Diastolic Stress Echocardiography

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Maurizio Galderisi and Eugenio Picano

Stress echocardiography is an established and mainstream method for the diagnosis and risk stratification of patients with known or suspected coronary artery disease [1, 2]. While the accuracy and clinical usefulness of echocardiography in assessment of regional and global systolic function is undisputed, the assessment of diastolic function is important, but it remains difficult by echocardiography at rest [3, 4], and even more during stress [5, 6]. In spite of the many unsolved conceptual and methodological issues, there are three reasons for the still high interest of the cardiological and echocardiography community in diastolic stress echocardiography: (1) experimental [7–9] and clinical [10–12] studies clearly show that acute diastolic dysfunction is an early event in the ischemic cascade, even earlier than regional systolic dysfunction, which remains the cornerstone of clinical diagnosis with stress echocardiography [13]; (2) diastolic dysfunction can accompany systolic dysfunction, but its presence and severity adds risk to the negative prognostic value of resting [14] or stress-induced systolic dysfunction [15]; and (3) in a high proportion of patients, "diastolic" heart failure is the dominant form of dysfunction, without any detectable systolic dysfunction at rest and during stress [16].

According to European Society of Cardiology 2007 guidelines, in presence of clinical symptoms, mainly dyspnea, and normal left ventricular (LV) systolic function with normal LV volumes, the diagnosis can be achieved by combining standard transmitral Doppler (transmitral E velocity), pulsed tissue Doppler of the mitral annulus (early diastolic velocity = e′) and clinical biochemistry criteria. Diastolic heart failure will be diagnosed in the presence of the E/e′ ratio >15 + BNP >300 and excluded with E/E′ ratio <8 and BNP <200 [17]. However, it is not infrequent for the patient to fall within a gray zone of indeterminate values (Fig. 25.1). In these patients, resting echocardiography may help with indirect, supportive signs showing structural changes frequently associated with LV diastolic dysfunction, such as left atrial dilation (left atrial volume index>28 mL m^{-2}) [18] and LV hypertrophy, but obviously a more direct documentation of diastolic dysfunction would be most helpful. These patients are the main clinical target of diastolic stress echocardiography.

E. Picano, *Stress Echocardiography*,

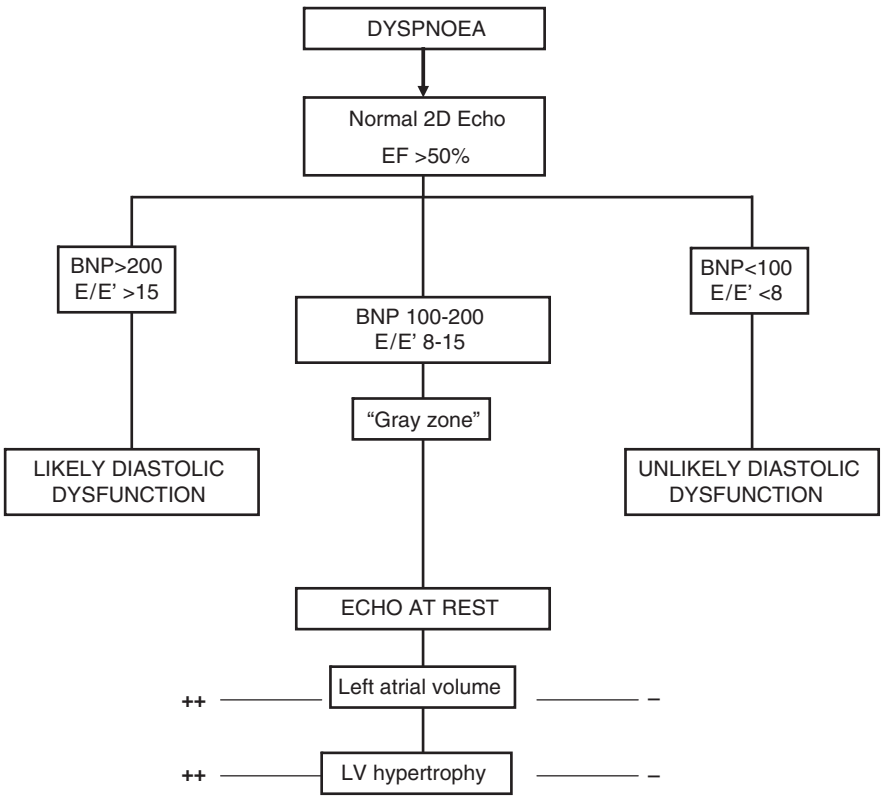

Fig. 25.1 The diagnostic algorithm for diastolic heart failure proposed by the European Society of Cardiology in 2007. Transthoracic echocardiography has a central role and is the imaging test of choice in excluding systolic dysfunction (normal ejection fraction and left ventricular volumes) and in including diastolic dysfunction, through direct, specific signs (E/e′ increase) and indirect, supportive signs (left atrial dilation and left ventricular hypertrophy). (Modified from [17])

25.1 Pathophysiological Basis of Diastolic Dysfunction

From a pathophysiological standpoint, LV diastolic function should always be viewed as a two-dimensional (2D) problem, including not only LV filling pressures but also LV volumes. ESC criteria establish a cut-off value of wedge pressure greater than 15 mmHg and LV end-diastolic pressure (LVEDP) greater than 15 mmHg to clearly document LV diastolic heart failure [17]. However, for any given pressure, LV end-diastolic volume is also important. In fact, at normal LV filling pressure, the normal heart shows end-diastolic volumes necessary to grant the adequate stroke volume. The stiff heart of diastolic dysfunction has similar volumes at higher pressures or, alternatively, normal pressures and smaller volumes. Any stress on the heart, including a simple tachycardia, is also a

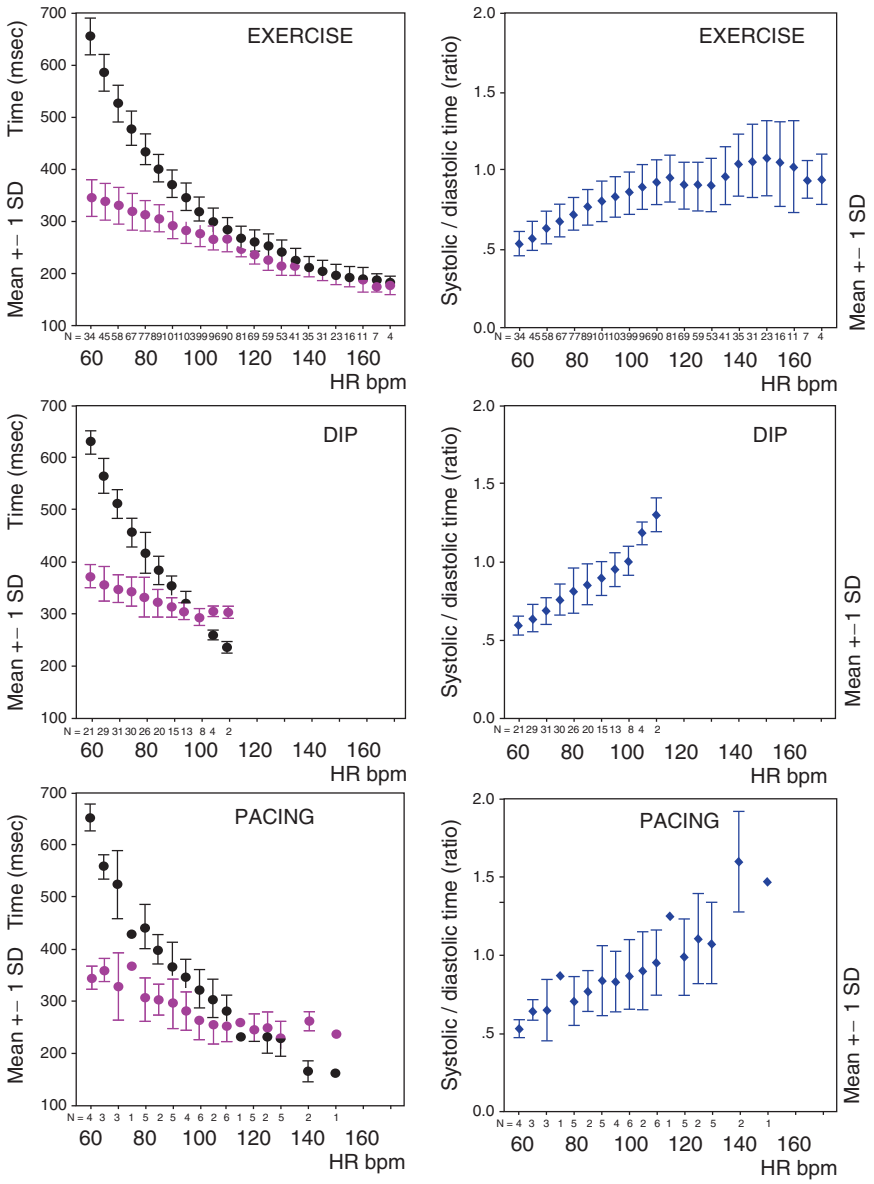

Fig. 25.2 The reduction of diastolic time (*black dots*) is much steeper than the reduction in systolic time (*red dots*) during tachycardia associated with exercise (*upper left panel*), dipyridamole (*middle left panel*), or pacing (*lower left panel*). The ratio between systolic and diastolic tissue increases during exercise (*right upper panel*), dipyridamole (*middle right panel*) and pacing (*lower right panel*)

powerful diastolic stress, since the same filling must be achieved in a much shorter time (Fig. 25.2). The positive lusitropic (improved myocardial relaxation) effects of adrenergic stress (or exercise) determine better filling in a shorter time, and in fact the normal diastolic

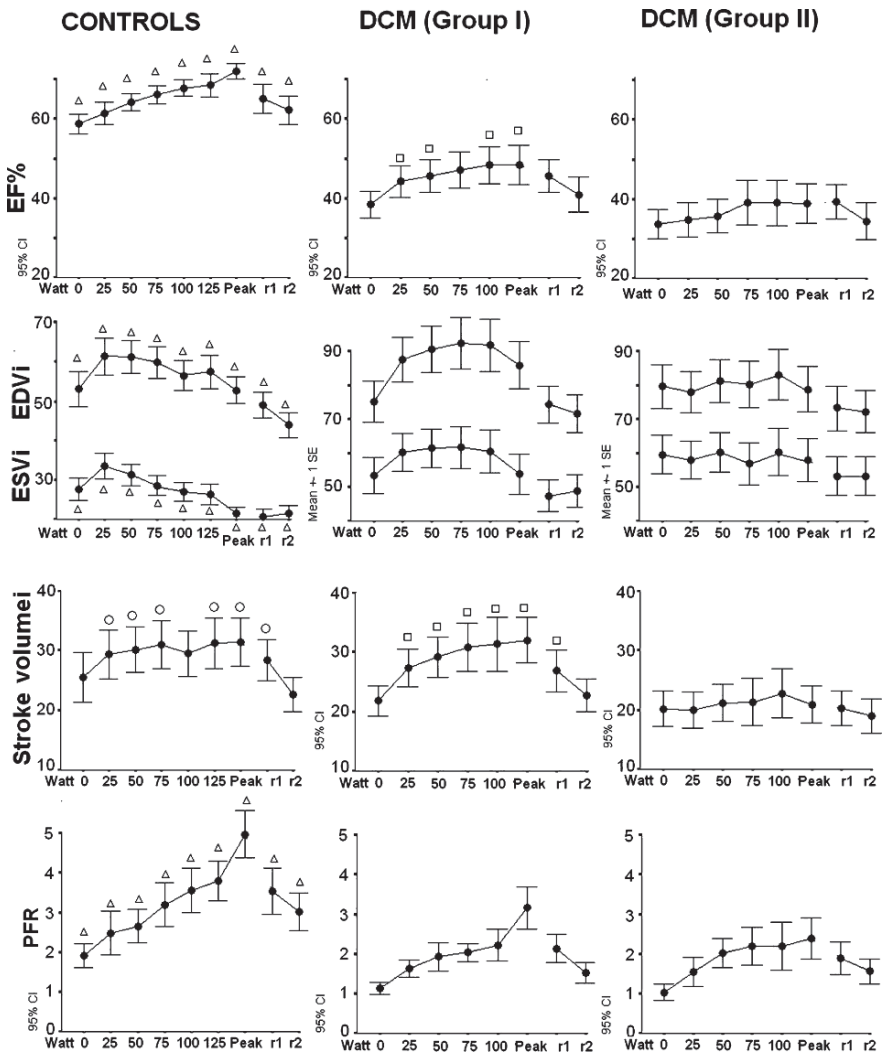

Fig. 25.3 Changes in left ventricular volume during exercise in controls, dilated cardiomyopathy patients with relatively good (Group I) and relatively poor (Group II) prognosis with exercise. The end diastolic volume is increased only in normal and Group I dilated cardiomyopathy patients. (Modified from [19]) DCM, dilated cardiomyopathy; EF, Ejection Fraction; ESVi, end-systolic volume index; EDVi, end-diastolic volume index; Stroke Volume i, stroke volume index; PFR, peak filling rate

response to stress is a reduction of LV end-systolic volume (due to an increase in contractility) and an increase in end-diastolic volume, normally occurring in the early phases of exercise, and then a plateau at intermediate to high levels of stress, up to a point when the diastolic reserve is exhausted and LV filling declines [19] (Fig. 25.3). This decline in LV filling can occur at lower heart rates in ventricles with diastolic dysfunction. The lower the diastolic filling, the lower the stroke volume, and, for any given level of systolic dysfunction, the worse the prognosis.

25.2 The Echocardiography Assessment of Diastolic Function

The four grades of LV diastolic function can be summarized as in Fig. 25.4 [20]. The assessment of diastolic function has been made easier, simpler, and more accurate by combining the transmitral E velocity with the E′ derived from pulsed tissue Doppler, i.e., the E/e′ ratio [21], which is obtained more often, more rapidly, and more easily than pulmonary blood flow and transmitral flow during Valsalva [22], which are alternative ways to separate a normal from a pseudonormal pattern. Left atrial volume is also useful as a marker of the severity and duration of diastolic dysfunction [18, 23], perhaps obviating the need for more complex characterization of diastolic function and filling pressures with Doppler echocardiography. Doppler indexes reflect LV filling pressures at one point in time, whereas increased left atrial size may better reflect the memory of the same filling pressures, i.e., the cumulative effect of filling pressures over time, in a way conceptually similar to glycated hemoglobin in diabetes. As a single assessment of glycemia is affected by dietary factors such as the sugar intake immediately prior to testing, Doppler index changes are affected by loading conditions unrelated to true LV diastolic dysfunction, and an increase in heart rate or a preload increase induced for instance by nitrates may induce a pseudorestrictive pattern. The integration of structural, 2D-based parameters on left atrial

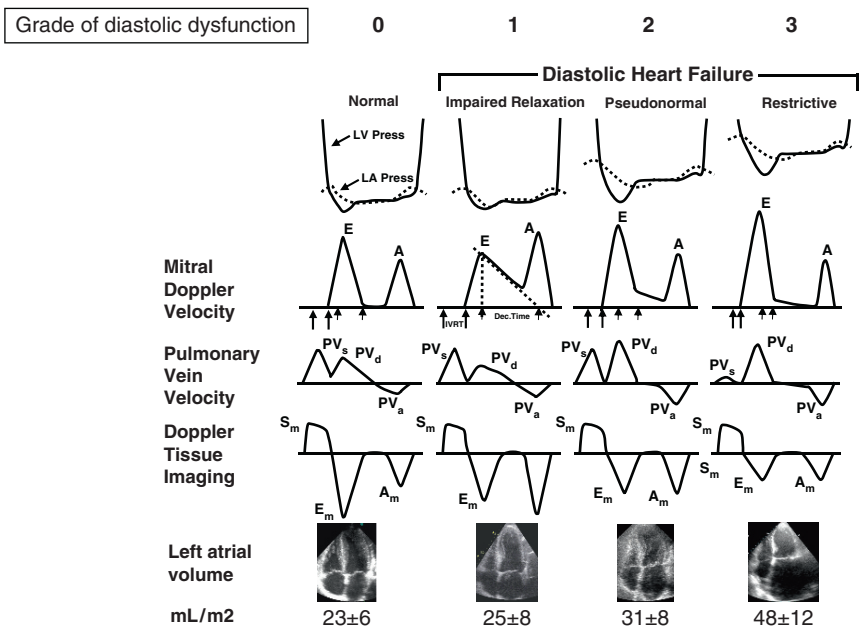

Fig. 25.4 The severity of diastolic dysfunction (from 0 = absent to 3 = severe) titrated by a combination of transthoracic echocardiography assessed with Doppler, tissue Doppler, and 2D indices. (Modified from [20])

remodeling and functional, Doppler-based markers of altered filling offers a comprehensive picture of diastolic dysfunction.

The assessment of LV diastolic function at rest has independent and additive prognostic value [24] over resting and stress-induced systolic dysfunction. The worse combination is represented by a LV diastolic restrictive pattern [25] with lack of contractile reserve in dilated cardiomyopathy patients [26] or a dilated left atrium with inducible ischemia in coronary artery disease patients [27].

The abnormal diastolic stress echocardiographic response is characterized by lower LV cavity dilation, higher E/e′ values, and possibly increases in pulmonary arterial systolic pressure (PASP) and ultrasound lung comet (ULC), surrogate signs of diastolic dysfunction mirrored in backward failure with pulmonary congestion up to interstitial lung edema [28, 29]. However, the clinical data supporting this framework are scanty to date. In a more advanced phase of LV diastolic restrictive dysfunction, the stress should be aimed at unloading the ventricle and improving the lusitropic properties: a viability test of diastole. For this purpose, the nitroprusside test can be useful [30].

25.3 Clinical Results

The ratio of transmitral E (peak early diastolic velocity) to pulsed tissue Doppler-derived e′ of the mitral annulus can be used to estimate LV filling pressures at rest and during exercise. Healthy individuals will show a similar increase in mitral E and annular e′, such that the ratio has no or minimal change with exercise. Patients with impaired LV relaxation develop an increase in LV filling pressures with exercise as a result of tachycardia and the abbreviated diastolic filling period. Accordingly, transmitral peak E velocity increases. However, given the minimal effect of preload on annular e in the presence of impaired relaxation, annular e′ remains reduced. Therefore, the E/e′ ratio increases with exercise in patients with diastolic dysfunction [31, 32]. Diastolic stress echocardiography has been demonstrated to be feasible using supine bicycle exercise [31] and is based on the assumption that the ratio of early diastolic transmitral velocity to early diastolic tissue velocity correlates with invasively measured LVDP during exercise [31, 32]. The algorithms to interpret the exercise-induced changes of the E/e′ ratio are summarized in Fig. 25.5 [31]: the three different responses to exercise have a different meaning and the passage from normal to abnormal LV filing pressure is crucial to unmask patients who cannot be appropriately defined by simple Doppler assessment at rest. Diastolic stress echocardiography has been applied in several clinical settings, including patients with normal systolic function [33] and with myocardial diastolic relaxation at rest [34], patients with ischemic heart disease [35], diabetic patients [36], and those with hypertrophic cardiomyopathy [37].

The methodological approach during stress is less clearly standardized and issues remain on feasibility, accuracy, and prognostic value. The parameters should be the same as at rest, but the two-key signal of transmitral flow and annular velocities are extremely sensitive to tachycardia, which leads to diastolic wave fusion making the tracings impossible to read and to loading condition changes that can make them difficult to interpret (Figs. 25.6 and 25.7). The E/e′ ratio is somewhat correlated to increase in LV end-diastolic pressure, but it

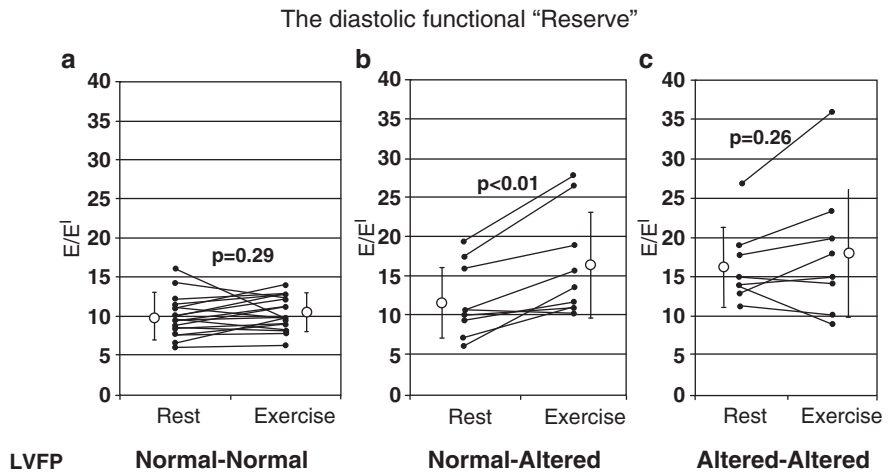

Fig. 25.5 The three possible changes of the E/e′ ratio during exercise. (Modified from [31])

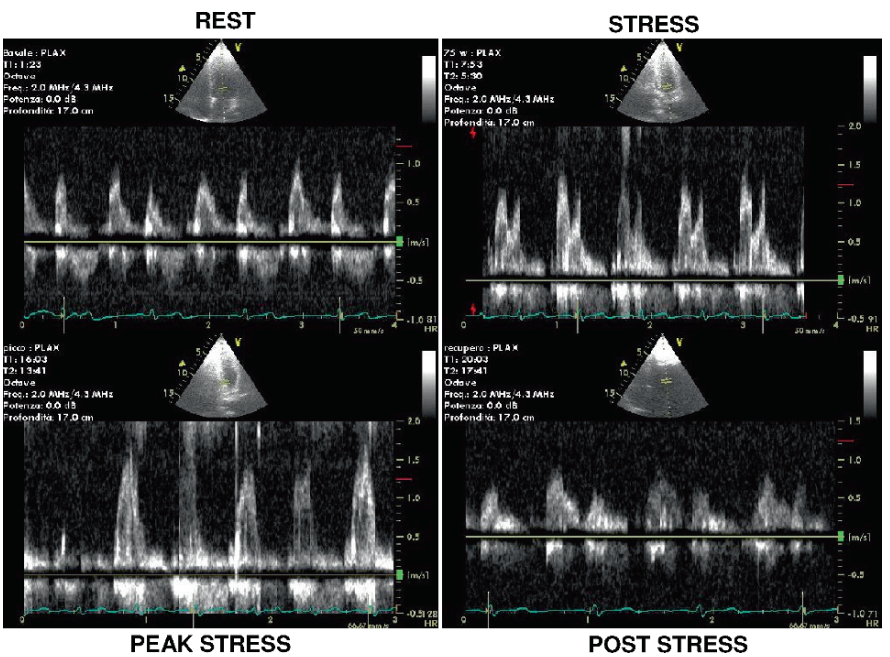

Fig. 25.6 Transmitral flow velocity tracing at baseline (*left upper panel*) and during stress. There is an obvious fusion of waves of mitral flow profile at high heart rate (*lower left panel*). The flow becomes readable again in the recovery phase (*lower right panel*)

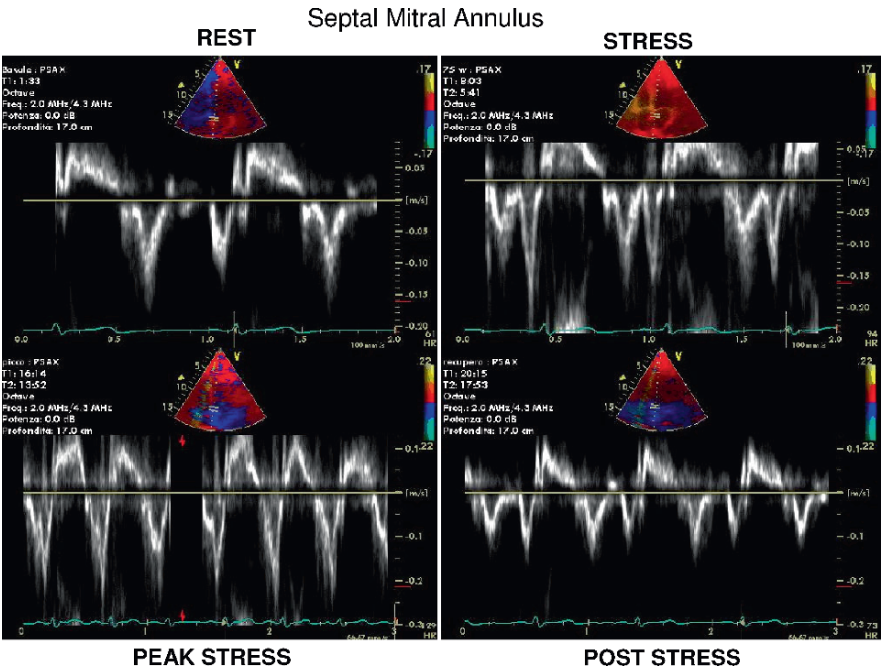

Fig. 25.7 Tissue Doppler imaging of mitral annulus (septal side). The 2 E′ and A′ waves are clearly distinguishable at rest and in the recovery phase, but are fused (and uninterpretable) at peak stress

can usually be measured only at submaximal levels of stress or in the recovery phase. The ventricular volumes are easy to obtain from 2D apical 4- and 3-chamber views with the Simpson biplane method, and may benefit substantially from real-time three-dimensional (RT3D) technology, which provides more information (avoiding geometric assumptions and foreshortened views inherent to the 2D approach) in less imaging time. However, they have been virtually ignored to date in the assessment of diastolic function during stress echocardiography, whereas they are a known key player in diagnosis with radionuclide angiography [38].

According to ASE guidelines on stress echocardiography released in 2008, diastolic stress echocardiography might be useful "for the evaluation of patients with dyspnea of possible cardiac origin" [39]. This evaluation is especially important in patients without inducible wall motion abnormalities, in whom dyspnea can be an angina equivalent, and in patients in whom the diagnosis of diastolic heart failure can be ascertained by the findings of resting transthoracic echocardiography, as described above. When stress testing is needed, the recommended stress is exercise, which is necessary to evoke dyspnea independent of inducible ischemia, and the semisupine approach allows the acquisition of Doppler recordings during exercise. Doppler assessment of the mitral inflow velocities should be assessed at rest, during exercise, and in the recovery when the E and A velocities are no longer fused. Doppler recordings should be acquired at a sweep speed of 100 mm s^{-1}.

There are conceptual and practical limitations to the above methodology. They include atrial fibrillation, technically challenging imaging windows, and limited validation. It remains to be seen how abnormalities in regional function influence the accuracy of a single site measurement (septal or lateral) of e′ . Using pulsed tissue Doppler, early diastolic velocities are usually lower at the septal portion of the mitral annulus, where the alignment of the Doppler scan is optimal, and higher at the lateral corner where the Doppler alignment is suboptimal, but the signal is free of the influence of right ventricular longitudinal motion [40, 41]. More importantly, the value of reduced LV end-diastolic volume reserve, coupled with E/e′ , remains to be evaluated as a marker of diastolic dysfunction. This approach requires two coordinates (volume and pressures) instead of one to be correctly described. Also PASP might offer ancillary, supportive information, since diastolic failure during stress induces a backward failure with pulmonary congestion. PASP is also important per se in the work-up of patients with dyspnea of unknown origin, since for instance a rise in PASP in the absence of diastolic dysfunction and valvular or other pulmonary disease can be found in patients with primary or secondary pulmonary hypertension as an initial stage of the disease, when resting values are still normal [29].

At present, we still need more data collected in a more comprehensive fashion in patients where they are more meaningful. To document diastolic dysfunction in patients with established resting systolic or diastolic dysfunction is probably of limited value. It would be more important to establish the diagnostic and prognostic correlates, and the true feasibility, of integrated (ventricular pressures and volumes, PASP and ultrasound lung comets) diastolic stress echocardiography in the subset of challenging patients such as those in the present diagnostic gray zone of diastolic heart failure. These studies are conspicuously missing to date but will determine the ultimate clinical role of the promising, challenging, yet innovative diastolic stress echocardiography.

25.4 A Roadmap to the Future

It is now possible, at least in theory, to test an array of candidate markers prospectively for the characterization of LV diastolic function during stress (Table 25.1). It is also possible to outline for diastolic dysfunction different stages of natural history, corresponding to different stages of severity, as clearly coded with stress echocardiography for systolic dysfunction: normal (at rest and during stress), initial damage (normal at rest, abnormal during stress), advanced damage (abnormal at rest; fixed abnormality during a diastolic unloading stress), and irreversible damage (abnormality at rest, fixed with an unloading stress) (Table 25.2). At most advanced stages of overt diastolic dysfunction, stress echocardiography may help in unmasking fixed vs. reversible patterns, the latter being less prognostically malignant. A restrictive fixed pattern is more dangerous than a restrictive reversible pattern, which can be at least partially normalized by nitroprusside infusion [42].

Compared to the conceptually germane systolic dysfunction, the pathophysiological basis of diastolic dysfunction is more complex, the technical approach more difficult, stresses less well standardized, and clinical experience largely immature. Still, there is

Table 25.1 Diastolic function during stress: the challenge continues

	Normal	Diastolic dysfunction
2D (RT3D)		
LV ED volume	↑	↔
Left atrium	↔	↑
Pulsed-wave Doppler (TDI)		
E/e′	↔	↑
CW Doppler (TR)		
PASP	↔	↑

Potential use in patients with dyspnea of *possible* cardiac origin
Still limited validation. Exercise is still the recommended stress (*ASE guidelines 2007*)
CW continuous wave, *RT3D* real-time three-dimensional, *LV* left ventricular, *ED*, *TDI* , *TR* , *PASP* pulmonary arterial systolic pressure 2D (RT3D), Pulsed-wave Doppler (TDI), CW Doppler (TR) in bold.
Legend: *ED* end-diastole, *TDI* Tissue Doppler imaging. *TR* Tricuspid regurgitation

Table 25.2 Stages of diastolic dysfunction

	Rest	Stress	Which stress	Key parameter
Stage I (normal)	Normal	Normal	Exercise	E/E′
Stage II (initial)	Normal	Abnormal	Exercise	E/E′
Stage III (advanced)	Abnormal	Normal	Nitroprusside	E wave DT
Stage IV (irreversible)	Abnormal	Abnormal	Nitroprusside	E wave DT

DT deceleration time

little doubt that diastolic dysfunction exists: it is clinically and prognostically important in CAD and in many other patients such as cardiomyopathy, valvular, and congenital heart disease patients, and echocardiography must find a better way to study it.

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