Echocardiography in heart failure: Beyond diagnosis

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It is a great honor for me to deliver this year’s Euro Echo Lecture in this beautiful city of Florence and I appreciate the European Association of Echocardiography for the invitation and this opportunity to discuss the role of echocardiography in systolic and diastolic heart failure as the single most useful test, not only in diagnosis, but also as a tool to provide insights into the pathophysiological mechanism of various etiologies of heart failure, as a tool to monitor patient’s response to various treatment options, and as a tool to help us develop innovative new therapies for heart failure.

According to the ESC guidelines published in 2005, heart failure is defined as “a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of ventricles to fill with or eject blood” which includes diastolic as well as systolic heart failure.1 ACC/AHA guidelines as well as ESC guidelines state that echocardiography is the single most useful test in the diagnosis of heart failure since symptoms of heart failure are not specific and more than a third of patients with a clinical diagnosis of heart failure may not actually have heart failure. It is also interesting to note from the SHAPE study that only 3% of more than 7000 subjects surveyed from nine European countries recognized breathlessness, fatigue, and edema as symptoms of heart failure.3 Moreover, even when someone presents with typical symptoms of heart failure, a diagnostic test, most frequently an echocardiography examination, is required to establish the underlying etiology for an optimal management strategy. In this lecture, I like to review with you how echocardiography is used for detection of structural abnormality, diagnosis and management of systolic heart failure, and evaluation of diastolic function in patients with symptoms of heart failure.

When a cardiac structural abnormality is responsible for patient’s heart failure, it is usually obvious on 2-dimensional echocardiogram. Fig. 1 illustrates an echocardiographic image of a middle age woman with increasing dyspnea. Parasternal long axis view demonstrated an enlarged right ventricle consistent with right ventricular volume or pressure overload which provides a list of differential diagnoses such as...
as severe tricuspid regurgitation, pulmonary hypertension, or intracardiac shunt to be investigated. A subcostal view subsequently showed a large secundum atrial septal defect with left-to-right shunt resulting in right ventricular volume overload. Ao = Aorta, LA = left atrium.

Figure 1  Parasternal long axis view from 54-year-old woman with increasing shortness of breath. The right ventricle (RV) is enlarged. In real time 2-D echocardiographic image, the left ventricle (LV) was of normal size and function. Subsequent views from subcostal window showed a large secundum atrial septal defect with left-to-right shunt resulting in right ventricular volume overload. Ao = Aorta, LA = left atrium.

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Heart failure due to systolic dysfunction is relatively easy to diagnose by echocardiography which demonstrates a dilated left ventricle with a reduced ejection fraction. In systolic heart failure, however, echocardiography has many other roles beyond the recognition of systolic heart failure since dilatation of the LV results in alteration of intracardiac geometry and hemodynamics leading to increased morbidity and mortality. New medication strategies, devices, and surgical therapies are being developed to manage systolic heart failure. Echocardiography has been essential in identifying structural and hemodynamic abnormalities associated with systolic heart failure and to monitor patient’s response to new and novel therapies. It has been well documented for the last 20 years that the left ventricular volume is one of the most important prognostic indicators for the patients with ischemic or dilated cardiomyopathy.4,5 White and his associates demonstrated that worsening of patient’s survival was associated with increased end systolic volume after an acute myocardial infarction. With LV remodeling and a progressive dilatation of the heart, although initially an adaptive process to the myocardial damage, the left ventricle becomes more spherical, and the mitral annulus is dilated with apical displacement of the mitral leaflets causing functional mitral valve regurgitation.6–8 A combination of LV dilatation and mitral valve regurgitation results in increased filling pressure, symptoms of heart failure, and death. Grayburn and his collaborators in the BEST trial demonstrated that among various echocardiography parameters, LV end-diastolic volume, deceleration time of early diastolic mitral inflow velocity which is a surrogate for pulmonary capillary wedge pressure, and severity of mitral valve regurgitation were the strongest predictors of survival in patients with ejection fraction of 35% or less5 (Fig. 2). Since a smaller LV with less amount of mitral regurgitation is associated with a better functional capacity and survival, there are many management attempts to reduce the left ventricular volume and MR with the use of surgical, pacing, and other percutaneous devices, as well as medications and surgical revascularization. In this context, echocardiography is a valuable tool since it can measure or assess LV dimensions, volumes, sphericity index, severity of mitral regurgitation, diastolic filling parameters, and pulmonary artery systolic pressure.
SAVE trial was one of the first studies where echocardiography was used in a systolic heart failure trial to show that adverse cardiac events were associated with increased left ventricular size after acute myocardial infarction. Echocardiography has been used to assess the success of the device therapy by documenting smaller LV volume, increased LVEF, increased dP/dT, and less amount of mitral regurgitation which are all signs of reverse remodeling. Another potentially critical role of echocardiography in biventricular pacing is to identify the patients who respond positively to the therapy since 30-50% of patients who receive biventricular pacing may not improve. Currently, mechanical dyssynchrony measures obtained by tissue Doppler imaging velocities are considered to be best in that role. Time intervals from the onset of QRS to the time of peak systolic velocity of 2 to 12 segments of LV are used to determine the maximum or standard deviation interval. The maximum interval of 100 msec or standard deviation interval of 34 msec were found to have the highest predictive power for identifying positive responder. However, we have found that such intervals by TDI are present in normal subjects and patients without reduced ejection fraction or conduction delay. Instead, time intervals from the QRS to the peak negative strain from 12 segments correlated best with LV remodeling parameters and hemodynamic improvement after cardiac resynchronization therapy. It is, however, time consuming and challenging to obtain tissue Doppler or strain imaging from 12 segments. Further clinical experience and simplification of current strain imaging procedure are required to use echocardiography in identification of positive responders to CRT routinely in clinical practice.

Another innovative device for reducing LV volume or achieving reverse LV remodeling is a passive constraint device called ACORN. Mayo Clinic Echocardiography Core Lab has been working for the US clinical trial of this device which was recently completed. I would like also to acknowledge that many European centers were the initial clinical sites for development and testing the safety of this device. There was a progressive and significant reduction in left ventricular volume measured by echocardiography in patients treated with this device compared to a control group. The left ventricular volume reduction was associated with significant reduction in major cardiac procedures including cardiac transplantation.

Surgical Ventricular Restoration or Reconstruction (SVR) procedure which was pioneered by Dr. Dor in 1984 is a surgical attempt to restore LV size and shape by excluding the scarred and akinetic apical segments. The results from SVR procedure in more than 1000 patients were reported with an overall 30 day mortality of 5.3%. LVEF improved from 29% to 39% and LV end-systolic volume index decreased from 80 mL/m² to 56 mL/m². Patients with a larger ventricle and worse functional class preoperatively had worse outcome after SVR. However, this report did not come from a randomized trial, and there is no way to compare this result with the intensive medical therapy, other device therapy, or revascularization therapy alone in patients with reduced ejection fraction and akinetic apex. If SVR procedure has an incremental beneficial effect, echocardiography should be helpful in identifying ideal patients with the large left ventricle, akinetic scared apex, reduced LVEF as well as normally functioning inferior-lateral wall. Structural and hemodynamic response to the SVR can be readily assessed by comprehensive echocardiogram. However, the long-term structural and functional result of SVR procedure is not
yet clearly understood and further clinical trial is required to demonstrate beneficial effects of SVR compared to other device and medical/surgical therapy. Also, the long axis dimension becomes shorter by SVR, but the short axis dimension and mitral annulus may continue to dilate in some patients which may lead to more severe mitral valve regurgitation and higher diastolic filling pressure. Hence, echocardiography should be able to provide an insight and understanding of the long-term structural and hemodynamic outcome of the SVR.

In patients with severe coronary artery disease, reduced LVEF, and heart failure, there has not been a clinical trial data to guide our treatment. Is surgical revascularization better than medical therapy? What if the patient has akinetic apex? Does additional SVR procedure improve functional class and survival of the patients with ischemic cardiomyopathy? These important clinical questions are being investigated by STICH (Surgical Treatment In patients with Coronary artery disease and Heart failure) trial in which patients with ischemic cardiomyopathy and revascularizable coronary disease are randomized to intensive medical therapy, coronary artery bypass surgery, and bypass plus SVR. The first phase of STICH trial of comparing two surgical strategies was completed in the early 2006 with 1000 patients enrolled. The comparison of medical therapy versus surgical therapy will be completed in early 2007. STICH trial is truly an international trial with many European clinical centers participating. In fact, STICH investigators in Poland has enrolled the largest number of patients in the study and should be congratulated on their fantastic effort. This study will provide a very rich imaging data since all patients in the trial have baseline, four month, and two year follow-up echocardiograms and selected patients will have cardiac magnetic resonance imaging and nuclear studies. Mayo Clinic Echo Core Lab is analyzing all echocardiography data for the STICH to address the following two aims; 1) To determine if the use of echocardiography at baseline to assess LV systolic and diastolic dysfunction, cardiac hemodynamics, and valvular regurgitation in the STICH Trial population can identify a subgroup that derives a substantial benefit or incurs a significant harm from surgical intervention as determined by all-cause mortality. 2) To assess the effects of the different randomized treatment strategies on echocardiographically-derived parameters of cardiac systolic and diastolic dysfunction, hemodynamics, and the severity of valvular regurgitation at 4 months and 2 years as compared to baseline as they relate to changes in HF functional class and the clinical endpoints of death and HF hospitalization. Echocardiography data from 2000 patients from all over the world with ischemic cardiomyopathy will be analyzed independently and correlated with clinical and other laboratory data. We are looking forward to sharing many interesting results from this rich STICH echocardiographic data.

Now, I like to turn my discussion to assessment of diastolic function in patients who present with symptoms of heart failure. It has been well established that about 50% of patients with new onset of heart failure do have a normal ejection fraction. Some have suggested that this condition be called heart failure with preserved ejection fraction (HFPEF) since intrinsic diastolic dysfunction may not be a unifying underlying mechanism for heart failure. However, the terminology is also troublesome since there are many conditions other than a myocardial disease which can cause heart failure with preserved ejection fraction such as constrictive pericarditis, severe valvular heart disease, congenital heart disease, and intracardiac tumor. Since the most common etiology for heart failure with preserved ejection fraction and no other structural abnormalities is due to diastolic dysfunction, diastolic heart failure appears to be a better terminology to describe this disease entity. This condition is often difficult to be diagnosed by 2-D echocardiography alone although there are diagnostic clues in two-dimensional echocardiography if carefully looked for. Compared to a normal heart, the heart with diastolic dysfunction has less vigorous mitral annulus motion during systole and early diastole corresponding to the period of active myocardial relaxation, the left ventricular wall tends to be thicker than normal, and the left atrial size is increased. These abnormalities suggest that the patient’s diastolic dysfunction is abnormal and a more detailed Doppler assessment is required. Doppler evaluation of diastolic function requires the following: Early (E) and late (A) diastolic velocities of the mitral inflow, Deceleration time (DT) of mitral E velocity, early diastolic mitral annulus velocity (Ea), pulmonary vein velocities, hepatic vein velocities, and mitral inflow propagation velocity obtained by color M-mode (Fig. 3). It usually begins with recording of mitral inflow velocities which reflect the relationship between LA and LV pressures. This relationship, hence the configuration of mitral inflow velocities, alters predictably with impaired myocardial relaxation and increased filling pressure. Mitral inflow velocity pattern of normal subjects is characterized by predominant early diastolic filling with DT of 200 ± 40 msec. Impaired myocardial relaxation which is usually an
initial diastolic abnormality decreases E velocity, increases A velocity, and lengthens DT. On the other hand, increased filling pressure has an opposite effect on these Doppler parameters. Therefore, during an early stage of diastolic dysfunction with impaired myocardial relaxation and normal filling pressure, mitral inflow velocity recording alone is usually sufficient for diastolic function assessment. As filling pressure increases with worsening of diastolic function, mitral E velocity increases with shortened DT and A velocity decreases with shorter flow duration. When diastolic dysfunction is far advanced and/or filling pressure is markedly elevated, mitral inflow velocity is again sufficient. However, mitral inflow velocity pattern may appear normal (so called pseudonormalized mitral inflow) in patients with moderate elevation of filling pressure. Even in this situation, a careful attention to isovolumic relaxation time and atrial flow duration is helpful in distinguishing pseudonormalized from truly normal mitral inflow velocities. It is important to keep in mind that pseudonormalized mitral inflow is a product of impaired myocardial relaxation and moderate elevation of filling pressure. Even in this situation, a careful attention to isovolumic relaxation time and atrial flow duration is helpful in distinguishing pseudonormalized from truly normal mitral inflow velocities. It is important to keep in mind that pseudonormalized mitral inflow is a product of impaired myocardial relaxation and moderate elevation of filling pressure. In this context, Doppler parameter to evaluate myocardial relaxation is critical in our evaluation of diastolic function. Tissue Doppler Imaging allows recording of myocardial tissue velocities and it turned out that early diastolic velocity of the mitral annulus (Ea) correlates well with tau or the status of LV myocardial relaxation.\textsuperscript{23,24} Ea is lower at the septal annulus (normal $\geq 10$ cm/sec) compared to the lateral annulus (normal $\geq 15$ cm/sec) and at our institution we use the septal annulus in all patients except when there is regional wall motion abnormality in the basal inferior septum. Pulmonary vein, hepatic vein, and the color M-mode of mitral inflow may be beneficial in certain cases to evaluate diastolic function and estimated filling pressures. Pulmonary venous flow velocity shows progressive reduction of systolic forward flow velocity compared to the diastolic flow velocity with increased filling pressure (Fig. 4). In all phases of diastolic dysfunction, early diastolic velocity of the mitral annulus (Ea) is reduced and it does not increase with high filling pressure. Since mitral E velocity increases with a higher filling pressure and tissue Doppler Ea remains reduced, the ratio of the mitral E velocity and the mitral annulus velocity of Ea (E/Ea) has a good correlation with the pulmonary capillary wedge pressure or left ventricular filling pressure.\textsuperscript{25,26} E/Ea ratio $>15$ usually indicates pulmonary capillary wedge pressure of greater than 20 mmHg when septal Ea is used; and if the ratio $<8$, the filling pressure is usually normal and for the ratio between 8 and 15, we may require further diastolic parameters to estimate diastolic filling pressures.\textsuperscript{25} As mentioned earlier, motion or velocity of the mitral annulus can also be appreciated from parasternal long axis and apical four chamber views of 2-dimensional echocardiography. Assessment of filling pressure by 2-dimensional and Doppler echocardiography is useful in patients with systolic dysfunction as well as in patients with normal LVEF. Numerous studies have demonstrated a good prognostic value of Doppler echocardiographic assessment of filling pressures. We also have demonstrated that E/Ea $>15$ was one of the strongest predictors for survival after acute myocardial infarction whether LVEF was normal or abnormal.\textsuperscript{27,28} This work of retrospective review was carried out by G. Hillis who was a research fellow from the United Kingdom, and he has led a prospective study upon return to his University of assessing prognostic power of clinical, laboratory, and echocardiographic parameters in patients with acute myocardial infarction. His group presented at the last AHA meeting that Plasma BNP, LVEF, and septal E/Ea were strong independent predictors of morbidity following acute myocardial infarction. Of them, septal E/Ea ratio was found to be the single most powerful predictor of death.\textsuperscript{29} In our retrospective review, LA volume was found to be also prognostic for death after acute MI.\textsuperscript{30} Therefore, estimation of filling pressure should also be an integral part of echocardiographic
evaluation in patients with reduced LVEF. Let me then share with you how echocardiographic evaluation of diastolic function should be used in patients with symptoms of heart failure and normal LVEF. I will start with an example of a 70-year-old man who presented with sudden onset of dyspnea to the emergency room. Chest X-ray suggested pulmonary edema, but serum BNP was within normal limits, and the echocardiography showed normal ejection fraction and mild increase in LV wall thickness. Because of the normal BNP and ejection fraction, the patient’s clinical and chest X-ray findings
were thought to be of non-cardiac etiology initially. However, when his echocardiogram was reviewed more carefully, it was obvious that LV myocardial relaxation was markedly impaired (mitral annulus motion was reduced) and diastolic evaluation showed E/Ea ratio was 22, much higher than the 15 cutoff value. Subsequent evaluation showed a critical left main coronary artery disease and LV end diastolic pressure of 28 mmHg. When plasma BNP and mitral E/Ea ratio were correlated with pulmonary capillary wedge pressure, correlation was better for the mitral E/Ea ratio compared to the BNP. BNP has been shown to be helpful in triaging patients with dyspnea in the emergency room, but we should be mindful that the sensitivity and the specificity of BNP for increased filling pressure is less than optimal, and probably lower than echocardiographic estimation of filling pressure. Even when high filling pressure is detected by BNP in patients with heart failure, echocardiography will be necessary to establish an etiology and to provide an optimal treatment. In patients with heart failure and normal LVEF, optimal therapy has not been established although there are several studies demonstrating some benefits in this group of patients. I anticipate several large clinical trials to identify the most efficacious management strategy in diastolic heart failure and echocardiography will be an integral part of those trials for identification of ideal patients and monitoring the response of various therapies.

Constrictive pericarditis is an elusive entity which causes heart failure in patients with normal LVEF and is potentially curable. Its hemodynamic manifestations are similar to those of myocardial diseases and it is often challenging to differentiate constrictive pericarditis from restrictive myopathy or other myocardial diseases. With comprehensive echocardiography, we now should be able to diagnose constrictive pericarditis in almost all cases non-invasively. There are two unique features of constriction which are not present in myocardial diseases: 1. Dissociation between intrathoracic and intracardiac pressures; and 2. Exaggerated interventricular dependence. These two features in constriction result in characteristic diastolic filling pattern which can be reliably recorded by Doppler echocardiography. Typically, mitral inflow early diastolic velocity decreases with inspiration and hepatic vein diastolic reversal flow increases with expiration. However, the respiratory variation of mitral inflow velocities may not be present in some patients. Tissue Doppler recording of mitral annulus velocity has added an important diagnostic value in constrictive pericarditis since Ea velocity which is reduced in almost all myocardial diseases is increased in constrictive pericarditis (Fig. 5). Other subtle findings such as ventricular septal motion changes with respiration and septal shudder in constriction can be appreciated from careful evaluation of 2-D echocardiography. At our institution, more careful attention to these echocardiographic features of constrictive pericarditis allowed increased detection of this entity in patients with heart failure which has been partly responsible for higher number of pericardiectomies (from 15 in 1990 to more than 60 in 2005). More reliable detection of constrictive pericarditis has been a valuable product of assessing diastolic function by 2-D, Doppler, and myocardial tissue imaging.

Let me summarize what I have discussed so far regarding the role of echocardiography in patients with symptoms of heart failure. When heart failure is clinically suspected, echocardiography is the single most important diagnostic imaging technique. In a half of patients with a final diagnosis of heart failure, transthoracic and/or transesophageal echocardiography demonstrates a major structural and/or functional abnormality such as in systolic heart failure, severe valvular heart disease, congenital heart disease, or cardiomyopathies. It is more difficult to diagnose heart failure when there are no obvious structural and functional abnormalities in 2-dimensional echocardiography. In the other half of patients with heart failure, most likely diastolic heart failure, there may be subtle abnormalities in 2-dimensional echocardiography, but a firm diagnosis of diastolic heart failure requires Doppler and myocardial (or tissue Doppler) imaging. If mitral inflow velocity demonstrates clearly a restrictive filling pattern and/or high filling pressure at resting stage, the diagnosis of diastolic heart failure or constrictive pericarditis is usually secured. Tissue Doppler imaging of the septal mitral annulus is helpful in differentiating pseudonormal from true normal mitral inflow velocities, and differentiating constrictive pericarditis from myocardial diseases. It is more problematic when diastolic function evaluation shows relatively normal filling pressure or predominantly impaired myocardial relaxation at rest. In this situation, right heart failure, pulmonary process, and non-cardiopulmonary process should be considered. Another important condition we need to consider is exercise-induced increased filling pressure responsible for patient’s symptoms of shortness of breath with exertion. Therefore, we need to assess filling pressure with exercise as well as at resting stage.

The mitral annulus Ea velocity which reflects the status of mitral relaxation does actually increases...
with exercise or increased preload when myocardial relaxation is normal, but Ea velocity does not augment as much with higher transmural pressure gradient or with exercise when relaxation is impaired with increased tau. Therefore, we hypothesized several years ago that in normal subjects mitral inflow E velocity and mitral annulus Ea velocity increase proportionately with a result that E/Ea remains less than 15 at resting stage and exercise.\textsuperscript{36,37} However, in patients with diastolic dysfunction and exertional dyspnea, E velocity increases with exercise but the Ea velocity remains reduced or increases less with exercise. Therefore, E/Ea ratio increases with exercise compared with the resting stage in patients with diastolic dysfunction. We used a supine bike or treadmill exercise test for assessment of filling pressure with exercise, and we analyzed diastolic parameters of mitral inflow and mitral annulus velocities which provided E/Ea ratio. Initially we performed diastolic stress test in healthy individuals. This work was initially carried out by Dr. F. Lulic from Croatia, and then completed by Dr. J. Ha from Korea working in our laboratory.\textsuperscript{36,37} It was found that in normal subjects, mitral E velocity increased about 25% with exercise from baseline data and deceleration time decreased an average of 16 msec (from 192 to 176 msec) and Ea velocity increased also about 25%. Therefore, E/Ea ratio remained the same (mean of 6.6) at baseline and

**Figure 5** Three patients with a similar degree of shortness of breath. Upper panel shows the mitral inflow velocity. Middle panel shows tissue Doppler velocity from the mitral septal annulus. The bottom row shows color M-mode of the mitral inflow velocity. The left column is from a normal subject. Middle column from a patient with a myocardial disease (restrictive cardiomyopathy). The right column is from a patient with constrictive pericarditis. The mitral inflow velocity appeared the same in all three examples with E/A ratio of 2. However, early diastolic velocity of the mitral annulus is markedly reduced (5 cm/sec) in the patient with myocardial disease (in the center column) whereas early diastolic velocity (E') of the mitral annulus is normal in normal subject and exaggerated in the patient with constrictive pericarditis (right column, middle row). Color M-mode of mitral inflow demonstrates normal flow propagation in the normal subject and the patient with pericardial disease. However, it is usually reduced (in the center column, bottom row, left color M-mode) in patients with myocardial disease but may be falsely normal when LV cavity is relatively small such as in hypertrophic cardiomyopathy for early stage of cardiac amyloid (in the center column, bottom row, right color M-mode).
exercise in individuals with normal diastolic function. Subsequently, we performed supine bike diastology stress test in patients with exertional dyspnea and were able to identify three groups of patients. The first group of patients was found to have E/Ea >15 (increased filling pressure) at rest and the ratio remained elevated with exercise in all. The second group of patients had relatively normal filling pressure and impaired myocardial relaxation pattern on mitral inflow velocity at rest which turned to a restrictive filling pattern with E/Ea >15 with exercise (Fig. 6). The third group of patients had normal filling pressure and E/Ea <15 at rest and with exercise. The patients with increased filling pressure with exercise whether or not they had normal filling pressure at rest (the first and second groups), had shorter exercise duration compared to the patients (the third group) with normal filling pressure at rest and with exercise. Subsequently, we have performed simultaneous cardiac catheterization and Doppler echocardiography in patients who were undergoing exercise testing in the cardiac catheterization laboratory for an evaluation of exertional dyspnea. In this patient population, E/Ea >15 correlated well with pulmonary capillary wedge pressure >20 mmHg, not only at resting stage but also with exercise. Therefore, we believe that we now have a very reliable non-invasive diagnostic technique to estimate diastolic filling pressures both at resting stage, and with exercise. Marwick and his group have also demonstrated a similarly good correlation between E/Ea and PCWP at rest and exercise. Several investigators also have explored use of the duration between the onset of mitral E velocity and the onset of mitral annulus Ea velocity. This duration or time interval has been reasonably well correlated with the filling pressure and also the status of myocardial relaxation. I had an opportunity to write an editorial to summarize the current status of using diastolic parameters to estimate diastolic filling pressures, and I concluded in my editorial from numerous investigations in this area that "now echocardiography is able to estimate left ventricular filling pressure under various clinical conditions". When I submitted this editorial, the Journal suggested that the phrase, "now echocardiography is able," be changed to "echocardiography is now able to" which I did not think was a big issue. I agreed to the change, but when the paper was published, I was horrified to see my conclusion. Instead of "echocardiography is now able to estimate left ventricular filling pressures", it read "echocardiography is not able to estimate left ventricular filling pressures" which completely changed the conclusion of my editorial, but I hope that some of you who read the editorial were able to see the obvious error.

Ladies and gentleman, I tried to summarize for you today in my lecture the current clinical use of echocardiography in patients with heart failure, not only systolic but also diastolic heart failure.

**Figure 6** (Left) Baseline mitral inflow (top) and tissue Doppler (bottom) demonstrate grade 1 diastolic dysfunction (E velocity of 50 cm/sec and deceleration time of 250 msec) with impaired myocardial relaxation but normal filling pressure with E/E' ratio of 7, and tricuspid regurgitation velocity was also normal indicating normal pulmonary artery systolic pressure. (Right) With 50 watts of supine bike exercise, E velocity increased to 85 cm/sec and deceleration time shortened from 250 msec to 140 msec. E' velocity, however, increased slightly from 7 to 8 cm/sec. Therefore, E/E' ratio increased from 7 to 11. TR velocity increased from 2.4 m/sec to 3.8 m/sec (Reproduced with permission from Ref. 37).
There are many exciting new technologies and applications of echocardiography such as 3-dimensional echocardiography, strain imaging, and myocardial perfusion imaging. I am certain that these new applications will strengthen and improve the role of echocardiography in general, but more so in patients with symptoms of heart failure. Undoubtedly, echocardiography is the single most useful test in patients with symptoms of heart failure. It is essential in the diagnosis and identification of underlying etiology of heart failure. However, we should move beyond the concept of echocardiography as only a diagnostic tool in patients with heart failure, hence, use this versatile and easily available technology to gain pathophysiologic insights of various forms of heart failure, to help identify or establish an optimal therapy for the patients with heart failure, to monitor treatment response, to prognosticate, and to be an important tool in clinical heart failure trials.

Thanks again for the invitation to Euro Echo Lecture 2005 and thank you all for listening.

References


