Review
Of
Literature
History of hypertension in the modern era dates back to Stephen Hales in 1873 who first ventured to investigate blood pressure by the use of saline manometer. Today, though blood pressure measurement is a simple bedside procedure, the criteria for the diagnosis of hypertension has been arrived at after much controversy.

Life insurance actuaries (1959), identified mortality increments in policy holders with what many clinicians considered trivial elevation of blood pressure. Framingham heart study (1968), showed discrete risks for isolated systolic and diastolic elevation. 'HANES' survey of United States (1977) selected a blood pressure of 160/95 mm Hg or more to define hypertension. Perloff & Sokolow (1978) documented the value of 24 hours blood pressure surveillance in interpreting the need for medication, the response to medication and the inherent variability of hypertension measured under a variety of circumstances.

Fowler et al (1980), made the criterion to 95 mm Hg in patients over age 30 years & applied thus standard to recumbent or standing measurements.

Current standards for defining & diagnosing hypertension rests on the Blood Pressure levels which confers an increased risk of developing a morbid cardiovascular event and/or will clearly benefit for medical therapy. In adults, according to the VIth report of Joint
treatment of high blood pressure the following values are now considered:

### Diastolic blood pressure:

<table>
<thead>
<tr>
<th>Range</th>
<th>Classification</th>
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</thead>
<tbody>
<tr>
<td>below 80 mm Hg</td>
<td>Optimal</td>
</tr>
<tr>
<td>less than 85 mm Hg</td>
<td>Normal</td>
</tr>
<tr>
<td>85 mm Hg – 89 mm Hg</td>
<td>High normal</td>
</tr>
<tr>
<td>90 mm Hg – 99 mm Hg</td>
<td>Hypertension stage I</td>
</tr>
<tr>
<td>100 mm Hg – 109 mm Hg</td>
<td>Hypertension stage II</td>
</tr>
<tr>
<td>110 mm Hg or above</td>
<td>Hypertension stage III</td>
</tr>
</tbody>
</table>

### Systolic blood pressure:

<table>
<thead>
<tr>
<th>Range</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>below 120 mm Hg</td>
<td>Optimal</td>
</tr>
<tr>
<td>less than 130 mm Hg</td>
<td>Normal</td>
</tr>
<tr>
<td>130 – 139 mm Hg</td>
<td>High normal</td>
</tr>
<tr>
<td>140 mm Hg – 159 mm Hg</td>
<td>Hypertension stage I</td>
</tr>
<tr>
<td>160 mm Hg – 179 mm Hg</td>
<td>Hypertension stage II</td>
</tr>
<tr>
<td>greater than 180 mm Hg</td>
<td>Hypertension stage III</td>
</tr>
</tbody>
</table>

These levels should be persistent and blood pressure should be measured on two separate occasions under the proposed near ideal conditions before labeling a patient as hypertensive. Higher values of systolic or diastolic pressure will be taken for staging.
The myocardial function is divided into two cardiac cycles:

Diastole:– A period of relaxation followed by Systole – a period of contraction.

*Diastole:*– This is further divided into following phases:–
- Isovolumic relaxation
- Rapid inflow
- Diastasis
- Atrial systole

*Systole:*– Similarly this has been divided into:–
- Isovolumic contraction
- Period of ejection
- Protodiastole

The extent of shortening of mammalian heart muscle and therefore the stroke volume of the intact ventricle are in final analysis determined by three influences (Guyton A.C. et al).

1. The length of the muscle at the onset of contraction, i.e. the preload.
2. The inotropic state of the muscle.
3. The tension that the muscle is called upon to develop during contraction i.e. the afterload.

**Ventricular Afterload**

The afterload on the intact heart may be defined as the tension or stress developed in the wall of the ventricle during ejection. Therefore, the afterload on the ventricular muscle
well as on the volume and thickness of the ventricular cavity, since Laplace's Law indicates that:

\[
\text{Myocardial Fibre Tn} = \frac{\text{Vent. Rad.} \times \text{Intra cavity Vent. Press.}}{\text{Wall thickness}}
\]

Thus at the same level of aortic pressure, the afterload faced by a dilated left ventricle is higher than that encountered by a ventricle of normal size. (Harrison's principles of internal medicine, 15th ed. Vol. I; pp.1315).

**Diastolic properties of the Left ventricle**

Left ventricle pressure and volume during diastole, reflect the interaction of ventricular elastic, viscous and inertial properties, and the completeness of myocardial relaxation (Grossman et al.). In their study they summarized the factors determining left ventricular diastolic properties as follows:-

**Principal factor determining left ventricular diastolic properties**

(A) *Properties intrinsic to the ventricular chamber*

1. Completeness of ventricular relaxation.
2. Passive elastic properties of ventricular chamber (stiffness or compliance).
   (a) Thickness of ventricular walls.
   (b) Composition of ventricular wall (muscle, scar amyloid, calcium, iron).
3. Viscous properties.
5. Influence of contractile state.

6. Influence of other factors (e.g. myocardial ischemia, temperature, osmolality).

**(B) properties extrinsic to the ventricle**

1. Pericardial properties.

2. Atrial contraction (presence, strength).

3. Increased diastolic inflow (shunts, high output states, valvular insufficiency).


5. Overload of the right ventricle.

   Recent studies indicate that for cardiac muscle, relaxation is not a passive, but a complex energy dependent process (Langer et al.).

   Left ventricular relaxation may be impaired in patients with congestive heart failure, without systolic dysfunction (Litwin JE.et.al 1993).

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

   Earliest function cardiac changes in hypertension are in left ventricular diastolic function, with prolongation and in coordination of isovolumic relaxation. (Braunwald 2001 page 949, Dibello.V.et al 1999).

   Topol et al (1985) found elderly patients with hypertension having abnormal diastolic function, but excessive LV emptying.
Mechanism of L.V.H in systemic hypertension

Pathogenesis of L.V.H involve a number of variables other than the pressure load, one of which is haemodynamic volume load. Devereux and colleagues (1992) found a close correlation between left ventricular stroke volume and left ventricular mass, with diastolic then with systolic blood pressure. Other determinants are obesity (Gottdiener et al 1997), levels of sympathetic nervous system and renin angiotensin activity and whole blood viscosity (Braunwald et al 1994). The correlation is much closer between L.V.H. and pressure readings taken during the stress of work by ambulatory monitoring, than between L.V.H. and casual pressure reading (Gottdiener et al 1994). By echocardiography, left ventricular mass is shown to progressively increase with increase in blood pressure (Kahan T. et al 1998). Left ventricular mass is greater in those whose pressure does not fall during sleep because of a more persistent pressure load (Deveurex et al 1992).

Different pattern of hypertrophy may evolve often starting with asymmetrical left ventricular remodeling from isolated septal thickness, which has been noted in 22% of untreated hypertensives with normal total left ventricular mass (Verdecchia et al. 1994).

Treatment with all antihypertensive drugs except those that further activate sympathetic nervous activity, e.g., direct vasodilators such as hydralazine when used alone has been shown to cause L.V.H. regression (Ofili EO et al 1998) with regression, left ventricular function usually improves and cardiovascular morbidity decreases (Verdecchia et al 1998).

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

Dreslinski et al (1981) studied 10 normal subjects, 11 hypertensive patients without echocardiographic evidence of L.V.H. and 10 patients of hypertension with L.V.H. They observed that progressive decrease in diastolic function in the 3 groups respectively.

Systolic dysfunction, however does not correlate well with L.V.H. in hypertensive cases. Toshima et al reported a normal echocardiographic ejection fraction in 11 patients with concentric L.V.H caused by hypertension.
LV 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 tissues and/or can do only from an abnormally elevated filling pressure.

Abnormalities during systole and/or diastole may be present in heart failure (Vasan RS et al 1996). Blacks are more prone, to progression to heart failure and death than whites. In so called systolic heart failure i.e. classic heart failure, an impaired inotropic state causes weakened systolic contraction which leads, ultimately to a reduction in stroke volume, inadequate ventricular emptying, cardiac dilatation, and often elevation of 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 a normal diastolic volume. Failure of relaxation can be caused by a stiffened thickened ventricle as in hypertension. So hypertension is common cause of diastolic dysfunction and diastolic heart failure (Dougherty AL et al 1984). Recognition of this fact has a lot of clinical relevance in terms of treatment.

Dougherty et al (1984) studied 184 patients with CHF among which 64% had reduced EF and 36% turned out to have normal EF. On further analysis 65% cases of those normal EF turned out to be cases of hypertension. So they concluded that among patients with clinical heart failure
pulmonary congestive symptoms are associated with abnormal LV compliance. Also they did not find any correlation between severity of symptoms and EF. Another observation made by them was the finding, that internal ventricular dimensions in 13 out of 16 patients with a normal EF were normal by quantitative echocardiography, which is more direct measure of chamber size. Thus in their study patients of hypertension had isolated diastolic, isolated systolic and combined dysfunction as well. Most episodes of C.H.F. in hypertensive patients are associated with dilated cardiomyopathy and a reduced ejection fraction, however, about 40% episodes of C.H.F. are associated with preserved L.V. systolic function, but with diastolic dysfunction induced by L.V.H., fibrosis and ischemia and increased afterload (Bonow & Udelson 1992).

**Echocardiographic parameters of LV function**

Safar et al (1990), found LVH in hypertensive patients by the increase in IVS and LVPW, which was statistically significant (p < 0.001 and p < 0.01 respectively).

LVIDd and LVIDs were not different from normal diameters in subjects with sustained hypertension except for LVIDs which was significantly increased. The relative reduction in diameter (LVIDs – LVIDs/LVIDd) was equal in normotensive subjects and patients with borderline hypertension (0.33 ± 0.08 VS 0.30 ± 0.06). It showed some decrease in patients with sustained hypertension ( 0.26 ± 0.09) but the difference was not statistically significant.
Savage et al (1989) found increased IVSd in 30%, increased LVPW (61%) and increased LV mass in 51% of hypertensives, and this increase was statistically significant. LVEF was increased in 15% and EF slope decreased in 6%. Mean values of LVIDd and LVIDs, left atrial and aortic root dimension and LVEF for the hypertensive subjects were not significantly different from values in normal subjects.

There was no significant correlation (positive or negative between ventricular septal or LV free wall thickness and EF. Although only a small number of hypertensive subjects had a mitral valve EF slope below the 95% prediction interval, the mean EF slope of hypertensive subjects was significantly lower than that of normal subjects (p < 0.001).

Nine hypertensive subjects had ventricular septal thickening that was disproportionate to the LV free wall thickness (i.e. septal free wall ratio ≥ 1.3). Their measured septal thickness ranged from 15 - 27 mm with septal free wall ratios from 1.3 – 1.9. None of the nine had systolic anterior motion of the anterior leaflet of the mitral valve. However, one subject who had concentric LV wall thickening did have systolic anterior motion of the anterior mitral leaflet.

Hannath et al (1992), observed that mean LVIDd and LVIDs in the patients of chronic overload was 48.5 ± 7.9 and 26.9 ± 6.1 mm respectively. LVIDs was significantly reduced in comparison with control group (p < 0.05). The sum of IVSd and LVPWd ( 29.1 ± 48 mm ) was greater than that in normal subjects (p < 0.001).
Assessment of LV diastolic dysfunction by echocardiography

Good pulse wave spectral Doppler transmitral flow pattern recorded at the tips of the mitral leaflets in apical 4 Chamber view show following parameters as suggestive of LV diastolic dysfunction (Oh J.K. et al 1994).

- Low E velocity and high A velocity.
- Reversed E/A ratio (<1).
- Prolonged ‘E’ deceleration time (DT) (>240 m sec).
- Prolonged isovolumetric relaxation time (IVRT) from aortic closure to MV opening (<110 m sec).

Normal values of E = 0.85 ± 16 m sec

\[ A = 0.56 ± 0.13 \text{ m sec} \]

E/A ratio = 1.6

Assessment of left ventricular hypertrophy or enlargement

In X-ray cardiothoracic ratio is normally well below 50% in PA view but in AP films normal value can be accessed 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 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 venacava to the posterior border of heart horizontally at the level 2 cm above intersection of the diaphragm and the inferior venacava. This is known as
Holman 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 anatomical variation can cause discrepancies (David Sutton 2003).

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

<table>
<thead>
<tr>
<th>Points</th>
<th>1. R or S in limb lead</th>
<th>≥ 20 mm</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SV₁ or SV₂</td>
<td>≥ 30 mm</td>
<td></td>
</tr>
<tr>
<td></td>
<td>RV₅ or RV₆</td>
<td>≥ 30 mm</td>
<td></td>
</tr>
<tr>
<td>2. Intrinsics deflection in V₅ or V₆</td>
<td>0.05 sec or more</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>3. Left axis deviation</td>
<td>[30° or more]</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>4. QRS interval 0.09 sec or more</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Left atrial abnormality/enlargement</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. ST – T changes</td>
<td>without digitalis</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>with digitalis</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

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 Reichek 1977.

\[
LVM = 1.04 \left( \left( IVST + LVID + PWT \right)^3 - LVID^3 \right) - 13.6
\]

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
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 1.1 cm, 1.1 cm and 122 gm/m² BSA for Indian men and 0.9 cm, 0.9 cm and 110 gm/m² BSA for Indian women (Trivedi et al 1991). In a study by Ghanem Wisam MA et al 2000 LVH was defined by echocardiography as LV mass index > 134 gm/m² in men and >110 gm/m² in women.