



Role of Echocardiography in Congestive Heart Failure

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INTRODUCTION

Heart failure is a growing and increasingly important chronic disease in the world, occurring in at least 2% of the adult population and rising to 3% in those aged over 75 years. In a recently conducted study in Harrow, of 1400 subjects who were invited to undergo echocardiography for assessment of left ventricular (LV) function, the overall prevalence of symptomatic and asymptomatic LV dysfunction was 2% and rising to 8% above the age of 65 years. Although the incidence of most cardiovascular diseases has declined over the past 20 years, the incidence of heart failure has continued to rise, due to the fact that more people are surviving after acute myocardial infarction and also to the increasing number of elderly people. A diagnosis of heart failure is associated with high mortality, morbidity, and cost. Heart failure costs the USA over \$8 billion (£5 billion) each year and 5% of all admissions in the UK have a diagnosis of heart failure. Indeed, hospital admission accounts for 70% of the cost of heart failure due to the number of day beds that are occupied.

Early detection of heart failure caused by left ventricular systolic dysfunction (LVSD) is important as early initiation of drug treatment, including angiotensin converting enzyme inhibitors, β blockers, and aldosterone receptor antagonists, has been shown to reduce mortality, morbidity, and hospitalisation. Indeed, it has been shown that early initiation of treatment in asymptomatic left ventricular dysfunction will prevent or retard progression to heart failure. However, the key to reduction of mortality, morbidity, and cost of heart failure is accurate and early diagnosis of LVSD. Unfortunately, heart failure is difficult to diagnose accurately on clinical grounds. The prevalence of heart failure continues to increase and it remains a major public health threat, particularly in the elderly. The overall annual healthcare expenditure for heart failure continues to increase. While a new diagnosis of heart failure is associated with substantial risk of death within one year, the institution of appropriately guided pharmacologic treatment has led to substantial reductions in cardiovascular mortality. Identification of potential candidates for such treatment can be facilitated through use of echocardiography.

In many patients the diagnosis of a cardiomyopathy is made after the onset of heart failure symptoms, atrial or ventricular arrhythmias, or a stroke. These complications of the underlying cardiomyopathy represent major causes of cardiovascular morbidity and mortality and frequently result in referral for

echocardiography. In addition, specific echocardiographic features allow the clinician to determine more accurately the aetiology of the cardiomyopathy. Integration of clinical and echocardiographic features now allows for a better assessment of both immediate risk and long term prognosis in patients with a cardiomyopathy and heart failure.

ROLE OF ECHOCARDIOGRAPHY

Despite the availability of many cardiac imaging techniques, echocardiography is currently the most widely used and cost effective diagnostic imaging test to assess LVSD. On the basis of this, echocardiography has been proposed to be the screening method of choice.

Over a period of about 25 years, echocardiography has evolved from a tool, which saw bizarre shadows into a technique wherein pathophysiological diagnosis can be established with a reasonable certainty and accuracy. This technique continues to evolve and provides one of the most convenient, quick, easy to use, learn and interpret, portable cost effective tool available to us in the management of patients with CHF.

The current role of echocardiography in heart failure encompasses the following-

1. Diagnosis of heart failure
2. Pathophysiology of heart failure and assessment of hemodynamic derangements on the cardiovascular system
3. Etiology of heart failure
4. Guide to therapeutic interventions in heart failure
5. Assessment of therapeutic interventions in heart failure
6. Assessment of prognosis in patients with heart failure

Diagnosis of heart failure

Heart failure is commonly misdiagnosed despite symptomatology, mainly because of the non-specificity of the clinical symptoms and the non-sensitivity of the clinical signs. Not surprisingly, the validity of a clinical diagnosis of heart failure in primary care is poor. Rates of misdiagnosis when patients are assessed against objective criteria range from 25-50%. In another series only 26% of patients with suspected heart failure had a diagnosis of LVSD. Caruana and colleagues reported that of the 159 patients referred to hospital with suspected heart failure, 109 had preserved LV function of which only seven did not have an alternative explanation for their significance of heart failure.

Table 1 : Echo-based measurements

2D measurements
1. Ventricular dimensions and volumes in systole and diastole
2. Atrial volumes and dimensions
3. Ventricular wall thickness and mass
4. Ventricular fractional shortening and EF
5. Regional wall motion and thickening
Doppler measurements
1. Transvalvar velocities and pressure gradients
2. Transvenous velocities and pressure gradients
3. Effect of various physiological maneuvers on the above parameters
Derived measurements
1. Pulmonary artery pressures
2. LA and RA pressures
3. LV and RV end-diastolic pressures
4. Cardiac output/ Cardiac Index/ Cardiac Power

The clinical symptoms of heart failure- dyspnoea on effort, pedal edema etc are nonspecific and it remains difficult to diagnose clinical CHF on those grounds alone. The differential diagnoses of these symptoms are listed in table xyz. From the differentials, it is amply clear that symptoms are not reliable. Clinical signs of heart failure are insensitive too- for e.g. in 14507 patients in the CASS registry, a third heart sound or pulmonary rales had a respective sensitivity of 9% and 5% for the detection of significant LV dysfunction on contrast ventriculography. Only masking of signs and symptoms causes a part of this low sensitivity by therapy.

Thus, to establish positively a diagnosis of heart failure, patients must be referred for cardiac imaging. Are there alternatives to cardiac imaging in primary care for the diagnosis of left ventricular dysfunction? Although a completely normal ECG or even a narrow QRS complex ECG usually excludes left ventricular dysfunction, an abnormal ECG has a low predictive value for LVSD. In addition to this, ECG changes may be subtle and may not be recognized by primary care physicians.

Also, as our understanding of CHF has increased, we now know that a significant number of patients with clinical CHF, in fact do not have systolic heart failure but diastolic heart failure. In as many as 35-40% of men and 65-70% of women with CHF, LV function may be preserved (LVEF > 50%). Recent studies suggest that diastolic dysfunction is a major cause of CHF. Diastolic function predicts prognosis of patients with CHF independently of the degree of LV systolic dysfunction, and is therefore important to clinicians. In these patients, the conventional clinical signs maybe misleading and the ECG may be completely normal. This confuses the issue even further.

All of these factors ultimately mean that referral for cardiac imaging is still required for the majority of patients. Echocardiography is a powerful tool for assessing systolic and diastolic heart failure, the specific etiology of the cardiomyopathy, assessing response to treatment, and categorising prognosis. It is non-invasive, relatively low cost, does not expose patients to ionising radiation, and serial studies can be done at the bedside. Thus echocardiography continues to have advantages over competing technologies.

Table 2 : Etiology of Heart Failure

Conditions leading to CHF
- Valvular Heart Disease
- Ischemic Heart Disease
- Cardiomyopathies
- Dilated
- Hypertrophic
- Restrictive
- ARVD
- Pericardial Diseases
- Constrictive Pericarditis
- Pericardial Effusion with or without tamponade
- Intracardiac Tumours
- Diastolic Dysfunction
- Congenital Heart Disease

Pathophysiology of heart failure and assessment of hemodynamic derangements

Use of 2-D echo and Color Doppler echocardiography has enabled us to assess the pathophysiologic basis of heart failure. It gives us a unique non-invasive bedside assessment of cardiovascular hemodynamics and helps assess the severity of hemodynamic derangement and heart failure. Combining stress testing (exercise or pharmacological) along with echocardiography provides an assessment of myocardial viability and myocardial reserve or contractility. It a useful tool in the elucidation of the mechanism and assessment of CHF symptoms in a given patient e.g. exercise induced or aggravation of MR, ischemia or elevation in PA pressure.

Some of the useful echo-based measurements which help us elucidate the above are given in Table 1.

These parameters help us in differentiating systolic versus diastolic heart failure; between various types of cardiomyopathic heart failure viz. dilated vs. hypertrophic vs. restrictive; between restrictive and constrictive diseases of the heart; between ischemic and non-ischemic causes of cardiomyopathic heart failure, between high output and low output heart failure and between right sided and left sided heart failure. These differences are extremely important to the clinician in deciding the type of therapeutic intervention to be undertaken and in prognostication.

Etiology of heart failure

The various generic conditions where patients present with symptoms suggestive of heart failure are listed in Table 2.

Valvular Heart Disease

Despite the fact that in the Western world there is a declining trend of rheumatic heart disease, we in India still continue to see fresh cases of this disease. Echocardiography has almost replaced all modalities in the diagnosis of valvular heart disease. Use of echocardiography provides information on the number of cardiac valves involved, helps in assessing the severity of each valvar lesion individually and their effect and contribution to ventricular function and symptoms of heart failure, provides a method to differentiate rheumatic from non-rheumatic etiology of valvar disease and planning for therapy.

Significant valvar disease is sometimes missed clinically, particularly mitral stenosis (occult or silent MS). This may happen in cases with severely calcified mitral valves, with severe PAH or RV dilation. Aortic stenosis is not always easy to grade accurately in the elderly especially with LV dysfunction and heart failure and in the presence of other stenotic valvar lesions. Murmurs of acute and subacute mitral and aortic regurgitation causing CHF are notoriously difficult to hear and assessment of severity is extremely difficult. Echocardiography provides a rapid and accurate bedside assessment of these conditions and may make a difference between life and death in these emergent conditions.

Ischemic heart disease

Ischemic heart disease is the leading cause of congestive heart failure worldwide. The pathophysiology and proximate cause for congestive failure in a patient with ischemic heart disease is varied. Ischemia can cause heart failure by causing both systolic and/ or diastolic dysfunction. Ischemia leading to myocardial infarction results in damage to the heart muscle and subsequent systolic heart failure due to reduced muscle mass and/or adverse re-modelling of the remainder of the ventricle. Repeated episodes of ischemia, short of infarction, may result in stunning and hibernation of the myocardium which then results in large areas of mechanically non-functional myocardium and heart failure. This can be seen on echocardiography as abnormalities of wall motion and loss of diastolic myocardial wall thickness and reduced or absent systolic myocardial thickening. Congestive failure may also be a result mechanical complications of ischemic heart disease such as mitral regurgitation due to papillary muscle ischemia, infarction or rupture, a ventricular septal rupture or aneurysm formation. These conditions are easily diagnosed on echocardiography.

However, it is often difficult to differentiate ischemic from non-ischemic causes of heart failure by echocardiography alone. This is important because of the attendant, investigative, therapeutic and prognostic implications. Detection of regional wall motion abnormalities in contiguous areas concordant with known coronary arterial territories, presence of areas of infarction and fibrosis and absence of right ventricular dysfunction point to a likely ischemic etiology of heart failure. However, these methods are not infallible and cannot be relied on to differentiate between the two entities.

Cardiomyopathies

The distinguishing features of the various forms of cardiomyopathies are easily identified by echocardiography. In the case of dilated and hypertrophic cardiomyopathies—the most common forms of cardiomyopathy—the definitions reflect the underlying ventricular function, wall thickness, and chamber size. In hypertrophic cardiomyopathy the ventricular walls are hypertrophied, the cavity is small, and ventricular function is normal or hyperkinetic. In dilated cardiomyopathy the cavity is enlarged, wall thickness is normal or thin, and ventricular function is depressed. Restrictive cardiomyopathies are characterised by impaired or restricted ventricular filling as demonstrated by the typical transmitral Doppler profile (increased E/A ratio, rapid E wave deceleration time). The wall thickness, cavity size, and ventricular function can vary depending on the underlying

aetiology and duration of the restrictive cardiomyopathy. Echocardiographic features of arrhythmic right ventricular dysplasia include focal dilatation, thinning, and hypokinesis of the right ventricle.

Dilated cardiomyopathy is characterised with echocardiography by the presence of a dilated left ventricle with impaired ventricular systolic function. Echocardiography may be utilised to determine the degree of impairment of LV systolic function and to characterise diastolic function. The presence of isolated wall motion abnormalities which correlate with a specific coronary artery distribution suggests the presence of underlying coronary artery disease. In addition to LV size and systolic function, specific features of dilated cardiomyopathy which can be assessed by echocardiography include the degree, if any, of RV dilatation and systolic dysfunction, an estimation of RV systolic pressures derived from the tricuspid regurgitation Doppler velocities, the presence of LV thrombus and an assessment of left atrial (LA) and LV end diastolic pressure (LVEDP).

Hypertrophic cardiomyopathy is characterised by variable degrees of LV hypertrophy and diastolic dysfunction. Characteristic echocardiographic features of hypertrophic cardiomyopathy include variable degrees of RV and/or LV hypertrophy. Several forms of the disease are seen with echocardiography: hypertrophic non-obstructive cardiomyopathy, hypertrophic obstructive cardiomyopathy (HOCM), and apical variant of hypertrophic cardiomyopathy. In those with HOCM, systolic anterior motion of the mitral valve and the presence of left ventricular outflow tract (LVOT) obstruction are noted on echocardiography. LV wall thickness in excess of 13 mm without apparent cause, or a ratio of the septal to posterior wall thickness of > 1.3 , is diagnostic of HOCM. Right ventricular outflow tract obstruction occurs less frequently.

Restrictive cardiomyopathies are less common than dilated and hypertrophic cardiomyopathies. They are associated with impaired ventricular filling and increased LV end diastolic pressure. Echocardiographic features include biatrial dilatation, hypertrophied ventricles with decreased compliance, initially small LV cavities, and normal to depressed systolic function. Specific echocardiographic features when present are helpful in identifying the aetiology of the restrictive cardiomyopathy. The echocardiographic features of endocardial fibrosis associated with hypereosinophilia include haemodynamic evidence of restriction and obliteration of the ventricular apices caused by deposition of thrombus and eosinophilic cationic protein. The regional myocardial motion adjacent to this deposition remains normal. Idiopathic restrictive cardiomyopathy is a rare entity distinguished from the other forms of restrictive cardiomyopathy by the presence of normal ventricular wall thickness. Characteristic two dimensional and Doppler echocardiographic features have been described in individuals with cardiac amyloidosis. Two dimensional features include thickening of the LV walls, increased reflectivity of these walls (the “speckled” or “granular” myocardium), biatrial enlargement, thickening of the interatrial septum, thickening and regurgitation of the mitral and tricuspid valves, and the presence of a small pericardial effusion. Transmitral Doppler flow patterns in patients with amyloidosis exhibit evidence of diastolic dysfunction. Characteristic transmitral Doppler patterns representative of early impaired relaxation and later restrictive filling have been demonstrated

in this population. An increase in both the pulmonary venous Doppler atrial reversal duration and the ratio of this atrial reversal duration to the transmitral A wave duration have been observed in these patients and reflect increased LA pressure. Doppler tissue echocardiography is helpful in differentiating amyloid patients from those with similar two dimensional echocardiographic features but without amyloidosis. Abnormally low tissue Doppler diastolic velocities are present in individuals with cardiac amyloid compared with control patients.

Arrhythmic right ventricular dysplasia (ARVD), also known as arrhythmic right ventricular cardiomyopathy, is an idiopathic cardiomyopathy that is associated with RV fibrosis, fatty infiltration, and dysfunction. It can be complicated by symptoms of heart failure, heart block, and ventricular arrhythmias, and is associated with an increased risk of sudden cardiac death. While RV involvement is universal, the left ventricle is involved less frequently and the degree of involvement is less severe. Echocardiographic features are variably present and include RV dysfunction and RV outflow tract dilation, RV wall thinning, aneurysms of the posterior RV wall or RV free wall, and highly reflective moderator band and trabecular disarray.

Constrictive pericarditis

Constrictive pericarditis remains an important surgically curable form of “restrictive” heart disease causing heart failure due to diastolic dysfunction. While the clinical distinction between a restrictive cardiomyopathy and pericardial constriction in a patient with symptoms of systemic congestion can be challenging, echocardiography can non-invasively separate these two distinct entities. This distinction is extremely important since the treatments are dramatically different; pericardial constriction can be successfully treated with pericardial stripping while the treatment of restrictive cardiomyopathy is largely aimed at improvement in symptoms. The constraining effect of a thickened pericardium leads to rapid early diastolic filling characterised by a tall transmitral Doppler E wave with rapid deceleration phase (< 150 ms), > 30% increase in the mitral inflow peak velocity with exhalation, hepatic venous dilation and flow reversal, and two distinct septal motions which are unrelated to contraction. One of these septal motions is high amplitude and low frequency, and reflects differential filling of the ventricles during the phases of the respiratory cycle which is due to ventricular interdependence. The second is a low amplitude, high frequency diastolic septal motion which reflects differences in timing of the filling of the ventricles (the “septal bounce”). While the restrictive pattern of the transmitral E wave is similar in both constriction and restriction, there is no significant respiratory change in the transmitral inflow pattern associated with restrictive cardiomyopathy. Restrictive processes are also much more likely to be associated with pulmonary hypertension and the presence of diastolic mitral regurgitation. Tissue Doppler imaging can also distinguish between constriction and restriction. While the early mitral annular velocity (Ea) is notably decreased in restriction reflecting a myocardial abnormality, it remains normal in the presence of pericardial constriction since in this disease the myocardium typically remains normal.

Diastolic Heart Failure

Heart failure can arise from any condition that compromises the contractility of the heart (systolic heart failure) or that

interferes with the heart’s ability to relax (diastolic heart failure). Hospital- and community-based reports indicate that about one fourth to one half of patients with heart failure have normal left ventricular systolic function. Observational studies indicate that diastolic heart failure is more common in women and elderly persons. Although patients with diastolic heart failure have a lower annual mortality rate than patients with systolic heart failure, they have a higher rate than the general population. They also have hospitalization rates similar to those of patients with systolic heart failure.

The signs and symptoms of heart failure are nonspecific (dyspnea, exercise intolerance, fatigue, weakness) and often can be attributed to other conditions, such as pulmonary disease, anemia, hypothyroidism, depression, and obesity. Furthermore, it is difficult to distinguish diastolic from systolic heart failure based on physical findings or symptoms. Systolic heart failure is defined as a left ventricular ejection fraction of less than 45 percent, but diagnostic criteria for diastolic dysfunction are still controversial. Cardiac catheterization remains the gold standard for demonstrating impaired relaxation and filling, because it provides direct measurement of ventricular diastolic pressure. However, the balance of benefit, harm, and cost argue against its routine use in diagnosing diastolic dysfunction. Doppler echocardiography has assumed the primary role in the noninvasive assessment of cardiac diastolic function and is used to confirm the diagnosis of diastolic heart failure.

Guide to therapeutic interventions in heart failure

Valvular heart disease and heart failure

Mitral stenosis with CHF

1. Echo criteria for balloon mitral valvotomy
2. Echo criteria for open mitral valvotomy
3. Echo criteria for mitral valve replacement

Mitral regurgitation with CHF

1. Echo criteria for operability

On occasion, patients with mitral stenosis or regurgitation may have symptoms of dyspnoea out of proportion to the echocardiographic finding at rest. Addition of stress to the echocardiographic study often reveals greater hemodynamic derangements like increased PA pressure, increased trans-mitral gradients and greater severity of MR which helps explain the symptoms and take appropriate management decisions.

Aortic regurgitation with CHF

1. Echo criteria for operability

Aortic stenosis with CHF

1. Echo criteria for operability

Stress echocardiography with dobutamine infusion is particularly useful in clinical decision making in patients with aortic stenosis with LV dysfunction and low transvalvular gradients. In this group of patients, an important clinical question arises : is the low gradient a consequence of low cardiac output due to severe aortic stenosis which has led to LV dysfunction or is the low gradient a consequence of LV dysfunction unrelated to aortic stenosis with the aortic stenosis being an incidental finding? Assessment of valve area does not solve this diagnostic dilemma, because it cannot distinguish between severe fixed stenosis from

Table 3 : Selection of patients for cardiac replacement therapy

Risk	Low (5-10%/year)	High (> 25-30%/year)
Exercise capacity	> 8 -10 min	< 8 min
Contractile reserve (> 5% increase in global EF)	yes	No
Pulmonary HTN	< 45 mmHg	> 45 mmHg
RV dysfunction	No	Yes
MR	decreased or same	Increased

flow dependent (relative) aortic stenosis. Use of dobutamine stress with concomitant evaluation of cardiac output, aortic valve area and gradients provides a way out. On the basis of test results, it is possible to distinguish 3 groups of patients - 1) Patients with an improvement of contractile function but no significant increase in valve area and an increase in transvalvular gradients. These patients have severe fixed aortic stenosis and are good candidates for surgery with an acceptable operative risk. 2) Patients with contractile reserve with an increase in valve area without substantial increase in trans-valvular gradients. These patients have no critical aortic stenosis and should be treated conservatively. 3) Patients without contractile reserve and no modification of valve area and transvalvular gradients. These patients represent an ambiguous group representing patients with end-stage severe aortic stenosis with severe LV dysfunction or with severe LV dysfunction without contractile reserve and incidental non-significant aortic stenosis. This group has a very poor prognosis.

Ischemic heart disease with CHF

The identification of viable hibernating myocardium in patients with ischemic heart disease and chronic LV dysfunction or heart failure is of vital importance. Studies done over the past few years in this group of patients show that those with evidence of hibernating myocardium who do not undergo revascularisation have poor prognosis with high incidence of cardiac events at follow-up. In contrast, evidence of viable myocardium in patients undergoing successful revascularisation is associated with longer survival with improvements in both symptoms and LV function.

End diastolic wall thickness of akinetic segments with resting echocardiography can be used as an initial screening technique for assessment of viability. Indeed, akinetic regions with an end diastolic wall thickness <6 mm do not contain viable myocardium and do not improve in function after revascularisation. However, in segments with a thickness of 6 mm or more, additional testing is needed because approximately 40% of these regions do not contain viable myocardium and will not improve after revascularisation. This testing is easily accomplished using Dobutamine stress echocardiography.

Also, there seems to be a relationship between improvement in global LV function and the number of segments with viability, indicating that the extent of jeopardized but viable myocardium determines the magnitude of improvement in LVEF after revascularisation. Usually a level of 4 or more viable segments, which corresponds to an improvement in wall motion score index >0.25 (about 25% of LV), predicts improvement in LVEF.

However, despite the presence of significant viability, some patients do not improve their LVEF after revascularisation largely because of extremely adverse LV remodelling and enlargement. This is especially true for patients with high LV end-diastolic volumes (140 ml or more) who have a low likelihood of improving LVEF. Such patients are better candidates for alternative treatments like volume reduction, cardiac transplantation or cell transplantation.

End-stage heart disease

Selection of patients for cardiac resynchronisation therapy

Echocardiography appears to be of value in defining individuals who may or may not be candidates for CRT.

With the advent of cardiac resynchronization therapy (CRT), echocardiography has found new purpose in determining the effects of the therapy and appropriate settings for biventricular pacemakers. LV ejection fraction (EF) and LV synchrony, as measured by echocardiography, have both been noted to be important parameters used to identify patients most likely to benefit from CRT.

Selection of patients for cardiac replacement therapy

Rest and stress echocardiography are useful tools in the assessment of prognosis of patients with end-stage heart disease and to select recipients for heart replacement therapy. Indeed, some patients with marked reduction in myocardial contractility at rest, but with good residual contractile reserve on stress echocardiography, have a favourable exercise capacity and prognosis, whereas patients with milder symptoms and similar degree of abnormal myocardial contractility at rest, but without contractile reserve, have a poor outcome. In terms of discriminating survivors from non-survivors, it appears that VO₂ max < 10 ml/kg/min defines high risk, while a value of >18 ml/kg/min defines low risk; those with values in between may represent a gray zone. Stress echocardiography yields the greatest incremental prognostic value in these patients as shown in Table 3.

Guide to therapy in patients with HOCM

Echocardiography can be utilised to tailor treatment for individuals with hypertrophic cardiomyopathy. The response to medical treatment for hypertrophic cardiomyopathy, including a decrease in an outflow tract gradient or improvement in diastolic function, can be evaluated by serial echocardiographic studies. The decision to proceed with non-medical therapeutic options, including surgical septal reduction, percutaneous alcohol septal reduction, or DDD pacing, can also be guided by echocardiography. The presence and degree of LVOT obstruction detected by echocardiography aids the clinician in determining whether or not to perform septal reduction. Surgical septal reduction is recommended in individuals with symptoms refractory to medical treatment in the presence of a resting gradient of > 50 mm Hg. Indications for percutaneous alcohol septal reduction include a septal thickness > 18 mm Hg, the presence of a resting LVOT gradient of > 30 mm Hg, or an inducible gradient of > 60 mm Hg in the presence of NYHA functional class III or IV symptoms unrelieved by maximal medical treatment.

Echocardiographic guidance during alcohol septal ablation, using intravenous contrast selectively into septal coronary vessels,

allows the operator to identify the correct vessel supplying the appropriate myocardial territory, thus decreasing the likelihood of infarction of other portions of the heart and reducing the need for permanent pacing

Guide to prognosis

Numerous studies have been performed demonstrating the prognostic value of echocardiographic indices of cardiac size and function in heart failure patients. Regardless of the aetiology of the heart failure, the findings suggest that these echocardiographic measures provide prognostic value and should be integrated in the assessment of these patients.

Echocardiographic measures with documented prognostic value in heart failure patients include LV systolic function, RV systolic function, LV diastolic function, LV size, RV size, LA size, severity of mitral regurgitation, severity of tricuspid regurgitation, RV systolic pressure, ventricular synchrony, and measures of contractile reserve. LV ejection fraction has long been the primary index used as a marker of risk in heart failure and recently the strength of this marker has been demonstrated even in the elderly. While most studies have focused on ejection fraction as a marker of prognosis in chronic heart failure, the same relation holds in acute heart failure and cardiogenic shock. Particularly in cardiogenic shock the value of ejection fraction has been shown to remain present regardless of type of treatment.

While initial measures of diastolic filling and function by echocardiography were shown to be of value in patients with symptoms of heart failure but preserved systolic function, the prognostic value of these diastolic indices remain even when the ejection fraction is low. These measures appear to be additive in value to the ejection fraction and may even be stronger measures of prognosis than LV ejection fraction. The transmitral pulse wave Doppler deceleration time has been shown to be a powerful predictor of functional capacity and correlates with maximum oxygen consumption. Thus it is no surprise that measures of diastolic filling have prognostic value in heart failure patients. For example, in symptomatic congestive heart failure patients the restrictive filling pattern of transmitral Doppler, especially a deceleration time 140 ms, has been shown to be the single best predictor of cardiac death in patients with ischaemic and

dilated cardiomyopathy. For those with cardiac amyloid diastolic function has been shown to be a stronger predictor of cardiac death than LV wall thickness or systolic function regardless of symptom status. The pattern of transmitral Doppler velocity profiles are highly dependent on preload and, as described above, pseudonormal patterns can be produced. Since the diastolic measures with prognostic value are indirect measures of LA and LV diastolic pressure and the pseudonormal patterns are a response to increased diastolic pressures, it is logical to ask if pseudonormal patterns have prognostic value in heart failure patients. In fact the pseudonormal transmitral Doppler filling pattern has been shown to identify patients with an intermediate prognosis. Specifically those with abnormal relaxation patterns appear to have the lowest all cause death and rehospitalisation for congestive heart failure compared to those with pseudonormal filling or restrictive filling.

In both ischaemic and idiopathic dilated cardiomyopathy, the prognostic value of tricuspid regurgitation has been demonstrated. Additionally for patients with impaired systolic function increasing degrees of mitral regurgitation have a direct impact on survival. While the majority of investigations have focused on the value of valvar regurgitation in assessing prognosis in chronic heart failure, similar relations are seen with acute heart failure shock where the one year survival has been shown to depend on the extent of mitral regurgitation assessed at presentation with shock.

Contractile reserve and myocardial viability are important prognostic factors in ischaemic cardiomyopathy. Viable myocardium identified by dobutamine echocardiography identifies the subset of ischaemic cardiomyopathy patients who benefit most from revascularisation. Specifically those with myocardial viability who undergo revascularisation have threefold lower long term mortality than those ischaemic cardiomyopathy patients without viability or those with viability who do not undergo revascularisation.

Recently investigators have focused on LV synchrony as a marker of prognosis and shown that those with widened QRS duration have a poorer long term survival than those with coordinated wall motion and dilated cardiomyopathy.