Review Article

Diastolic Function: Current Approach to Echocardiographic Diagnosis

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Abstract

Echocardiography allows the detection and quantification of diastolic dysfunction using numerous parameters that seem to make the task very complex, particularly in more severe dysfunctions. In this article, the parameters used, and the available technological resources are reviewed and updated. An algorithm is proposed to guide the progressive incorporation of the necessary variables, according to the findings. The evaluation, performed in a methodical way, following an adequate algorithm allows reaching the diagnosis with high sensitivity and specificity in almost all cases, making echocardiography the best non-invasive method for the diagnosis and evaluation of this frequent dysfunction.

Introduction

Diastolic function is the end result of a complex interaction of factors that determine the ventricular elasticity and mechanical forces that restore the volume after systole. Failure in these mechanisms leads to diastolic dysfunction, which result in increased filling pressures.

Echocardiography allows detecting the dysfunction and quantifying it, providing numerous parameters, which, in turn, must be related to clinical data.

Mitral flow analysis continues to be a fundamental part of diastolic function evaluation and is complemented by anatomical data and information derived from tissue Doppler, strain and strain rate.

Physiological and physiopathological concepts

It is considered that diastole begins and ends with aortic and mitral valves closing respectively. Diastole initial phase called isovolumetric relaxation period, finishes with mitral valve opening. In this period, left ventricle sudden "untwisting" begins, this phenomenon is not totally passive since during systole last fibers that contract, (subepicardial ascending fibers of the single muscular band that constitutes left ventricle) prolong their contraction throughout the isovolumetric period and tend to elongate the cavity in longitudinal direction, collaborating to a negative pressure gradient generation of approximately 2 to 5 mmHg between apex and base. This is responsible in large part for the rapid filling period that follows mitral opening. To this phenomenon is added the counter-torsion resulting from accumulated energy release during ventricular torsion, which was generated by the elastic compression of myocardial wall, especially the subendocardial region. Diastolic filling also depends on ventricular distensibility grade resulting from the functional integrity of the myocardial walls and the pericardium. All these mechanisms play an important role in diastolic function expressing the indisputable influence of systole on diastole, especially in its initial stage. In turn, inversely, influence of diastole on systole will occur predominantly in the diastole final phase.

The rapid filling period caused by the initial suction effect is followed by the *slow filling period* or diastasis in which there is a tendency to balance the pressures between the atrium and ventricle. The filling flow comes from the pulmonary veins. Finally, *atrial contraction* occurs, completing the ventricular filling followed by mitral valve closing.

In this way, atrium fulfills three functions during cardiac cycle.[1] *Reservoir* during systole, relaxing to receive pulmonary veins flow while mitral valve is closed.[2] Passive duct in rapid and slow filling period.[3] *Pump* during its end-diastole contraction.

One or more of the following alterations characterize diastolic dysfunction (DD): *decreased and slow relaxation, increased stiffness and decreased restoration capacity*. Filling pressures are normal initially and tend to increase with dysfunction progression. Ventricle final diastolic pressure is the first one that rises. Chronic increase in diastolic pressures leads to chambers dilation.[1]

DD is classified into three grades of severity: [2]

1. Grade I (mild): Characterized by decreased ventricular relaxation, with normal diastolic pressure
2. Grade II (moderate, or pseudonormal): Decreased relaxation and moderately increased diastolic pressure
3. Grade III (severe): Characterized by restrictive ventricular filling, and markedly increased diastolic pressure.

In DD, filling pressures will be higher than normal for a given volume. Likewise, progressive increase in diastolic volume, as happens during exercise, will induce abnormal and marked elevations in filling pressures, causing pulmonary congestion that will manifest as dyspnea.[3] The concomitant elevation of systolic pressure in pulmonary artery, in absence of pulmonary pathology, is considered criterion of diastolic pressure elevation in left chambers. In DD advanced stages, diastolic pressure will be elevated in basal conditions, with marked limitation of exercise tolerance and symptoms to minimal efforts.

Keywords

Echocardiography; Blood Pressure; Diagnosis.

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Diastolic dysfunction diagnosis by echocardiography

First patient data that we should consider are sex and age. Parameters analyzed may have different values for each sex and vary throughout life. To interpret them, we must use the corresponding reference values.[3] For example; some values considered normal in young people would be pathological in old age and vice versa.

To interpret them, we must use the corresponding reference values.3 For example; some values considered normal in young people would be pathological in old age and vice versa.

To interpret DD findings and its possible etiology, it is necessary to relate them to clinical data, symptoms, comorbidities such as hypertension, obesity, known coronary artery disease, pneumopathies, hemodialysis and others. Conditions that may interfere with the parameters used for DD diagnosis should be identified. In those cases, it is recommended to use those that have less interference. Conditions that interfere and limit diagnosis are the following: valvulopathies, especially mitral (leaflet thickening , stenosis, insufficiency or valvular ring significant calcification), aortic insufficiency, moderate to severe mitral insufficiency, valvular prostheses, elevated heart rate, rhythm disorders (especially atrial fibrillation), volume or pressure overload, ventricular hypertrophy, right heart pathologies, dyssynchrony (especially atrioventricular), pacemakers, pericardiopathies, assisted respiration.[4] In the referred cases, the variables that suffer least interference should be used. When the factors that interfere are reversible, it may be advisable to carry out a new evaluation under more favorable conditions.

Use of medication modifies the presentation pattern, being able to normalize some parameters while others persist altered, which can difficult interpretation.

Echocardiographic findings should be evaluated as a whole and not as isolated parameters. When lack of definitions or doubts appear, a greater number of variables must be incorporated for the diagnosis.

A reference electrocardiographic record should always accompany echocardiogram.

Next, most used registers and measurements will be reviewed, due to their predictive power and diagnostic utility, which are arranged according to the usual sequence of their obtaining.

Pulsed Doppler of transmitral flow

Wave E Speed (E) (cm / sec.)

It reflects the early diastolic gradient between left cavities. It is influenced by impaired ventricular relaxation and increased atrial pressure. It correlates with LV filling pressures when Left Ventricular Ejection Fraction (LVEF) is decreased. It decreases its speed inversely proportional to patient age. (Figure 1)

Wave A Speed A (A) (cm / sec.)

It reflects the late diastolic gradient between left cavities. Its decrease translates into fall in both ventricular compliance and atrial contractility. It increases its speed directly proportional to the patient age. It tends to merge with Wave A in presence of Sinus Tachycardia or First Grade Atrioventricular Block. (Figure 1)

Ratio Wave E / Wave A (E/A)

Together with the Wave E deceleration time, it is the most used parameter for DD study, to identify *a priori* the ventricular filling classic patterns: normal, relaxation alteration, pseudonormal and restrictive, in an easy reproducible way and with diagnostic and prognostic value (Figure 2). Age (decreases in direct proportion) and preload affect it at measurement time.[5] It cannot be applied in presence of atrial fibrillation or flutter due to Wave A absence. To establish a better appreciation of LV filling pressures, it is necessary to complement the E / A ratio with other parameters currently considered to be of equal or greater sensitivity for diastolic function analysis. (Figure 1)

Wave Deceleration Slope Time E (DTE) (ms)

It increases with age (normal finding), and with states that increase ventricular relaxation. Its decrease suggests high ventricular end diastolic pressures, even in presence of atrial fibrillation (not in flutter), when LVEF is decreased. (Figures 1)

Isovolumetric Relaxation Time (IVRT) (ms)

Time between aortic valve closure and opening of the Mitral Valve. It is prolonged with increased ventricular relaxation and decreases with increase in filling pressures.

Tissue Doppler of Mitral Ring

Wave e '(e'): It is the only variable related to decreased ventricular relaxation that remains altered in all DD grades.[6] It significantly translates the curve behavior of Pressure/Volume in time (tau), ventricular relaxation, forces of restoration forces and LV filling pressures[1]. (Figure 3) It is not very affected by changes in preload or afterload, although it can be altered by the coexistence of regional dysfunction (ischemia, necrosis) that involves the specific area of the ring (septal or lateral) in which it is obtained. It should be remembered that, normally, its speed is lower in the septal angle with respect to the lateral, and decreases inversely proportional to patient age.[3] The wave a' corresponds to atrial contraction and the S wave is the systolic wave.

Ratio Wave E / Wave e '(E / e')

This parameter is highlighted in most of algorithms for indicating normal or abnormal filling pressures, with values <8 and> 14 respectively, with high specificity.[3]

Although its value increases progressively with age, only 0.5% of subjects healthy will show E / e '(septal-lateral average)> 15, and 0.7% an E / e' (lateral)> 13, without significant changes according to sex.

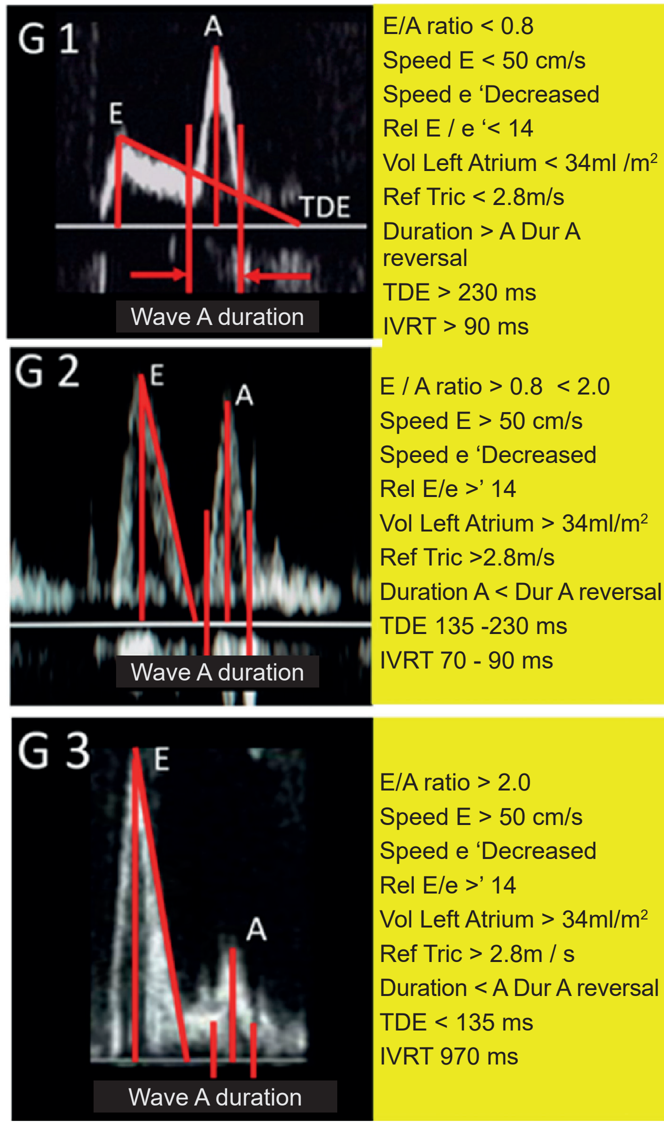

Figure 1 – *G1: Grade 1 diastolic dysfunction - Female patient 62 years - Reference values. G2: Grade 2 diastolic dysfunction - Pseudonormal Male patient 60 years - Reference values. G3: Grade 3 diastolic dysfunction - Restrictive pattern - Male patient 48 years.*

Left Atrium Volume Index (LAVI) (ml/m² SC.)

A dilated LA is important because it indicates increased diastolic pressures in left cavities in chronic form, situation with predictive value for atrial fibrillation, thromboembolism, heart failure and death.[7]

Although the Recommendations for Evaluation of Diastolic Function of ASE/EACI published in 2009 and 20164,8 suggest a cut-off point equal to or greater than 34 ml/m2.SC as abnormal, it is possible to find volumes equal to or greater than this, up to 10.5% of healthy general adult population,[3] especially in subjects with functional bradycardia, high-spending states (anemia), patients with mitral valve disease without DD and in athletes. Similarly, volumes obtained by three-dimensional echocardiography also show great variability in healthy subjects.[9] On the other hand, alterations in functional indexes (atrial strain) have been demonstrated in patients with smaller volumes.[10] Hemodynamic validation studies performed based on the aforementioned Recommendations report values with Standard Deviation limits (SD) of 26.1 (18.7-34.2) / 28.6 (± 10.9) ml / m2 SC for DD Grade I, and 26.5 (21.2-36.2) / 43.5 (± 13.3) for DD Grade II, respectively.[11] With this background,[12] in order to detect a possible atrial dilation as early as possible, it would seem advisable to consider abnormal LAVI with a lower cut-off point (> 28 ml / m2 SC), especially in subjects with established heart disease or a reasonable clinical suspicion of having it, as well as in obese (known conditioning factor for DD), in which this cut point would prevent underestimating a dilated LA due to its indexation.

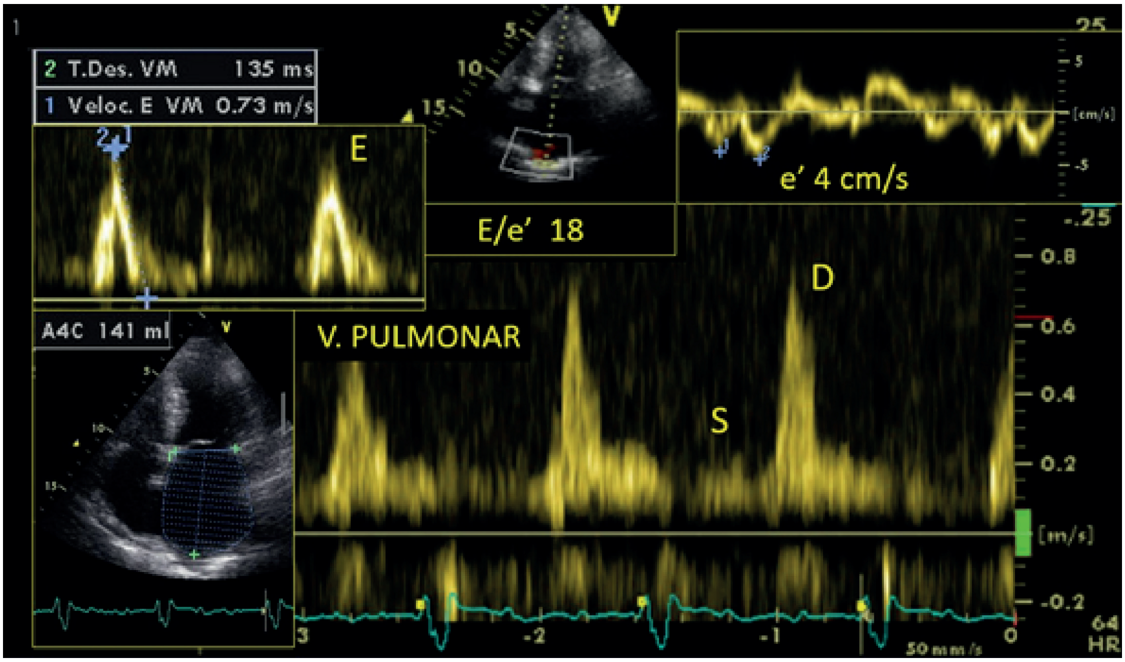

Figure 2 – *Female patient 45 years - Amyloidosis - Pulmonary vein with minimal systolic flow. Predominant diastolic flow coincides with rapid filling period, with marked decrease in meso and telediastole. It indicates restrictive diastolic dysfunction with diastolic pressure marked elevation of diastolic pressure.*

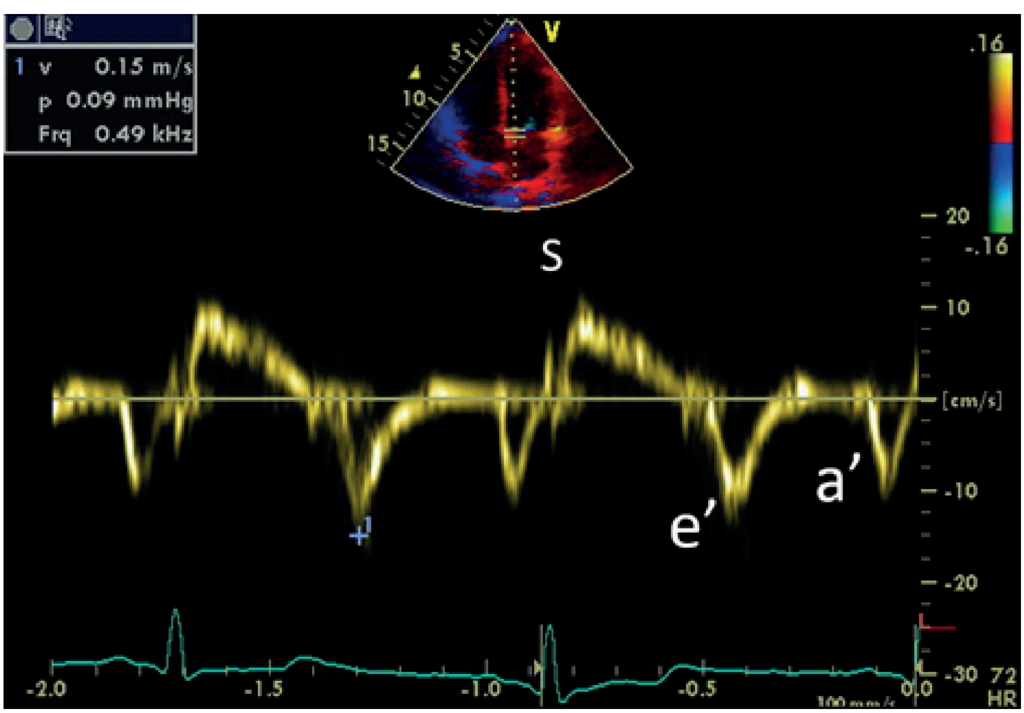

Figure 3 – *Normal tissue Doppler - Mitral ring waves.*

Maximum speed of tricuspid valve regurgitation flow (Vmax. IT) (m / sec.)

Applying Bernoulli Formula (4Vmax2), this variable calculates the peak systolic pressure gradient between the right cavities (in mmHg.). In addition to estimating the Pulmonary Artery Systolic Pressure (when adding the right atrium average pressure) when combined with other parameters of DD analysis and in absence of hypertensive pulmonary disease, it is considered that a speed equal to or greater than 2.8 m/sec. suggests LA pressure rise.[4]

Pulsed Doppler of pulmonary veins

It presents two positive waves, (Figure 4) being S (systolic), and D (diastolic) and a negative wave A retrograde (Ar).

Wave S (S) has two components of anterograde direction that are often observed fused. The first (S1) is product of the fall in LA early systolic pressure due to atrial relaxation and the second (S2) to the propagation, through the pulmonary circulation, of the pulse pressure coming from the right ventricle. Both components are influenced by the descent of the atrioventricular ring. This implies that the reduction in the speed of S translates into a decrease in the contractility of either or both ventricles. In addition, left atrial dysfunction itself may contribute to a reduced S wave, since after a poor atrial contraction, a slight fall in atrial pressure will happen during its relaxation. It is part of the S/D Ratio, a complement to diastolic study main variables. Wave D (D) is the first diastolic component of pulmonary veins flow, and like S wave, has anterograde direction. It is influenced by changes in early diastolic filling, translating the grade of ventricular and auricular compliance, which will be inversely proportional to this wave speed. It is the second component of S / D Ratio.

The retrograde A wave (Ar) is the final diastolic component of pulmonary venous flow. It has retrograde direction and represents the left atrial contraction. Its main utility is to be part of the Ar - A calculation described below.

Difference in the duration of Wave Ar minus duration Wave A (Ar - A)

Atrial contraction in a ventricle with increased end-diastolic pressure (IEDPV) prolongs the duration of pulmonary venous component (Ar) with an abnormal value by subtracting the duration of transmitral wave A greater than 30 ms. It is a variable not affected by age or ventricular contractility, and may be useful when mitral insufficiency coexists. Unfortunately, only in 65% of the cases the correct measurement of the Ar Wave is achieved.[13]

Wave S/Wave D ratio: In 73% of patients it is possible to measure S and D waves of pulmonary venous flow in LA.[13] Their normal finding is S/D> 1 and finding an inversion in that relation is considered predictive of cardiovascular events, dysfunction of one or both ventricles, diastolic pressure increase as well as of left atrium. For this reason, it is part of the complementary parameters, especially in detection of DD Grade II and III, being also useful in atrial fibrillation.

Maneuver of Valsalva: transmitral flow behavior deserves special attention with Valsalva maneuver. Properly performed (attempting to exhale air with the glottis closed, generating positive intrathoracic pressure of approximately 40 mmHg for 10 seconds), it allows to discover a normal diastolic function false, evidencing the underlying pseudonormal dysfunction when observing decreased radius E/A greater than 50% indicating increased ventricular filling pressures.[4]

(Figure 2) However, these method limitations have been highlighted since the flow radius inversion may be a normal finding in middle-aged subjects,[2] so it is suggested that the study should be complemented with other DD indices.

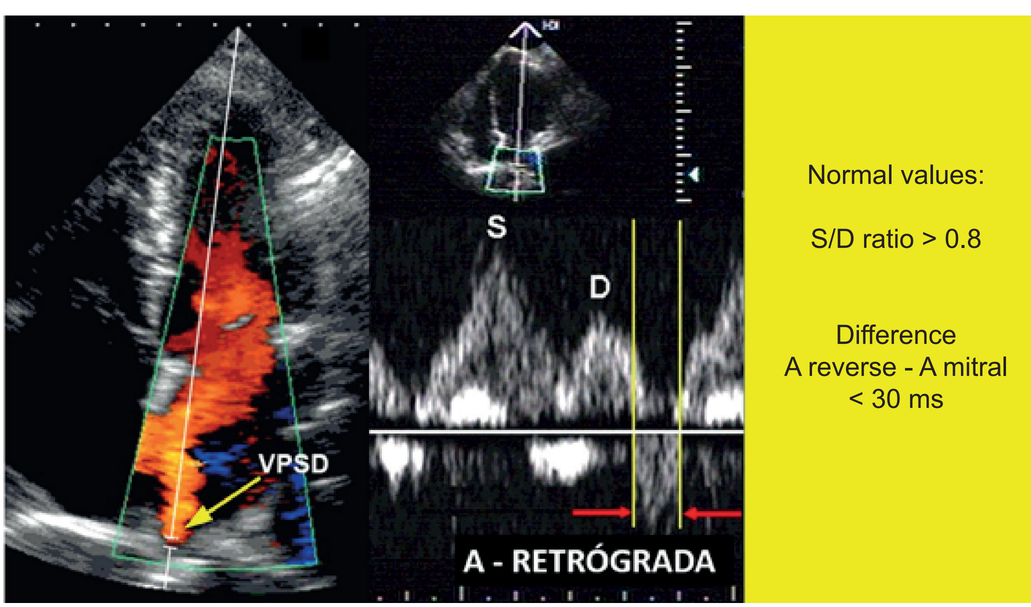

Figure 4 – *Normal flow in the pulmonary vein.*

Diastolic pressure estimation

The elevation of the diastolic pressure is confirmed when at least two of the three following variables are present: Ratio E / e '> 14; left atrial volume ≥ 34ml /m² and tricuspid reflux (RT) with speed> 2.8m / s. When only one variable is positive, we are facing what in the 2016 ASE guide was called indeterminate grade. In this case, it is complemented with pulmonary vein flow analysis. If this is normal, it indicates that the patient's evolutionary clinical picture is transitional (indeterminate) or intermediate between grade 1 and grade 2. Recent publications indicate that using left atrium strain and/or strain rate, it is possible to advance by defining DD grade in almost all patients.

New techniques and their application in diastolic dysfunction evaluation

Stress echocardiography for diastolic analysis

Although stress echocardiography has been implemented for more than 30 years and the first publications on stress and diastolic function date back more than a decade, its use has not been generalized. This technique was designed to clarify the cause of dyspnea on exertion, either as a manifestation of angina equivalent or as a relevant clinical datum of DD.[4] Ideally, this procedure is indicated in symptomatic patients, with DD Grade I estimate which does not should give symptoms. A cycloergometer or ergometric band is used (dobutamine is not recommended because of its ability to decrease diastolic pressure) to bring the patient to the desired grade of exercise (usually limited by symptoms). The test is considered abnormal when the following criteria are met: E/e 'ratio (septal - lateral average)> 14 or E / e' (septal)> 15 with exercise, Vmax. IT> 2.8 m / sec. with exercise and e' septal, <7 cm / sec. or e 'lateral <10 cm / sec. at rest. It is considered normal if the E / e 'ratio (average or septal) <10 with exercise and Vmax. IT <2.8 m / sec. with exercise. Similarly, finding exercise-induced pulmonary hypertension (Right Ventricular Systolic Pressure> 50 mmHg) concomitantly with E / e' increased at rest or with exercise, proved to be predictive of adverse cardiovascular events.[16]

Left systolic and diastolic ventricular strain

The Global Longitudinal Strain (GSL), the Systolic Strain Rate (SSR) and the Early Diastolic Strain Rate (EDSR) have proven their usefulness in DD diagnosis and classification. Retrospective study performed in patients with various pathologies, comparing them with healthy subjects and establishing cut-off points of -17%, -0.94 s-1 and 1.0 s-1 for GSL, SSR and EDSR respectively, a decrease of the first two was demonstrated in grade II and III dysfunctions, while the EDSR decreased even in DD Grade I, with Sensitivity of 83.9% and Specificity of 100% .[15] (Table 1)

Left atrium strain

Just as the left atrial volume increase (LA) is a known parameter in DD diagnosis and prognosis , the left atrial Strain analysis leads to a better understanding of diastolic phenomena by quantifying the deformation of the atrial myocardium and its speed in the different periods of the cycle, recognizing its ability to predict cardiovascular events.[16]

In the left atrium, multiple indices of diastolic function have been described. Both the volumetric analysis and the strain analysis have shown alterations associated with DD. The first one shows superposition, especially in DD grade I and II, which prevent a clear definition of the different stages only with this parameter. However, the Peak values of LA Strain maintain a tendency to decrease in all DD grades with a clear difference between them. The cut points are: normal = 37 +/- 13%, grade I = 29 +/- 8%, grade II = 22 +/- 9% and grade III = 13 +/- 6%, making it a valuable tool to stratify DD.[17] (Table 1)

Integrating it all

In recent years have been published valuable guides and algorithms that facilitate DD diagnosis. All of them propose a stratified analysis, both of the presence of diastolic alteration itself and of its grades of involvement.[4,5,8] The algorithm model detailed below (Figure 5) is based on the 2016 ASE4 Guide and the algorithm published by Mitters et al.,[13] with additional adaptations and inclusions. Its practical value lies in following the examination sequence, divided into two parts, essential analysis and complementary analysis, to estimate whether there are high LV filling pressures. For the case of indeterminate or transitional condition, left ventricular and ventricular strain analysis is incorporated. In each stage, reference values are mentioned. The incorporation of variables is progressive and it stops when sufficient information has been obtained to define and confirm the dysfunction grade. In most cases, the first part defined as essential analysis is sufficient. In complex situations, it may be necessary to use all presented parameters. Alterations detected must be confirmed by at least two different variables.

Estimation of diastolic function in special situations

Certain physiopathological conditions prevent the use of the usual parameters and algorithms developed for DD analysis. Although variants applicable to these conditions have been established, an accurate diagnosis of DD grade in

Table 1 – Behavior of ventricular and atrial strain in normal and in different grades of diastolic dysfunction.

Ventricular Strain	Normal	Grade 1	Grade 2	Grade 3
Global Longitudinal Strain (GLS)	-17 %	Normal	Decreased	Decreased
Systolic Strain Rate (SSR)	-0.94 s-1	Normal	Decreased	Decreased
Early Diastolic Strain Rate	1.0 s-1	Decreased	Decreased	Decreased
Atrium Strain				
Longitudinal Strain (LS-LA)	37 +/- 13 %	29 +/- 8	22 +/- 9 %	13 +/- 6 %

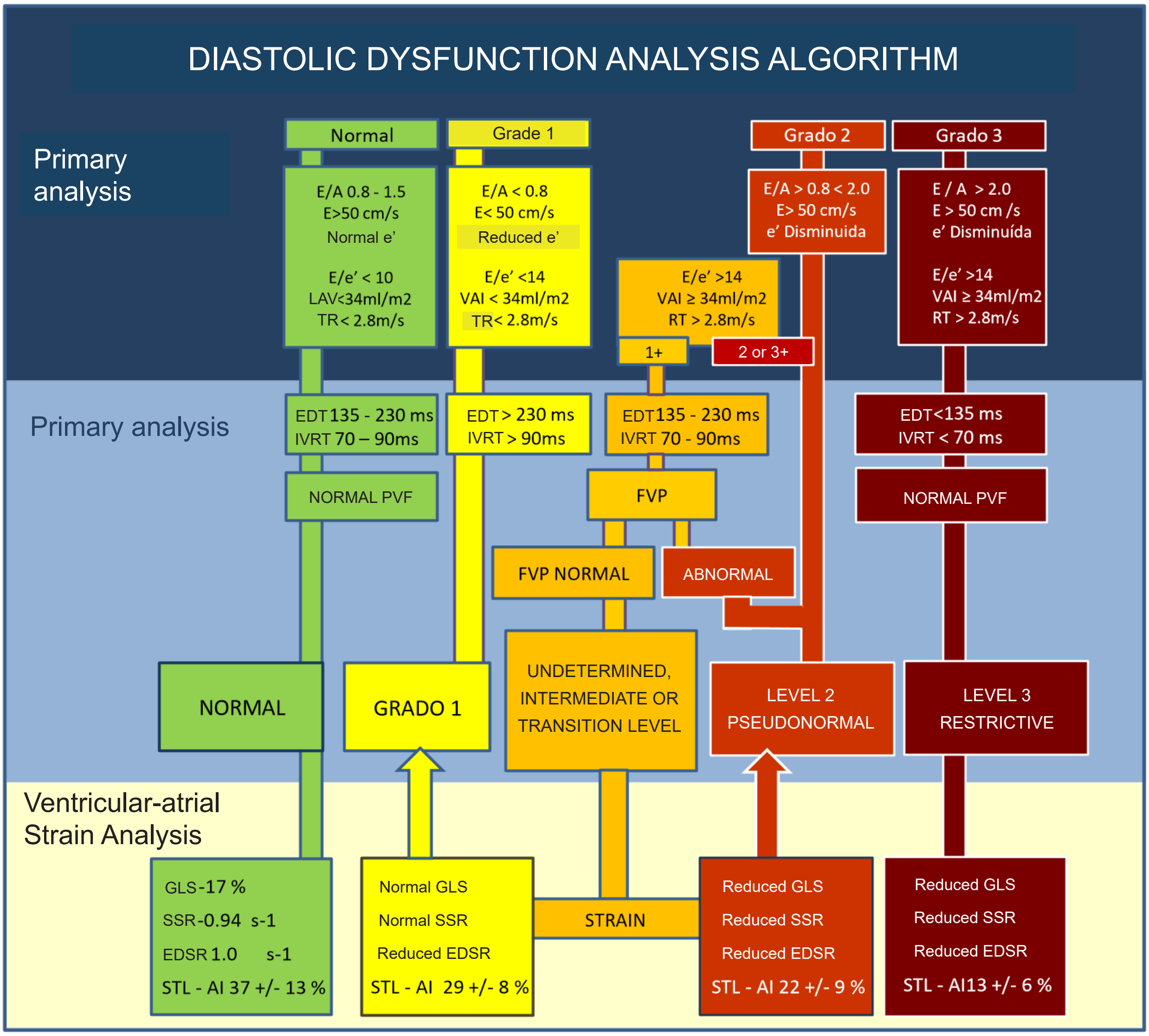

Figure 5 – *Algorithm for analysis of diastolic function. Abbreviations: E/A = Ratio Wave E / Wave A of the mitral flow. e '= Wave e' of the mitral ring. LAV = Left Atrium Volume. TR = Tricuspid Reflux. TDE = Time of deceleration of the wave E. IVRT = Isovolumetric relaxation time. FPV = Flow in pulmonary vein. GLS = Global Longitudinal Strain. SSR = Systolic Strain Rate. EDSR = Early Diastolic Strain Rate. LS-LA = Longitudinal Strain of the Left Atrium.*

patients who presents them is not always possible. It should be remembered that, finally, in absence of underlying lung disease, to have a Vmax IT greater than 2.8 m / sec. with or without dilatation of LA will be an important DD indicator, with atrial pressures elevation in all cases. The most frequent pathophysiological changes should be reviewed.

Atrial Fibrillation (AF)

The investigation of DD in subjects with AF allows not only stratifying the alteration grade, but also the prediction of cardiovascular events.[4] Progression of DD is an important condition for AF appearance. Ideally, most parameters for DD study are applied during sinus rhythm, so analyzing diastolic behavior of a patient in AF can be challenging for the echocardiographer. He faces atrial contraction absence, frequently high heart rates with irregular cadence in addition to the conditioning pathophysiological substrate. It is advisable to perform the measurements with an average of 5 to 10 beats, not necessarily consecutive, preferably at a heart rate between 60 and 80 beats per minute.

Markers used most frequently are: IVRT <65 ms. and DTE <150 ms. (both in presence of decreased LV contractility, to estimate LVEDP> 15 mmHg., and prediction of cardiovascular events), Wave D deceleration time of pulmonary venous flow> 220 ms. (related to LVEDP> 12 mmHg.), Wave S <D and E/e 'ratio (septal)> / = 11 (LVEDP > 15 mmHg with sensitivity of 75% and specificity of 93% in patients with chronic AF and contractility preserved), the latter is the most commonly used parameter, suggesting E/e '(lateral)> 10 in case of RV dysfunction or septal ischemia. These parameters are not applicable in the case of patients in AF with valve prostheses.

Mitral valve disease

The mitral ring calcification, in different grades, is present in various pathologies and in advanced age. A moderate or severe affectation can generate an increase in transmitral pressure gradients, and the consequent speed increase in their respective waves. In addition, ring restriction, secondary to calcification, will cause a decrease in e' speed, and, in turn, a rise in E/e' ratio, with a false estimate of altered LV filling pressures.

The leaflets thickening affects their mobility. Consequently, E wave speed decreases, observing a false pattern of grade 1 dysfunction, even when the function was normal.

Pure mitral stenosis occurs with normal diastolic pressures in the ventricle. Although the barrier offered by the valve affects LV filling pressures estimation, a good correlation is possible with parameters that suggest LA pressures greater than 15 mmHg. (In protodiastole IVRT <60 ms and in end-diastole Wave A> 1.5 m / sec.). The delay in e 'wave expression in the tissue Doppler of mitral ring respect to Wave E of the transmitral flow is a sensitive data of LA increased pressure establishing the relationship IVRT / T (E-e') <4.2. The E / e 'ratio is not useful in mitral stenosis.

In the case of mitral insufficiency, DD parameters can be safely applied in secondary or functional valvular diseases, as these reflect alterations in secondary intracavitary pressures both to myocardial damage and to the underlying valvular damage. In the case of primary mitral insufficiency, alterations could be due to volumetric changes of adaptation to valve disease before DD. They are suggestive of high LA pressure (greater than 15 mmHg.) In the presence of normal LVEF: IVRT <60 ms., Ar-A> 30 ms. And IVRT / T (E-e ') <5.6; with depressed LVEF '> 14, and IVRT / T (E-e') <3 with or without LVEF depression.[4]

Cardiomyopathies (CM)

Unlike dilated CM in which diastolic function alterations coexist with dilated cavities and global and segmental ventricular contractility disorders, and broad application of DD parameters in the majority of cases, Restrictive and Hypertrophic CM have specials characteristics that should be known and applied.[4]

Restrictive CM, with varied etiologies, can show gradual evolution in its diastolic function deterioration until reaching Grade III with altered relaxation and increased left ventricular filling pressures. It will have unequivocal restriction Doppler signs, that is, E / A ratio> 2, narrow IVRT (<50 ms.) and DTE <150 ms., As well as Wave e '(lateral and medial) greatly diminished and E / e' ratio > 14. The diagnosis is complemented with dilatation of one or both atria and increased parietal thickness of the ventricular myocardium. In the case of cardiac amyloidosis, there will be an increase in echogenicity with a characteristic "granular pattern". Contractility is generally preserved, allowing this CM to be differentiated from other pathologies with restrictive filling. In the particular case of Amyloidosis, the GSL will allow us to verify the null or slight involvement of the apical zone regional strain with significant decrease in middle and basal regions values .

Differentiation with Constrictive Pericarditis, from the point of view of Doppler, is that the Wave e 'of the septal DT is always greater than the lateral one with generally normal velocities, unlike the restrictive CM which keeps the lateral relation e' greater than septal 'e. Hypertrophic CM, on the other hand, shows a wide range of individual presentation (differences in ventricular mass, myocyte disorders, presence and grade of obstruction, etc.). However, it is useful to stratify risk to find increased left atrial volume (> 34 ml / m2), restrictive E / S ratio (> 2), Vmax IT> 2.8 m / sec, Ar - A time> 30 ms. and Increased E / e 'ratio (E / e'> 14 predicts heart failure, syncope, AF or sudden death). In case of severe mitral valve insufficiency, only the Ar - A and the Vmax IT are valid. The Strain Rate of the IVRT and the Early Diastolic Peak (SRedp) were observed to be diminished in patients with significant obstruction of the LV outflow tract, developing the wave ratio E/SRedp as an indicator of increased LV filling pressures.[4]

Intraventricular, interventricular and atrioventricular dyssynchrony

Intraventricular dyssynchrony

The myocardial deformity evaluation (strain) determines the intensity and moment of contraction of each myocardial segment, allowing evaluating intraventricular mechanical synchronism. Electrical dyssynchrony is always accompanied by mechanical dyssynchrony. However, there may be intraventricular mechanical dyssynchrony without electrical dyssynchrony due to myocardial abnormalities, such as in cardiotoxicity and in some cardiomyopathies. Myocardial ischemia slows down the contractile response of ischemic segments, and may cause post systolic contraction, that is, after aortic valve closure. Regardless of etiology, post-systolic contraction occurs once diastole has begun, which clearly compromises ventricular relaxation and distensibility, negatively impacting diastolic function.4 (Figure 6 A)

Systolic dyssynchrony is accompanied by varying grades of diastolic dyssynchrony, which also compromise ventricular relaxation and elasticity. (Figure 6 A)

Contraction and relaxation speed can be analyzed with strain rate. Depending on etiology and underlying myocardial injury grade, speed can be very heterogeneous, thus adding information for better DD understanding in the advanced forms of cardiomyopathy. (Figure 6 B)

Interventricular dyssynchrony generally accompanies intraventricular dyssynchrony, being an aggravating factor for both diastolic and systolic function.

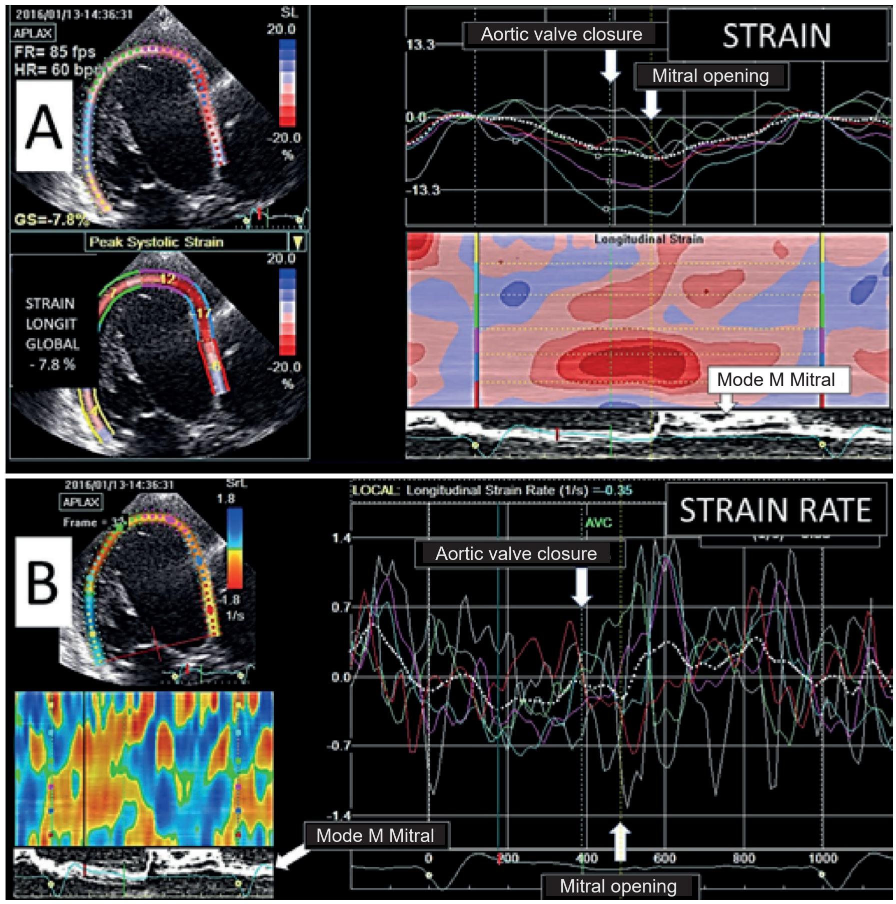

Figure 6 – *Male patient 51 years - Dilated cardiomyopathy with intraventricular dyssynchrony. A: Dyssinchrony longitudinal strain curves are observed, with postsystolic peaks occurring after the mitral opening, which can be seen in M mode in the lower part of the figure. B: Strain rate curves with great dyssynchrony and wide variation of the deformation velocities are observed.*

Atrioventricular dyssynchrony is recognized by the increase in the aortic pre-ejection period greater than 140ms and by diastole time shortening, with duration less than 40% of cardiac cycle. (Figure 7) In this dyssynchrony, atrial emptying time decreases, which increases the diastolic pressure at that level, causing symptoms in pulmonary circulation. This condition can be observed in important cardiomyopathies and arrhythmias with rapid ventricular frequency as in atrial fibrillation or flutter. It is also seen in pacemaker carriers with bicameral stimulation or resynchronizers, indicating then, adjustment of the cardiac stimulation device.[4]

Conclusion

The diastolic function evaluation is supported on numerous parameters that seem to make the task very complex, particularly in the most severe dysfunctions. However, the evaluation carried out methodically, following an appropriate algorithm, progressively incorporating the necessary variables according to findings, allows reaching the diagnosis with high sensitivity and specificity in the vast majority of cases. The small number of patients that, when evaluated by the usual parameters, present a grade of indeterminate, intermediate or transition dysfunction, can be defined with the use of longitudinal strain and strain rate techniques, or with stress echocardiography for diastolic analysis. Echocardiography is the choice method for DD evaluation.

Ideas to remember

1. Use reference values corresponding to patient sex and age
2. To evaluate if the patient has any alteration that could interfere with the variables interpretation used for DD evaluation
3. For DD evaluation, essential analysis parameters are E/A ratio, wave speed E, wave e ', E/e ratio, left atrium volume and tricuspid reflux rate.
4. For DD evaluation, complementary analysis parameters are TDE, TIVR and pulmonary vein flow. Indeterminate or transitional cases between grade 1 and 2 can be defined using Left Ventricle and Left Atrium Strain.

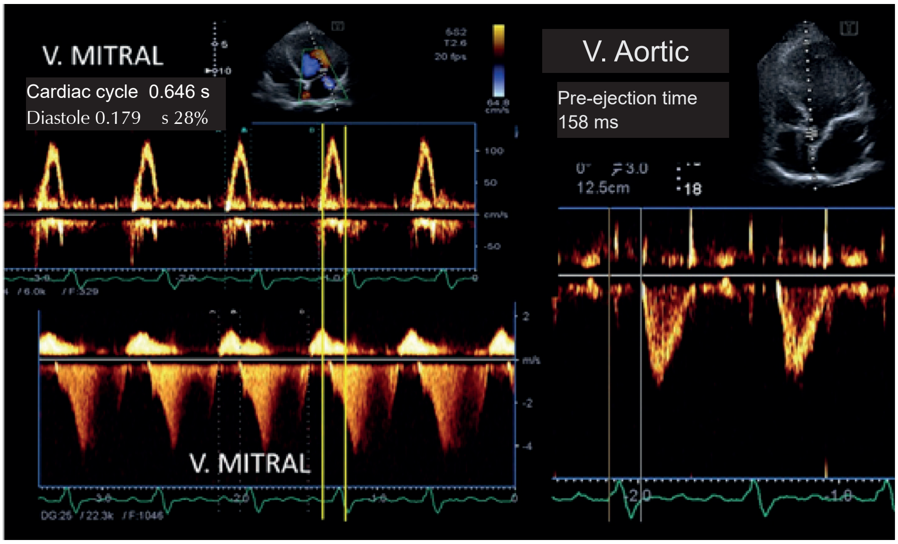

Figure 7 – *Male patient 46 years Atrioventricular dyssynchrony Aortic pre-ejection time 152ms (VN <140ms) Ventricular filling time equivalent to 28% of the cardiac cycle (VN> 40%).*

Authors contribution

Sanchez-Osella CF, Castilla-Fassio R and Campos-Vieira ML carried out the research conception and design, data collection, data analysis and interpretation, statistical analysis, manuscript writing and critical review regarding the important intellectual content.

Potential conflict of interest

The authors declare that there is no relevant conflict of interest.

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