Updated Clinical Importance of LV Diastolic Dysfunction and role of modern Echocardiography

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Abstract

In this review, we discussed about updated concept about diastolic dysfunction of left ventricle and mechanism behind the diastolic heart failure with normal Ejection fraction of LV using modern and advanced non-invasive technique of 2D Echocardiography. In actual way, the invasive data obtained from cardiac catheterization is considered the gold-standard to study LV filling pressures and LV pressure volume curves. 2D Echocardiography with advanced technology is useful, affordable and more convenient to the patients those who have Hypertension, diabetes, thyroid disorders, infiltrative, restrictive or hypertrophic cardiomyopathy, valvular disorders, pericarditis, severe pulmonary hypertension and pre and post operative procedures. We can differentiate diastolic heart failure from systolic heart failure based on parameters such as increased myocardial stiffness, delayed isovolumic relaxation, increased LV filling pressure and decreased LV Compliance but preserved LV Ejection fraction or systolic function when patients present with dyspnoea on exertion or worsen heart failure.

Keywords-2D Echocardiography, diastolic functions, heart failure, advanced diastology.

1. Introduction

Assessment of left ventricular diastolic function and filling pressure measurements can distinguish dyspnoea due to impaired diastolic dysfunction with preserved systolic function from other diseases such as pulmonary disorders that cause dyspnoea. They also can be used to assess prognosis and plan proper management. About 50 % patients with new diagnosis of heart failure have normal or fair LVEF are proven as diastolic heart failure. LV filling pressures including mean pulmonary wedge pressure or mean LA pressure, LVEDP (LV End diastolic pressure) and pre-A LV diastolic pressure are best measured by invasive cardiac catheterization procedure to diagnose abnormal diastole. Echocardiography is gaining popularity as a good tool to study the same measurement at a lower cost and are more acceptable to the patients.

We can assess the diastolic dysfunction by 2D Echo, color M –Mode transmitral flow propagation velocity of LV, Doppler mode on Mitral valve inflow with pulse wave DT (Deceleration Time) and peak E/A Ratio (E- Early Filling and A- Atrial filling), by TDI e’ and a’ with E/e’ ratio, pulmonary flow velocity and other parameter to assess diastolic dysfunction like, twisting or untwisting of LV or time to untwist rotation to mitral valve opening by speckle tracking mode, LA size and volume and rate of fall of mitral/aortic valve regurgitation velocity (Negative dp/dt).

2. Role of 2D Echocardiography to assess diastolic function

During diastole, initially LV pressure fall and passive rapid filling pattern depend on molecular based cyclic interaction of myofilaments. It is related to myocardial relaxation and passive LV properties which is modulated by myocardial tone. It is determined by load inactivation and beyond uniformity. The double helicoidal fiber orientation of LV leads to twisting or torsion during systole and untwisting or torsional recoil during diastole. Torsional recoiling depend on relaxation of circular and longitudinal fibres leads to sucking of and pulling
LV resulting in rapid LV filling passively in early diastole. In late diastole, active filling depend on Atrial contraction.

**Pathologic left ventricular hypertrophy** has myocardial stiffness which leads to impaired LV relaxation and reduce early diastolic filling and raised LV filling pressure. We can measure LV wall thickness and mass by 2D and M mode by echocardiography. We can also assess the sucking and pulling power of LV or rapid early filling by torsion recoil time and velocity with new tool of speckle tracking or strain imaging pattern. LA volume has role to assess diastolic dysfunction which can measured accurately by 2D Echo (4 chamber and 2 chamber views).

Study in patients with Atrial fibrillation and valvular diseases have shown that LA volume index > 34 ml/m2 is an independent predictor of heart failure, AF with stroke patients (Arq Bras Cardiol. 2013 ). The left atrium modulate ventricular filling through its reservoir, conduit and pump function. LA play as a role conduit between aortic valve opening and LA contraction. Reduction of LA conduit leads to decreased LV relaxation and fall early AV diastolic gradient while impaired reservoir-pump complex leads to reduced LA contractility results as raised LV End Diastolic Pressure and reduced LV filling. Some patients who presenting dyspnea and chest discomfort with diastolic dysfunction usually revealed high pulmonary artery pressure. Raised PA pressure without pulmonary disease may increased LV filling pressure and impaired LV relaxation leads diastolic heart failure. We can calculate PA systolic pressure (PASP) by TR (Tricuspid Regurgitation) peak velocity and PA diastolic pressure (PADP) by PR (Pulmonary Regurgitation) end diastolic velocity with addition of RA pressure. We can correlate PA diastolic pressure to LA pressure, as PADP is higher (> 5 mmHg) than mean wedge pressure.

**Pulse wave Doppler** is being used through apical 4 chamber view to assess mitral valve inflow velocities to assess LV filling pressure. During diastole we record a velocity profile by placement of 1 mm to 3mm sample volume between the mitral valve leaflet tips. Usually we measure parameters like peak E and A velocities, E/A ratio, Deceleration Time( DT) and IVRT using PW doppler. Mitral inflow pattern can differentiate Normal, Impaired LV relaxation, Pseudo normal and Restrictive filling. Patients (see at figure-1) with ischemic heart disease and hypertrophic cardiomyopathy with LVEF > 55 %, mitral velocities correlate poorly. However in patients with dilated cardiomyopathy filling pattern correlate better with functional class and prognosis than LVEF. A pseudonormal class is represented with mild to moderate increased LA pressure in the setting of delayed LV relaxation due to valsalva maneuver decrease pre-load during the strain phase. Forceful expiration against closed nose and mouth lead to decrease pre-load resulting reduced E velocity and normal or increased A velocity, hence E/A ratio decreased and its specificity denote > 50 % patients having increased LV filling pressure.
Assessment of LV diastolic function also possible by study of the pulmonary venous flow, performed by PW Doppler in 4 chamber apical view of PV flow with recording of 2 to 3 mm sample volume placed more than 5 mm into pulmonary venous flow. This spectral waveform include peak systolic (S) and diastolic (D), the S/D ratio, systolic filling fraction, peak Ar (Atrial reversal) velocity and Time between Ar velocity and MV A wave duration (Ar-A). Decreased S/D ratio, increased Ar and Ar-A suggest as increased LV End Diastolic Pressure (LVEDP) and decreased systolic filling fraction related to decreased LA function leads to diastolic heart failure with normal LVEF. The majority of pulmonary venous flow into the left atrium occurs during ventricular systole (S wave) as less in diastole (D wave); as a result, the S:D ratio is typically >1. With early impaired LV relaxation, the S wave velocity is accentuated such that the S:D ratio rises >1 (see at figure-2). However, with progressive LVDD and increasing LA pressure, there is the S:D ratio is <1, and the D wave deceleration time falls. PV Doppler is limited by the ability to accurately interrogate the pulmonary veins, and the Doppler profiles are also strongly impacted by the presence of significant mitral valve disease.

Color M Mode have also value to assess LV Diastolic dysfunction by obtain Propagation velocity (Vp). We can measure Vp on M mode with color flow from MV to apex is performed in the apical 4 chamber view and measured as the slope of the first aliasing velocity during early diastole or the slope of the transition for no color to color. On the base of recent studies Vp value < 40 cm/sec is related to increased LV filling pressure denote as LVDD on semiquantitative scale. On color M mode propagation velocity assessment with mitral E velocity to predict LV filling pressure and studies have shown E/Vp ratio is directly proportional to LA pressure, E/Vp >2.5 predict PCWP >15 mmhg but some limitations are there with normal ejection patients misleading Vp value may be normal with abnormal filling pattern.

Pulse Wave Doppler Tissue Imaging is performed to acquire mitral annular velocities. In the apical views (usually 4 chamber), sample volume should be placed at or 1 cm within the septal and lateral sites of the mitral annular leaflets or their average to assess LV Diastolic function globally. Primary measurements include the systolic and early (e') and late (a') diastolic velocities. e’ can be used to correct for the effect of LV relaxation on mitral E velocity, and the
E/e' ratio can be applied for the prediction of LVEDP and Pulmonary capillary wedge pressure (PCWP). Using the septal E/e' ratio, a ratio is 8 is usually associated with normal LV filling pressures, whereas a ratio is or more than 15 is associated with increased filling pressures. Be aware, when the value is between 8 and 15, other echocardiographic indices should be used. The E/e' ratio is not accurate as an index of filling pressures in normal subjects or in patients with heavy annular calcification, mitral valve disease, and constrictive pericarditis. Recent studies have shown in patients with normal EFs, lateral tissue Doppler signals (E/e’ and e’/a’) have the best correlations with LV filling pressures and invasive indices of LV stiffness. TE-e’ is particularly useful in situations in which the peak e’ velocity has its limitations, and the average of 4 annular sites is more accurate than a single site measurement for this time interval. It is useful in patients with normal LVEF or those with mitral valve disease and when the E/e’ ratio is 8 to 15. Especially on data based, an IVRT/TE-e’ ratio < 2 has correlated with accuracy in LVDD with raised LVEDP and PCWP.

3. Advanced Diastology

3.1 Diastolic Stress echocardiography

Diastolic Stress Test is useful to assess LV filling pressure with exercise as well, similar to the use of efforts to evaluate patients with valvular diseases or associated with atrial fibrillation and coronary artery disease. Asymptomatic patients with mild diastolic dysfunction may proceed to heart failure during stress. The E/e’ ratio, E/Vp, PA pressure has been applied for stress induced diastolic heart failure. In patients with impaired myocardial relaxation, the increase in e’ with exercise is much less than that of mitral E velocity, such that the E/e’ ratio increases. In that regard, E/e’ was shown to relate significantly to LV filling pressures during exercise, when Doppler echocardiography was acquired simultaneously with cardiac catheterization decreases slightly in normal individuals with exercise, but shortens <50 ms in patients with a marked elevation of filling pressures. In cardiac patients, mitral E velocity increases with exertion and stays increased for a few minutes after the termination of exercise, whereas e’ velocity remains reduced at baseline, exercise, and recovery. Therefore, E and e= velocities can be recorded after exercise, after 2D images have been obtained for wall motion analysis. Furthermore, the delayed recording of Doppler velocities avoids the merging of E and A velocities that occurs at faster heart rates. Exercise is usually performed using a supine bicycle protocol, and TR signals by CW Doppler are recorded as well to allow for the estimation of PA systolic pressure at rest and during exercise and recovery. Diastolic stress echocardiography (DSE) has been also performed with dobutamine infusion in obese, arthritic and immobile patient and restrictive filling with dobutamine was shown to provide prognostic information. (See figure-3)

**Figure-3** (A. Dobutamine-Diastolic-stress echocardiograph, B. Bike-stress-diastolic-echocardiography)
Using a supine bike protocol can provide useful data on LV systolic reserve (changes in stroke volume, EF, and global longitudinal strain) and diastolic reserve (changes in e’ velocity, E/e’ ratio, and PA pressures). It is often difficult to distinguish the effects of the valve lesion itself, systolic dysfunction, and diastolic dysfunction on abnormal resting or exercise echocardiographic and hemodynamic recordings. Notwithstanding, recent studies support the clinical promise of using exercise PA pressure estimation for the management of patients with asymptomatic severe AS. Left atrial enlargement reflects chronic exposure to increased LV filling pressure, and it may serve as an integrative, easily obtainable measure of diastolic dysfunction. Studies examining atrial function in patients with symptomatic diastolic HF recently confirmed the presence of reduced LA systolic function, particularly under stress conditions.

3.2 Assessment of twist and peak untwisting rates

The LV relaxation phase is accompanied by active movement of the mitral annulus away from the apex. Myocardial relaxation continues during early diastole to reach the minimal LV diastolic pressure, which helps with “sucking” or “pulling” the blood actively into the LV depend on geometric fibre orientation of myocardium. LV apical portion twist normally counterclockwise and basal part twist clockwise during systole and finally untwist during diastole. Abnormal myocardial strain and untwist rate, rotation and velocity are related to inadequate LV filling pressure can be determined by 2D speckle tracking techniques from short axis view and apical views (see figure-4). In both HfEF and HFrEF patients with invasively measured elevated LV filling pressures PCWP >12 mmHg, LV longitudinal and radial strain were reduced compared with controls, whereas circumferential strain and twist were similar between HfEF and controls, but lower in HFrEF using a cutoff value of global LV longitudinal strain at -16 %, HfEF patients could be distinguished from normal controls at a sensitivity of 90 % and specificity of 95% with an area under the curve of 0.98. In another study myocardial deformation measurements at rest and on submaximal exercise were compared between HfEF patients and controls.

Further more HfEF patients had higher end diastolic pressures, reduced and delayed untwisting and reduced LV suction at rest and on exercise . Recent studies indicate that LV twist may remain preserved in patients with diastolic dysfunction in presence of normal ejection fraction . Wang et all(Circulation 2007) studied 67 patients (34 with LV ejection fraction <50% and 33 with LV ejection fraction >50%) undergoing simultaneous right heart catheterization and echocardiographic imaging and compared the LV twist mechanics with 20 healthy subjects as a control group. The onset of untwisting occurred just before aortic valve closure in control subjects and was significantly delayed after aortic valve closure in patients with systolic and diastolic heart failure . The LV twisting and untwisting rates were reduced in patients with LV systolic dysfunction and depressed ejection fraction, but not in those with diastolic dysfunction and normal ejection fraction. In another study of 58 patients with hypertension, twist was not different among groups of patients with or without LV hypertrophy, although early diastolic LV untwisting and untwisting rates were significantly delayed and reduced in parallel to the severity of LV hypertrophy, as assessed by LV mass index. Thus, LV twist is either preserved or augmented in patients with diastolic dysfunction and normal systolic performance. However, the onset of LV untwisting and the magnitude of peak untwisting velocities either remain normal or are reduced and significantly delayed . More studies are required to explore the variability of this observation.
Figure-4 Global and Regional Daistolic Functions are assessed by 2D Speckle tracking, myocardial strain and strain rate, rotation time, velocity of untwisting during diastole.

4. Classified diastology based on age & grade and clinical relevance

According to Echo data from various studies, we can justify the normal diastolic functions in young or old age group (>60 yrs) and impaired diastolic dysfunction (Class-I DD), pseudonormal (Class-II DD), restrictive filling pattern (Class-III DD). See Table-1 (data) and Figure-5 (echo recorded pictures are being used to assess diastolic function).

Table-1: Classified diastology based on age & grade of diastolic dysfunction and clinical relevance

<table>
<thead>
<tr>
<th>Echo Parameters</th>
<th>Young Age (Normal)</th>
<th>Old Age (Normal)</th>
<th>Class-I DD (Impaired DD)</th>
<th>Class-II DD (Pseudonormal)</th>
<th>Class-III DD (Restrictive)</th>
</tr>
</thead>
<tbody>
<tr>
<td>E/A Ratio</td>
<td>0.8 to 2.1</td>
<td>0.6 to 1.3</td>
<td>0.3 to 0.8</td>
<td>0.8 to 1.5</td>
<td>2.1 to 2.9</td>
</tr>
<tr>
<td>DT (ms)</td>
<td>130-220</td>
<td>140-260</td>
<td>&gt;200</td>
<td>160-200</td>
<td>&lt;160</td>
</tr>
<tr>
<td>IVRT (ms)</td>
<td>40-90</td>
<td>70-100</td>
<td>&gt;100</td>
<td>60-100</td>
<td>&lt;60</td>
</tr>
<tr>
<td>E/e’</td>
<td>4 to 8</td>
<td>5 to 9</td>
<td>6 to 10</td>
<td>9 to 14</td>
<td>&gt;15</td>
</tr>
<tr>
<td>Septal E/e’</td>
<td>5 to 9</td>
<td>6 to 10</td>
<td>7 to 11</td>
<td>10 to 16</td>
<td>&gt;16</td>
</tr>
<tr>
<td>Lateral E/e’</td>
<td>3 to 7</td>
<td>4 to 8</td>
<td>5 to 9</td>
<td>8 to 12</td>
<td>&gt;14</td>
</tr>
<tr>
<td>PV S/D ratio</td>
<td>0.4 to 1.6</td>
<td>0.5 to 2.3</td>
<td>0.4 to 1.0</td>
<td>0.4 to 0.8</td>
<td>0.3 to 0.6</td>
</tr>
<tr>
<td>MV Vp</td>
<td>45 to 60</td>
<td>36 to 44</td>
<td>32 to 43</td>
<td>27 to 38</td>
<td>18 to 28</td>
</tr>
<tr>
<td>E/Vp</td>
<td>1 to 1.8</td>
<td>1 to 1.4</td>
<td>1.1 to 1.5</td>
<td>2.5 to 3</td>
<td>3 to 4.5</td>
</tr>
<tr>
<td>Ar (cm/s)</td>
<td>5 to 32</td>
<td>11 to 36</td>
<td>23-37</td>
<td>35-42</td>
<td>40-51</td>
</tr>
<tr>
<td>Ar-A (ms)</td>
<td>&lt;0</td>
<td>&lt;0</td>
<td>&lt;0</td>
<td>30-40</td>
<td>35-45</td>
</tr>
<tr>
<td>LA Vol (ml/m2)</td>
<td>21-27</td>
<td>29-33</td>
<td>30-33</td>
<td>34-40</td>
<td>40-50</td>
</tr>
<tr>
<td>rot-R *</td>
<td>0.6-0.8</td>
<td>0.4-0.5</td>
<td>0.34-0.48</td>
<td>0.2-0.3</td>
<td>-0.1-0.2</td>
</tr>
</tbody>
</table>

* further datas and studies are required.
Grade-1 (impaired diastolic dysfunction) has the best prognosis; patient is asymptomatic or has mild symptoms, may not require diuretics, can benefit from angiotensin converting enzyme inhibitors or angiotensin receptor blockers, beta blockers. Grade-2 (pseudonormal) moderate, diastolic dysfunction, patient has mild to moderate symptoms, likely to be in NYHA class 2, would require diuretics with angiotensin converting enzyme inhibitors or angiotensin receptor blockers, beta blockers. Grade-3 A (restrictive pattern) being severe but reversible has an adverse prognosis; it is characterized by symptomatic heart failure, NYHA class being 3–4, requiring increased diuretics with angiotensin converting enzyme inhibitors or angiotensin receptor blockers, beta blockers. Grade-3 B (restrictive) being the most severe and irreversible has the worst prognosis; it is suggestive of end-stage heart failure and diuretics are ineffective in that situation. Last one is frequently seen in end-stage restrictive cardiomyopathies such as amyloid cardiomyopathy.

The ACC/AHA guidelines 2013 give three class I recommendations to medically treat chronic diastolic congestive heart failure. The first is to control the heart rate in patients with atrial fibrillation in order to improve diastolic filling. Tachycardia shortens diastolic filling time and so keeping heart rates < 100 beats per minute and preferably between 60-80 beats per minute will improve cardiac output when significant diastolic heart failure is present. Rate control can be achieved using beta-blockers, non-dihydropyridine calcium channel blockers, or digoxin. The second recommendation is to control systolic and diastolic blood pressure using the standard treatment for hypertension. The third is to use diuretics to control pulmonary congestion and peripheral edema.

5. Our Study and few others

We have done an observational study with 100 healthy and 100 unhealthy cases Age 52 to 68 yrs assessed by 2D Echocardiography with pulse and tissue Doppler and Color M mode through mitral valve inflow. Our study revealed, that in healthy group had LVEF =55 to 70% and 82 % of them showed E/A Ratio between 1 to 2 and 78 % cases showed E/e’ ratio between 7 to 9 and 75 % cases ; DT between 160 to 190 ms and Color M Mode showed 89 % Propagation velocity 40 to 50 cm/sec. Other site among unhealthy group male and female as per data LVEF =54 to 68 %, according to PW Doppler showed 64% cases have E/A <1 and 30 % cases have E/A = 1 to 1.8., rest 6 % cases have E/A= 2 to 2.4 . we found 10 cases as pulmonary edema Tissue Doppler revealed E/e’=20 to 29 and by color M mode Propagation Velocity 20 to 25 cm/sec. 20 cases as NYHA Class III-IV with pedal edema, raised JVP, Tissue Doppler revealed E/e’=18 to 23 and by color M mode Propagation Velocity 25 to 30 cm/sec. 40 cases as NYHA Class II-III with pedal
edema, Normal JVP, Tissue Doppler revealed E/e’=12 to 18 and by color M mode Propagation Velocity 30 to 40 cm/sec. 30 cases as NYHA Class I-II with no pedal edema, normal JVP, Tissue Doppler revealed E/e’=4 to 10 and by color M Mode Propagation Velocity 40 to 45 cm/sec.

Further we found by cath invasive data have high LV filling pressure with normal pulse Doppler diastolic data or parameters, subsequently we revealed by echo advance mode speckle tracking – smaller peak of apical early diastole untwist (rotR). Again we compared in some patients they presented abnormal, pseudonormal and restrictive filling pattern. Those patients have more value of E/e’ with normal E/A (1 to 1.5) or >2 ratio revealed by speckle tracking data as 0.1 to 0.25 rotR as compare to Normal (0.45 to 0.6 rotR). Also we confirmed with lab data of BNP or pro BNP Level which were high value in patients those found low peak rotR and shorter time from the rotR (peak of apical early diastolic untwist) to MVO (Mitral valve opening), see at table-2.

**Table-2 (Clinical and Echo parameters before and after treatments of LV diastolic dysfunction)**

<table>
<thead>
<tr>
<th></th>
<th>Before Treatment</th>
<th>After Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>NYHA Class</td>
<td>III-IV</td>
<td>I-II</td>
</tr>
<tr>
<td>Pedal Edema</td>
<td>++</td>
<td>-</td>
</tr>
<tr>
<td>BNP (pg/ml)</td>
<td>300+/-34</td>
<td>67+/-9</td>
</tr>
<tr>
<td>E/A</td>
<td>0.6 to 2.4</td>
<td>1 to 1.5</td>
</tr>
<tr>
<td>E/e’</td>
<td>4 to 23</td>
<td>8 to 12</td>
</tr>
<tr>
<td>rot-R</td>
<td>0.1 to 0.25</td>
<td>0.3 to 0.6</td>
</tr>
<tr>
<td>Vp (cm/sec)</td>
<td>20 to 40</td>
<td>45 to 60</td>
</tr>
</tbody>
</table>

We treated some patients with beta blockers metoprolol or carvedilol or diltiazam to reduce heart rate and improve LV filling and LV relaxation. We treated some patients with Digoxin, Diuretics and ACE inhibitors and Nitrates with CoQ to cover severe breathlessness, pulmonary edema, pedal edema and lower the BNP Values (see at table-2). We transferred few patients for coronary artery bypass graft surgery and valvular replacement or repair those were revealed as diastolic dysfunction. The Doppler and tissue Doppler parameters of diastolic dysfunction normalised after treatment with clinical improvement of patients of hypertensive, diabetic, hypertrophic cardiomyopathy, restrictive cardiomyopathy, hypo/hyperthyroidism, pulmonary hypertension and reduction of mortality from 19 % (untreated cases) to 3 % (treated cases) after the surgical procedures like Coronary Artery Bypass Grafting, Valvular repair and replacement.

We found 10 cases referral from outside have pseudonormal and restrictive filling pattern suggested by speckle tracking low value of peak untwist flow (rotR), subsequently they were advised for diastolic management. Unfortunately 5 cases could not be treated by their physicians due to diastolic parameters were normal apart from rotR value, time to rotR and E/e’ and MVO. Results have shown 2 cases were died after surgery. Some longitudinal studies has suggested as subclinical diastolic dysfunction with low peak value of rotR associated with other diastolic parameters. Our study also revealed higher mortality in asymptomatic patients as compare to symptomatic patients. Modern 2D Echo technics with multifactorial parameters are necessary to assess the diastolic dysfunction and reduce mortality in asymptomatic or mild dysnpic patients associated with Hypertension, diabetes, restrictive cardiomyopathy, valvular diaseses, atrial fibrillation and mild PH, specially they were sent for surgery with out treatment on based of some old diastolic echo, Doppler parameter with preserved LV systolic functions.

**Temporelli et al 144 patients of Congestive heart failure with DT <125 ms with duration of 2.5 years**. Study revealed DT changes after 6 month of treatment and cardiac death event rate: 37% with persistent restrictive pattern vs 11% with reversible restrictive; prolongation of short
DT was the single criteria to diagnose the restrictive filling pattern in diastolic heart failure. **Olmsted study** described the predictive significance of preclinical LV diastolic dysfunction, using the comprehensive approach of combining new TDI indexes along with classical transmitral Doppler velocities. In multivariable-adjusted analyses while controlling for age, sex and EF, mild diastolic dysfunction (hazard ratio: 8.31; p < 0.001) and moderate or severe diastolic dysfunction (hazard ratio: 10.17; p < 0.001) predicted all-cause mortality. **Hurrell** et al 367 patients of Restrictive Filling pattern : DT <130 ms 2.2 yr duration have shown mortality which revealed Survival 42% for sinus rhythm and DT <130 ms and 39% for atrial fibrillation and DT < 130. **Bella** et al 3,008 American Indians 3 yrs study shown All-cause and cardiac death E/A, All-cause mortality was higher with E/A < 0.6 or E/A > 1.5 (12% and 13%), as was cardiac mortality (4.5% and 6.5%) vs 6% and 1.6% for normal E/A ratio.

**Wang** et al 252 patients of Hypertension, median EF =51% Median 19 months follow up Cardiac death Mitral, color-coded e' (average of septal, lateral, anterior, and inferior) and Vp .Cardiac death in 19 patients -7.5%; on multivariate regression, e’ <3.5 cm/s was the most powerful independent predictor of events. **Okura** et al 230 patients of Nonvalvular Atrial fibrillation with duration of 245 +/- 200 days which revealed total mortality, cardiac mortality, incident CHF Mitral DT, S/D, DT of pulmonary D velocity, PW E/e’ (septal) Total mortality was higher at 16.7% for E/e’ >15, and 4.3% for E/e’<15; cardiac mortality of 11.1% for E/e’> 15 vs 1.4% for E/e’ <15; CHF occurred more frequently with E/e’ >15 at 17.8% vs 5.7% with E/e’ <15; E/e’ and age independent predictors of mortality. **Sharma** et al 125 patients of End-stage renal disease, LVEF 66 +/- 14% with duration of 1.5 to 1.7 yrs which revealed total mortality Mitral, pulmonary veins, Vp, and PW E/e’ (average of septal and lateral) Total mortality was 9.6% and was significantly higher for E/e’ >15; no difference in mortality between patients with and without restrictive inf low patterns.

### 6. Helpful updated diastolic Echo data in various other cardiac disorders

According to various studies the echocardiographic parameters of diastology are useful in different clinical conditions, like heart rate variability as sinus tachycardia, atrial fibrillation, hypertrophic or restrictive cardiomyopathy and constrictive pericarditis, valvular heart diseases and pulmonary hypertension.

#### 6.1 Heart rate variability –

##### 6.1.1. Sinus tachycardia-
- Mitral inflow pattern with predominant early LV filling in patients with EFs <50%, IVRT <70 ms is specific (79%), systolic filling fraction <40% is specific (88%), lateral E/e’ >10 (a ratio > 12 has highest the specificity of 96%).

##### 6.1.2. Atrial fibrillation -
- Peak acceleration rate of mitral E velocity (<1,900 cm/s2), IVRT (<65 ms), DT of pulmonary venous diastolic velocity (<220 ms), E/Vp ratio (<1.4), and septal E/e’ ratio (>11).

#### 6.2 Cardiomyopathy/ pericarditis –

##### 6.2.1. Hypertrophic Cardiomyopathy-
- Lateral E/e’ (>10), Ar _ A (>30 ms), PA pressure (>35 mm Hg), and LA volume (>34 mL/m2) are focused echo data to diagnose heart failure with normal LVEF.

##### 6.2.2. Restrictive Cardiomyopathy-
- DT (<140 ms), mitral E/A (>2.5), IVRT (<50 ms has high specificity), and Mitral septal annular e’ Usually <7 cm/s septal E/e’ (>15) Mitral inflow respiratory variation –Absent, Mitral lateral annular e’ Higher than septal e’ Ventricular septal strain is Reduced.
6.2.3. Constrictive Pericarditis - DT (<150 ms), mitral E/A (>2.3), IVRT (<50 ms), and Mitral septal annular e’ Usually >7 cm/s septal E/e’ (>13) Mitral inflow respiratory variation - Present, Mitral lateral annular e’ lower than septal e’, Ventricular septal strain is usually Normal.

6.3 Valvular heart diseases -

6.3.1. Mitral stenosis (MS) - IVRT (<60 ms has high specificity), IVRT/TE-e’ (>4.2), mitral A velocity (>1.5 m/s), E/VP <1.5 with AF

6.3.2. Mitral Regurgitation (MR) - Ar _ A (>30 ms), IVRT (<60 ms has high specificity), and IVRT/TE-e’ (<3) may be applied for the prediction of LV filling pressures in patients with MR and normal EFs, whereas average E/e’ (>15) is applicable only in the presence of depressed LVEF.

6.3.3. Aortic Stenosis (AS) - Septal E/e’ (>15), Lateral E/e’ (>12), peak Ar >30 cm/sec, Ar _ A (>35 ms), PA pressure (>35 mm Hg), E/A >1.2, DT <160, S/D <1, IVRT (<60 ms), and IVRT/TE-e’ (<3.5) if with MS <4.2 or MR < 3 may be applied for the prediction of LV filling pressures.

6.3.4. Aortic Regurgitation (AR) - Ar _ A (>30 ms), E/A >1.8, DT <140, S/D <1, IVRT (<60 ms), septal E/e’ (>15), PA Pressure >40 mmHg and IVRT/TE-e’ (<3) may be applied for the prediction of LV filling pressures.

6.4 Pulmonary hypertension -

6.4.1. Noncardiac pulmonary Hypertension - Lateral E/e’ can be applied to determine whether a cardiac etiology is the underlying reason for the increased PA pressures (cardiac etiology: E/e’ > 10; noncardiac etiology: E/e’ < 8).

7. Clinical application of diastology; At a glance

In certain patients heart failure and cardiomyopathy ventricles are unable to relax properly and cannot fill completely so blood is pushed into other body parts. This abnormal stiffening and filling of ventricles during diastole is known as diastolic dysfunction. Internal ventricular pressure is raised in next heart beat and leads to pulmonary congestion as systemic congestion if left untreated diastolic dysfunction can leads to diastolic heart failure. As per some echo studies about 50 % patients more than 70 yrs and 15 to 20 % patients less than 50 yrs had diastolic dysfunction and 60 to 70 % patients with diastolic dysfunction are male. Diastolic dysfunction in patients with valvular heart disease is characterized by an impaired isovolumic relaxation, a normal or even enhanced early diastolic filling rate and an increased myocardial stiffness.

Several mechanisms are responsible for the occurrence of diastolic dysfunction, such as increased wall stress, altered myocardial structure, subendocardial hypoperfusion and/or diastolic calcium overload. Postoperative regression of myocardial hypertrophy is beneficial in regard to wall stress, subendocardial hypoperfusion and calcium overload but diastolic dysfunction might become worse after valve replacement due to a relative increase in interstitial fibrosis consequent to the decrease in myocyte mass (= myocardial remodeling). Persisting diastolic dysfunction with a substantial rise in left ventricular filling pressure can be observed during dynamic exercise in postoperative patients with preoperative severe pressure overload hypertrophy. Thus, diastolic dysfunction can be present as a primary derangement of cardiac function and can be unmasked as an abnormal response of diastolic filling pressure during exercise.

Causes of diastolic dysfunction are chronic hypertension, Hypertrophic cardiomyopathy, Diabetes and Aging and ischemic heart disease. Although diastolic dysfunction often produce no symptoms until late stages; but can be diagnosed by echocardiography early treatment prevents the worsening symptoms of heart failure. In a patient with symptomatic heart failure with normal LV Ejection fraction, Diastolic heart failure is diagnosed. Diagnosis of diastolic heart failure is
often unfortunately missed unless physicians look for it during echocardiography procedure and should be actively managed.

We can manage this situation with active treatment of high blood pressure with ACE inhibitors, coronary artery diseases or angina with selective beta blockers, careful management of atrial fibrillation, specific and timely management of valvular heart disorders pre and post operative stages with digoxin, CCB, Amiodarone and Anticoagulants some time and managing pulmonary congestion with diuretic medicines. Finally 2D Echocardiography with modern technics pulse doppler mitral valve inflow, pulmonary vein flow, tissue dopper, color M mode propagation velocity, speckle trackling untwist time and peak velocity are useful tools in various applications to reduce mortality and morbidity.

8. Conclusion

LV Diastolic dysfunction impaired diastolic pressure and volume relationship leading to increase left heart filling pressure with in a small range of LV preload. We can differentiate diastolic heart failure from systolic heart failure based on parameters such as increased myocardial stiffness, delayed isovolumic relaxation, increased LV filling pressure and decreased LV Compliance but preserved LV Ejection fraction or systolic function when patients present dyspnoea on exertion or worsen heart failure. 2D Echocardiography with advanced technology is useful, affordable and more convenient to the patients those who have Hypertension, diabetes, thyroid disorders, infiltrative, restrictive or hypertrophic cardiomyopathy, valvular disorders, pericarditis, severe pulmonary hypertension and pre and post operative procedures.

9. Acknowledgements

I would like to thanks to professor Dr. A. K. Mahto HOD Medicine department of Rajendra Institute of Medical Sciences, Ranchi and Professor Dr. George cherian HOD Cardiology department of Narayana Hrudayalaya Institute of Cardiac Sciences, Bangalore, Texila America University(TAU), South America and Dr. Sonal Tanwar helped me a lot to write my manuscript.

I would like to thanks to all publisher for providing diagrams and figures which i have written down in reference list and finally i shall be oblised to editor from TAU.

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10.1. Journal Article


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10.2 Book Articles

