one of the commonest cause of diastolic dysfunction and heart failure.

**Assessment of left ventricular hypertrophy or enlargement:**

In X-Ray cardiothoracic ratio is normally below 50% in PA view but in AP films normal value can be assessed as 55%.

In infants the normal value can be 55%. As the left ventricle enlarges, there is usually an increase in cardiothoracic ratio and
curvature of lower left heart border takes on large radius ventricles, ventricle enlarges towards lateral wall of thorax in a downward direction displacing apex laterally and inferiorly.

In lateral view we calculate distance from posterior aspect of inferior vena cava to the posterior border of heart horizontally at the level 2 cm above intersection of the diaphragm and the inferior vena cava, this is known as **Hofman sign**. A distance of greater than 1.8 cm indicate left ventricular enlargement. Such measurements can be helpful but great reliance cannot be placed on them as individual anatomic variation can cause discrepancies (David Sutton).

In ECG for LVH detection Ramhilt and Ester point score system was used. Criteria are as follows:

<table>
<thead>
<tr>
<th></th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. R or S Limb lead</td>
<td>( \geq 20\text{mm} )</td>
</tr>
<tr>
<td></td>
<td>( \geq 30\text{mm} )</td>
</tr>
<tr>
<td>2. Intrinsicsoid deflection in ( V_5 ) or ( V_6 )</td>
<td>0.05 sec or more</td>
</tr>
<tr>
<td>3. Left axis deviation</td>
<td>( 30^\circ ) or more</td>
</tr>
<tr>
<td>4. QRS interval 0.09 sec or more</td>
<td></td>
</tr>
<tr>
<td>5. Left atrial abnormality/enlargement</td>
<td></td>
</tr>
</tbody>
</table>
6. St-T changes - without digitalis 3  
- with digitalis 1

LVH is present if the total score is more than 5 points and probably present if score is 4 points.

In echocardiography LV mass was calculated by using formula given by Devereux and Reixhek 1977:

$$LVH = 1.04 \left[ \frac{(IVST+LVID+PNT)^3}{LVID^3} \right] - 13.6$$

LV mass index is LV mass per square meter body surface area. LV mass could also be calculated from 2D echo tracing of parasternal short axis showing LV at the papillary muscle level, showing good endocardial definition by area length method (Schiller N et al 1989).

The upper limits of IVST, PWT and LV mass index (gm/m²) are shown to be 1.1cm, 1.1cm and 122gm/m² BSA for Indian women (Trivedi et al 1991). In a study Ghanem Wisam MA et al 2000 LVH was defined by echocardiography as LBV mass index >134 gm/m² in men and >100 gm/m² in women.