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Left and right ventricular diastolic dysfunction and diastolic heart failure: does one lead to the other?

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Background and Objective Diastolic dysfunction of the left ventricle is a mechanical abnormality diagnosed primarily by echocardiogram, and can be distinguished into three separate degrees based on the severity of reduction in passive compliance and active myocardial relaxation. Methods A literature search was performed for basic science studies, clinical studies and major practice guidelines on the subject of diastolic dysfunction and diastolic heart failure. Important findings were analyzed and correlated with regard to clinical relevance. Results Left ventricular diastolic dysfunction appears to compromise exercise tolerance and is believed to contribute to the pathophysiology in patients with diastolic heart failure. In the clinical setting, however, often times no clear distinction is made between echocardiographically diagnosed diastolic dysfunction and diastolic heart failure, and adequate treatment recommendations are sparse and aimed to prevent worsening and progression of clinical symptoms. To date, there is a lack of high powered trials assessing the possible progression rate from echocardiographically diagnosed diastolic dysfunction to the clinical diagnosis of diastolic heart failure. Furthermore, there are no solid indices to assess the degree of severity of diastolic dysfunction or its progression. Pure right ventricular diastolic dysfunction appears to be even less understood and under-recognized, although it may play a role in the development of both right and left heart failure. Currently there are few but interesting data on the possible interaction between ventricles with diastolic dysfunction and the overall affect on the development of heart failure. Conclusions The timeline and progression of diastolic dysfunction to diastolic heart failure have not been well established and warrant further investigation. (J Geriatr Cardiol 2009; 6:3-10)

Key words Diastolic dysfunction; diastolic heart failure; left ventricular dysfunction; heart failure; cardiomyopathy

Introduction

By definition there is a distinction regarding the terminology of diastolic dysfunction and diastolic heart failure. Clinically, however, these terms are often exchanged. Based on a recent PubMed literature search, more than two hundred review articles on diastolic heart failure have been published between May 1990 and March 2008. Due to lack of randomized controlled trials there remains controversy regarding optimal therapy in patients with diastolic dysfunction and diastolic heart failure. Even less data are available on pure right ventricular diastolic dysfunction. An important question is whether non-invasively i.e., echocardiographically-diagnosed diastolic dysfunction does indeed lead to diastolic heart failure, and if so, over what time period and how this progression occurs. Moreover, does diastolic dysfunction consequently result in both diastolic and systolic heart failure? Should diastolic dysfunction be seen as a completely separate entity that requires a different treatment approach? In clinical practice, chronic heart failure is divided into systolic and diastolic heart failure principally based on the preservation of left ventricular ejection fraction, with the assumption that reduced ejection fraction heart failure is primarily due to systolic dysfunction while diastolic dysfunction is the main culprit in heart failure with preserved ejection fraction, normally considered as an ejection fraction above or equal to 45%. The terms “heart failure with preserved left ventricular function” or “heart failure with normal ejection fraction” are utilized to emphasize that the etiology of the pathophysiology for this group of patients may go beyond diastolic dysfunction alone.

Heart failure with preserved ejection fraction poses a significant financial burden and increasing consumption of health care resources among the elderly population (i.e., 65 years or older). The present article will touch upon current knowledge of diastolic dysfunction and its progression to diastolic heart failure in order to discuss current understanding of the following issues:

- Does non-invasively diagnosed diastolic dysfunction in an asymptomatic patient inevitably lead to development of diastolic heart failure? If so, what is the rate of progression?

- Can any intervention retard the progression to diastolic heart failure? If so, when should these interventions
be initiated?

**Diastolic dysfunction, in general**

Diastolic dysfunction of the left ventricle (LV) does not unequivocally equal diastolic heart failure. Diastolic dysfunction is a mechanical abnormality brought upon by a breakdown in the passive (compliance) and active (myocardial relaxation) intrinsic properties of the ventricle during diastole. Myocardial hypertrophy (e.g. left ventricular hypertrophy secondary to hypertension) and ischemic states (e.g. secondary to coronary artery disease) have been shown to impair the energy-dependant process of myocardial relaxation. The increased afterload in patient with aortic stenosis or hypertension can also inhibit myocardial relaxation by reducing the ability of the left ventricle to contract to small end-systolic volume, and hence limiting the ensuing elastic recoil's ability to enhance myocardial relaxation. Also, diastolic dysfunction can be secondary to pathological states that adversely affect the passive compliance during diastole, such as increase in myocardial wall thickness observed in concentric hypertrophy, as a result of longstanding hypertension, or in myocardial fibrosis seen in patients with myocardial interstitial pathology.

Diastolic dysfunction has been subdivided into three different grades of severity based on echocardiographic parameters of left ventricular compliance, relaxation rate, and filling pressures (Figure 1). Stage one is the mildest form of diastolic dysfunction with delayed relaxation defined by an early filling to late or atrial filling (E/A) ratio that is less than one, or $1 < E/A < 2$ with systolic to diastolic pulmonary venous (S/D) ratio $> 1$, and peak early diastolic myocardial velocity (Em) $< 8$ cm/sec or Em less than atrial component of the mitral annular tissue Doppler velocity (Am). Stage two of diastolic dysfunction is marked by a moderate level of dysfunction and defined by $E/A > 2$ and/or $= 2$ with $S/D < 1$ and Em $< 8$ cm/sec, or Em $< Am$, and often called pseudonormalization. Stage three is marked by restrictive filling and signifies severe diastolic dysfunction when the $E/A > 2$ with $S/D < 1$. Although hardly performed for evaluation of diastolic dysfunction alone, the most definitive diagnostic method is cardiac catheterization to determine elevated left ventricular end-diastolic pressure. Parameters of chamber stiffness are correlated with change in pressure to change in chamber volume; catheterization can therefore provide a direct measurement of end diastolic pressure. To our knowledge, there are currently no published studies that have directly tried to assess the duration or prognosis of the three separate forms of echocardiographically diagnosed diastolic dysfunction. Whether echocardiographically diagnosed diastolic dysfunction in fact results in progression of the disease to heart failure, has not yet been established.

**Left ventricular diastolic dysfunction**

In its simplest form, left ventricular diastolic dysfunction is defined as impairment in the capacity of the left ventricle to accept blood without a compensatory increase in left atrial pressure. Patients with left ventricular diastolic dysfunction tend to have elevated left ventricular diastolic pressure in the presence of normal or even reduced left ventricular volume, as the pressure-volume curve in these patients are shifted upwards. Over the years, a variety of comorbid conditions have been associated with development of left ventricular diastolic dysfunction, such as myocardial scarring, transmural myocardial infarction, chronic constrictive pericarditis, chronic coronary artery disease, dilated cardiomyopathy, hypertrophic

![Figure 1](image)

**Figure 1** Echocardiographic classification of diastolic dysfunction. Measurements of transmital flow velocity, E: peak early diastolic transmital flow velocity. A: peak late diastolic transmital flow velocity. A) Stage 1 represents delayed relaxation. B) Stage 2 represents pseudonormalization. C) Stage 3 represents restrictive filling
cardiomyopathy, diabetic cardiomyopathy, hypertension, aortic stenosis as well as normal aging. The underlying connection in the possible etiologies of left ventricular diastolic dysfunction is their ability to hinder one or both of the intrinsic diastolic properties of compliance or relaxation. Pathological states such as fibrosis and concentric hypertrophy can reduce compliance of the myocardium by increasing passive ventricular stiffness, thereby affecting the passive property of compliance in diastole. Ischemia and disease processes leading to increased afterload affect diastole by impairment of the active rate of relaxation.

**Left ventricular diastolic dysfunction and heart failure**

The prevalence as well as overall significance of diastolic heart failure has become distinctly apparent. Diastolic heart failure was originally reported in 1937 when Fishberg referred to it as “hypodiastolic failure”, a form of cardiac insufficiency secondary to inadequate filling of the left ventricle during diastole. A half a century later, Kessler became the first to discuss the clinical syndrome of diastolic heart failure. Over the years, a number of landmark publications have guided our current understanding for diagnosing diastolic heart failure. Recognizing the difficulty of non-invasive assessment of the LV diastolic function, in 2000, Vasan and Levy proposed a classification scheme for diagnosis of diastolic heart failure in hopes of reducing the difficulty for diagnosis of this rather prevalent pathology. According to the degree of diagnostic certainty, patients were partitioned into possible, probable, or definite diastolic heart failure. While keeping the need for evidence of heart failure for all categories, the diagnosis of probable or definite diastolic heart failure required evidence of normal left ventricular systolic function within three days of initial heart failure event. Most importantly it was argued that “evidence of abnormal LV relaxation, filling, diastolic distensibility, or diastolic stiffness” is required for a definite diagnosis of diastolic heart failure. Over recent years, Zile and colleagues have published several prospective studies concluding that the diagnosis of diastolic heart failure does not require objective recording of left ventricular diastolic dysfunction but only documentation of preserved systolic function. In two separate studies utilizing both Doppler echocardiography and cardiac catheterization, the authors observed a statistically significant percentage of patients with clinical diagnosis of heart failure and normal ejection fraction (EF > 45%) to be suffering from abnormalities in active relaxation or passive compliance.

The degree of involvement that left ventricular diastolic dysfunction plays in preserved ejection fraction heart failure is debatable and has been the major argument made by those that believe diastolic heart failure is the correct diagnosis for patients with heart failure and normal ejection fraction, given that these patients do not suffer from significant valvular, pericardial or pulmonary disease. Left ventricular diastolic dysfunction has also been found to be present in patients with heart failure and reduced ejection fraction, a form of heart failure that was originally believed to be mainly secondary to a systolic dysfunction pathophysiology.

**Clinical studies in patients with diastolic dysfunction**

In 1972 Gaasch and colleagues performed some of the first studies to evaluate the possible effects of left ventricular diastolic dysfunction. The authors described that left ventricular diastolic dysfunction has a negative impact on systolic function through its limitation of the Frank-Starling mechanism. Patients with conditions such as inappropriate hypertrophy have elevated left ventricular end diastolic pressure and decreased compliance, which affects the length-tension relationship by decreasing muscle fiber stretch at any given peak systolic stress. This might explain why decreased exercise tolerance is one of the first clinical symptoms associated with echocardiographically diagnosed diastolic dysfunction. Exercise tolerance in patients with left ventricular diastolic dysfunction that are asymptomatic at rest may be compromised secondarily to the inability to enhance diastolic filling by the degree necessary to increase the cardiac output during exercise without causing an abnormal elevation in left atrial pressure. Diastolic dysfunction has been found to be aggravated by exercise, especially with an increase in blood pressure. Recent studies have observed the development of left ventricular diastolic dysfunction in the presence of hypertension prior to the development of ventricular hypertrophy. Left ventricular diastolic dysfunction can therefore represent an early measure of myocardial end-organ damage prior to progression to heart failure, although further trials are needed to support this hypothesis.

The magnitude of asymptomatic left ventricular diastolic dysfunction in the general population is still unclear. In an attempt to determine the prevalence of preclinical diastolic dysfunction, Redfield et al performed a cross-sectional survey of 2,042 randomly selected residents over the age of 45 years in Olmsted County, Minnesota. The authors found the prevalence of asymptomatic echocardiographically diagnosed diastolic dysfunction to be 28.1% with increased prevalence seen in older age, diabetics, and in patients with cardiovascular disease (hypertension, coronary artery disease, cardiomyopathies).

A prospective trial in 206 patients with the clinical diagnosis of heart failure (New York Heart Association Grade II or higher) reported that-based on echocardiographic parameters-1% of 102 patients with EF=50% had some degree of diastolic dysfunction, 92% of 71 patients with EF<40% suffered from left ventricular diastolic dysfunction. Patients with reduced ejection fraction were more likely to have moderate to severe diastolic dysfunction in comparison to patients with preserved ejection fraction (27% vs. 62%,
respectively). In patients with heart failure with preserved EF, left ventricular diastolic dysfunction was accompanied by left ventricular hypertrophy, while in patients with heart failure and reduced EF, left ventricular diastolic dysfunction was associated with left ventricular dilation and marked systolic dysfunction. Overall prognosis and mortality appear to be significantly influenced by the degree of left ventricular diastolic dysfunction in heart failure patients, regardless of ejection fraction.26, 27

Clinical studies in patients with diastolic heart failure
The American College of Cardiology and American Heart Association (ACC/AHA) Task Force has previously stated that a definitive diagnosis can be made in heart failure patients with preserved EF if there exists a decreased rate of ventricular relaxation with elevated LV filling pressure, clarifying the need for coexistence of normal contractility (LV systolic function) and LV volume.28 In further assessing such assumptions, Zile and Lewinter.30 compared 75 patients with heart failure and normal ejection fraction with 75 patients without cardiovascular disease (controls). After analyzing both the echocardiographic parameters as well as catheterization data it was found that in patients with heart failure and normal ejection fraction, left ventricular systolic function, contractility and performance were intact, discounting contribution of left ventricular systolic dysfunction in those patients with presumed diastolic heart failure.29 In a review of data on left ventricular structure and function in heart failure patients with normal ejection fraction and hypertension, Zile et al.30 have argued that left ventricular end-diastolic volume is within normal range in patients with diastolic heart failure.

Right ventricular diastolic dysfunction
Similar to left ventricular diastolic dysfunction, there have been multiple etiologies associated with impairment in mechanical compliance as well as relaxation parameters that lead to right ventricular diastolic dysfunction. Over the years, right ventricular diastolic dysfunction has been observed in a variety of settings, including obesity, cystic fibrosis, chronic aortic stenosis, arterial hypertension and Chagas disease.31-38 Studies investigating the functional parameters of the right ventricle during diastole were slow to formulate due to the difficulty of correctly measuring right ventricular volume prior to the advent of Doppler echocardiography.36 The algorithm used for assessment and diagnosis of right ventricular diastolic dysfunction with Doppler echocardiography utilizes pulsed-wave Doppler of the transtricuspid flow, hepatic venous flow and tissue Doppler imaging of the tricuspid annulus or tricuspid annular velocity.37 Normal hepatic venous flow is defined as a ratio of systolic to diastolic velocities greater than one with the atrial wave reversal less than half the maximum systolic wave velocity.38 Mild right ventricular diastolic dysfunction is defined by E/A <1 in transtricuspid flow velocities, or 1 < E/A < 2 with S/D > 1 in hepatic vein flow and early component of the tricuspid annular tissue Doppler velocity (Et) less than atrial component of the tricuspid annular tissue Doppler velocity (At) or an atrial reversal wave more than half of the systolic wave of the hepatic vein flow. Moderate or severe right ventricular diastolic dysfunction can be assumed to be present if a reduced or inverted systolic waveform is present on the Doppler hepatic vein flow signal, respectively. Studies on pulmonary hypertension patients have led to the speculation that right ventricular diastolic dysfunction may be an independent factor contributing to right heart failure and death in patients with pulmonary hypertension.39

Gan et al.39 showed that in patients with pulmonary hypertension the increase in right ventricular afterload resulted in ventricular hypertrophy and right ventricular diastolic dysfunction. The degree of diastolic dysfunction correlated with the severity of pulmonary hypertension, which improved with medical therapy that reduced afterload. Right ventricular diastolic dysfunction in the setting of heart failure was first reported by Riggs in 1993.40 The author reported impaired right ventricular filling parameters in six children with dilated cardiomyopathy. Yu et al.41 published the first study that systematically assessed right ventricular diastolic dysfunction in 1996; comparing 114 patients with symptomatic heart failure (EF<50%) with 31 patients with pulmonary hypertension (pulmonary systolic artery pressure > 40mmHg) as well as 40 healthy subjects. The authors described a significant number of patients with systolic heart failure and/or pulmonary hypertension suffering from right ventricular diastolic dysfunction. Even after exclusion of patients with pulmonary hypertension, a statistically significant percentage of heart failure patients suffered right ventricular diastolic dysfunction. In their analysis of 105 patients with systolic heart failure, Yu and Sanderson42 demonstrated right ventricular diastolic dysfunction to be present in 21% of patients as assessed by echocardiography. Although a low powered study, the authors concluded that right ventricular diastolic dysfunction was an independent predictor for nonfatal hospital admissions for unstable angina or heart failure, even though it was not observed to be a prognostic factor for mortality, either alone or in combination with left ventricular diastolic dysfunction.

Right and left ventricular interaction in diastolic dysfunction
The French physician Bernheim43 was one of the first to report the concept of ventricular interdependence in 1910, noting that right ventricular performance can be compromised through compression of the right ventricle by a dilated or hypertrophied left ventricle. In 1956, Dexter44 explained a possible mechanism for diastolic interdependence. The “reverse Bernheim effect” hypothesized an increase in
right ventricular volume secondary to an atrial septal defect that can cause the septum to be displaced toward the left ventricular cavity and inhibit left ventricular filling mechanisms. A decade later in 1967, Taylor et al.\textsuperscript{46} reported that the distension of one ventricle during diastole can affect the compliance of the neighboring ventricle. The term diastolic ventricular interaction refers to the concept that compliance of one ventricle is influenced through a shared septum by the changes in volume, pressure, and/or compliance of the other ventricle.\textsuperscript{46} Although there are implications that diastolic ventricular interaction plays a role in exercise intolerance in patients with systolic heart failure, we currently do not have a great understanding of the possible role it may have in patients with diastolic heart failure. Ventricular interactions have been reported indirectly in patients with pathology of one ventricle and diastolic dysfunction of the neighboring ventricle. Right ventricular diastolic dysfunction has been observed in pathologic conditions that result in elevated left ventricular pressure such as systemic hypertension, aortic stenosis and hypertrophic cardiomyopathy.\textsuperscript{36, 47, 48} The reverse has also been reported in patients with elevated right ventricular volume or pressure with impaired left ventricular diastolic function.\textsuperscript{49} Furthermore, it has been suggested that right ventricular diastolic dysfunction observed in patients with heart failure but normal pulmonary artery pressures may be caused indirectly by coexistent left ventricular diastolic dysfunction secondary to ventricular interdependence.\textsuperscript{49} Although a realistic prospect, the possible role that diastolic ventricular interaction may play in the potential progression from diastolic dysfunction to clinical heart failure is currently not well established.

**Progression of diastolic heart failure**

In 2001, Aurigemma et al.\textsuperscript{50} published the possible rate of progression from asymptomatic diastolic dysfunction to clinical heart failure. The study analyzed 2,671 individuals without coronary heart disease, congestive heart failure, or atrial fibrillation. At baseline, 15% of the patients had diastolic dysfunction by echocardiography, with 170 participants eventually developing heart failure after a five-year follow up period (6.4%), concluding that echocardiographic findings can be suggestive of the development of heart failure. Despite arguments regarding exercise limitations and left ventricular diastolic dysfunction representing a possible early marker of myocardial damage, the rate of progression from diastolic dysfunction to diastolic heart failure remains uncertain (Figure 2).

Currently there are no large clinical trials assessing the possible progression from asymptomatic right ventricular diastolic dysfunction to clinical right ventricular failure.

**Guidelines and therapy for diastolic heart failure**

The difficulties in the diagnosis of diastolic heart fail-

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**Figure 2** Currently the rate of progression from the three different stages of echocardiographically diagnosed diastolic dysfunction in an asymptomatic patient to clinical diagnosis of heart failure has not been well studied. The possible effect of medical therapy or other comorbidities on this possible progression rate is not currently known.
ure have been partly responsible for the limited number of larger randomized controlled trials to guide treatment. In 1998, the European Study Group published one of the first widely analyzed guidelines for diagnosis of diastolic heart failure, stating the need for evidence of heart failure with normal systolic function (LVEF = 0.50) as well as evidence of abnormal filling, diastolic distensibility, LV relaxation or diastolic stiffness.\textsuperscript{30} The European Society of Cardiology recently published their latest guidelines for diagnosis of diastolic heart failure in 2007; providing specific guidelines on “How to diagnose heart failure with normal ejection fraction” and “How to exclude heart failure with normal ejection fraction”.\textsuperscript{52} The guidelines have three major criteria for diagnosing heart failure with normal ejection fraction: 1) signs/symptoms of heart failure, 2) normal or mildly reduced systolic function (EF > 50%) with a left ventricular end-diastolic volume index less than 97 ml/m\textsuperscript{2} and 3) evidence of left ventricular diastolic dysfunction. The diagnostic strategy provided in this set of guidelines allows for non-invasive methods of assessing for left ventricular diastolic dysfunction through tissue Doppler parameters (early mitral valve flow velocity to early tissue Doppler lengthening velocity (E/E’) > 15) and routine blood tests biomarkers (brain natriuretic peptide > 200 pg/mL) to play a role in situations when invasive hemodynamic measurements (LV end-diastolic pressure > 16 mmHg or mean pulmonary capillary wedge pressure > 12 mmHg) are not available.

Current treatment of diastolic heart failure has been aimed at controlling blood pressure and tachycardia, using diuretics to control pulmonary congestion and peripheral edema, and alleviation of myocardial ischemia.\textsuperscript{53} The ACC/AHA also recommend using beta-adrenergic blocking agents, angiotensin receptor blockers, angiotensin converting enzyme inhibitors and calcium antagonists in those patients with controlled blood pressure, and digitalis in order to control heart failure symptoms. In the latest update of the ACC/AHA practice guidelines for the diagnosis and management of chronic heart failure in the adult that comprise a document of 63 pages, the treatment of diastolic heart failure is summarized in less than one page.\textsuperscript{53}

Chinnaiyan et al.\textsuperscript{54} described the combined use of beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, calcium channel blockers, and spiranolactone as potential disease modifying therapy. The authors believe that the effects of these drugs improve diastolic dysfunction and diastolic heart failure by regression of left ventricular hypertrophy and decreased collagen content. They recommend these drugs to be utilized in both the setting of decompensated diastolic heart failure as well as for the chronic outpatient management of diastolic heart failure. In the recently published Hong Kong diastolic heart failure study 150 patients with heart failure and preserved ejection fraction were randomized to diuretics, ACE inhibitors or angiotensin 2 receptor blocker therapy. Only diuretic therapy reduced symptoms and improved quality of life during one year follow up.\textsuperscript{55}

Currently there are no large randomized clinical trials that have assessed the possible benefit of pharmacotherapy at different stages of noninvasively diagnosed diastolic dysfunction. Small trials have been carried out in an attempt to evaluate possible benefits of pharmacotherapy for patients with left ventricular diastolic dysfunction and decreased exercise tolerance. Warner et al.\textsuperscript{56} studied twenty patients with mild diastolic dysfunction diagnosed by Doppler echocardiography with a marked hypertensive response to exercise. The authors reported that using the angiotensin II receptor blocker losartan, resting blood pressure was unchanged but the hypertensive response to exercise was reduced (from a mean systolic blood pressure (SBP) of 226 mmHg to a mean SBP of 193 mmHg). Similar studies confirmed the benefits of angiotensin II receptor blockers on exercise tolerance by comparing their effects with calcium channel blockers (verapamil) or diuretics (hydrochlorothiazide). In two separate trials, Little et al.\textsuperscript{57,58} demonstrated that angiotensin II receptor blockers, calcium channel blockers, and diuretics all have the ability to blunt an increase in SBP during exercise in patients with asymptomatic left ventricular diastolic dysfunction, but only angiotensin II receptor blocker therapy increased exercise duration and improved quality of life, as assessed by questionnaires.

**Current issues of managing patients with diastolic heart failure**

Further research is needed to improve current knowledge of diastolic dysfunction and diastolic heart failure. No single echocardiographic index is established to classify the severity of diastolic dysfunction. There is no clear understanding of the natural course of diastolic dysfunction or diastolic heart failure. The effects of comorbidities such as hypertension, CAD or systolic dysfunction on progression of diastolic dysfunction and diastolic heart failure have not been established. Larger trials are required to assess progression from pure diastolic dysfunction-diagnosed by echocardiography-to the clinical diagnosis of (diastolic) heart failure.

Due to a lack of larger randomized trials, the management of diastolic heart failure is currently aimed at symptomatic management and control of physiologic factors known to affect ventricular relaxation. A timeline for initiation of treatment for diastolic dysfunction has yet to be defined. In light of the current evidence, it may prove beneficial to standardize echocardiographic diastolic parameters to determine severity of diastolic dysfunction, including Doppler tissue imaging, parameters denoting ventricular compliance, relaxation rate, and filling pressures, as well as ejection fraction. Furthermore, one may consider initiating pharmacotherapy once evidence of diastolic dysfunction has been established, regardless of symptoms. This ap-
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Conclusions

Diastolic dysfunction is a mechanical abnormality of the ventricle assessed by echocardiography. In contrast, diastolic heart failure is a clinical diagnosis, defined by signs and symptoms of (congestive) heart failure with documentation of preserved systolic function (LVEF=50%), in the absence of significant valvular, pericardial or pulmonary disease. Treatment of diastolic heart failure is aimed at management of symptoms and controlling factors that might exacerbate symptoms. The timeline and progression of diastolic dysfunction to diastolic heart failure has not been well established.

Further research is needed to 1) study progression of diastolic dysfunction, 2) develop a grading index of diastolic dysfunction and 3) evaluate the effects of pharmacotherapy on diastolic dysfunction in different populations.

References


