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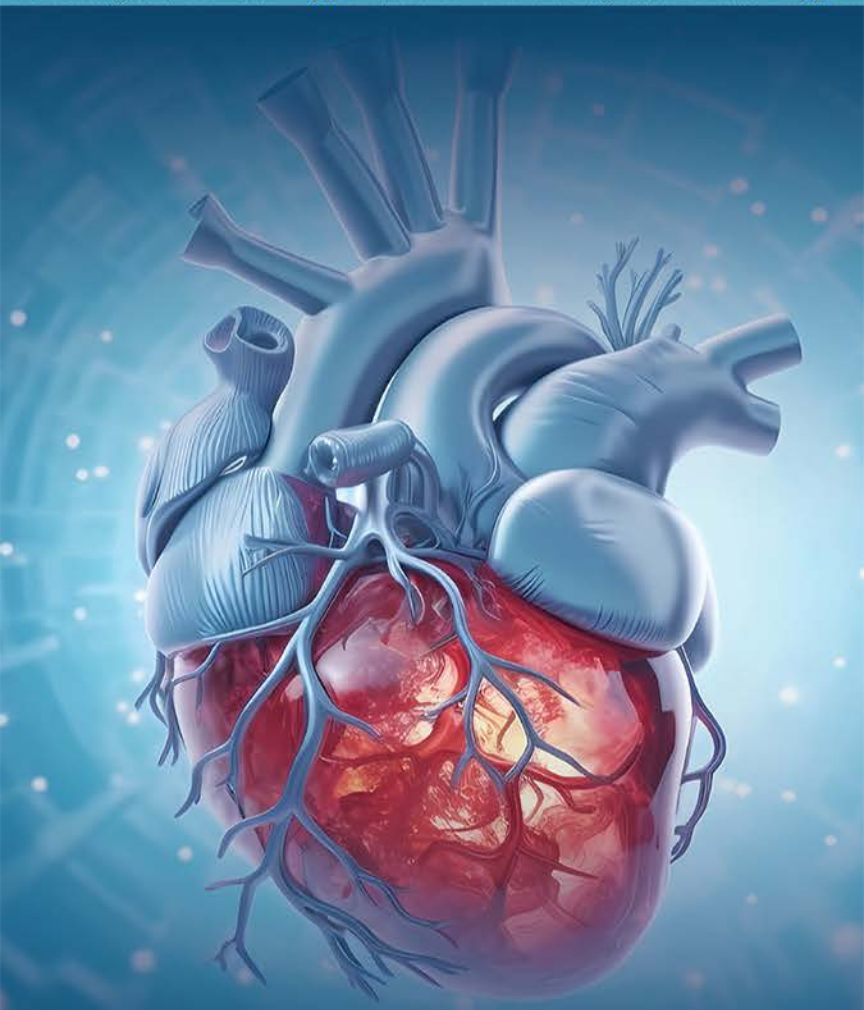

Manual of Heart Failure

Kanu Chatterjee
Mark Anderson
Donald Heistad
Richard E Kerber

JAYPEE

Contents

1. Heart Failure: Epidemiology **1**
Kanu Chatterjee

- *Introduction 1*
- *Epidemiology 1*
- *Prevalence 2*
- *Incidence 5*
- *Secular Trends 10*

2. Heart Failure: Diagnosis **15**
Kanu Chatterjee

- *Analysis of Symptoms 15*
- *Physical Examination 16*
- *Electrocardiogram 20*
- *Chest Radiograph 22*
- *Echocardiography 24*
- *Radionuclide Ventriculography 27*
- *Cardiac Magnetic Resonance 28*
- *Cardiac Tomography 29*
- *Routine Laboratory Tests 30*
- *Biomarkers 30*
- *Exercise Tests 36*
- *Six-minute Walk Test 37*
- *Coronary Arteriography 37*
- *Myocardial Ischemia 37*
- *Endomyocardial Biopsy 38*
- *Genetic Studies 40*

3. Systolic Heart Failure (Heart Failure with Reduced Ejection Fraction) **45**
Kanu Chatterjee

- *Introduction 45*
- *Historical Perspective 45*
- *Ventricular Remodeling 47*
- *Functional Derangements and Hemodynamic Consequences 61*
- *Initial Treatment of Systolic Heart Failure 62*
- *Symptomatic Systolic Heart Failure 64*
- *Follow-up Evaluation 83*

4. Diastolic Heart Failure (Heart Failure with Preserved Ejection Fraction) **96**
Kanu Chatterjee

- *Introduction 96*
- *Definition 96*
- *Epidemiology 97*
- *Pathophysiology 98*
- *Clinical Presentation 106*
- *Diagnosis 107*
- *Prognosis 109*
- *Treatment Strategies 113*
- *Future Directions 119*

5. Anemia in Patients with Chronic Heart Failure (Prevalence, Mechanism, Significance, and Treatment) 126
James Prempeh, Barry M Massie

- *Overview of the Problem 126*
- *Prevalence of Anemia in Heart Failure Patients 126*
- *Mechanisms Underlying Anemia in Heart Failure Patients 127*
- *Prognostic Significance of Anemia in Heart Failure Patients 128*
- *Should Anemia Be Treated in Heart Failure Patients? 129*
- *Safety Concerns Related to ESPs in a Variety of Anemic Patients 129*
- *Treatment of Anemia in Heart Failure Patients 131*

6. Hyponatremia and Congestive Heart Failure 142
Anne Mani, David J Whellan

- *Introduction 142*
- *Mechanisms Causing Hyponatremia in Heart Failure 147*
- *Treatment of Hyponatremia 151*
- *Role of Diuretic Therapy in Hyponatremia 152*
- *Role of Vasopressin Receptor Antagonists in Hyponatremia 153*
- *Tolvaptan 154*
- *Lixivaptan 157*
- *Conivaptan 158*

7. Cardiorenal Syndrome: The Interplay Between Cardiac and Renal Function in Patients with Congestive Heart Failure 163
Nestor Mercado, J Thomas Heywood

- *Introduction 163*
- *Epidemiology of Chronic Kidney Disease in Patients with Heart Failure 163*
- *Prognosis of Worsening Renal Function 164*
- *Definition of the Cardiorenal Syndrome 172*
- *Pathophysiology of the Cardiorenal Syndrome 175*
- *Role of Decreased Cardiac Output 175*
- *Role of Elevated Central Venous Pressure 178*
- *Role of Evidence-based Therapies in Patients with Heart Failure and the Cardiorenal Syndrome 180*
- *Role of Ultrafiltration on Diuretic Resistance and the Cardiorenal Syndrome 187*
- *Treatment of the Cardiorenal Syndrome: An Approach to the Individual Patient 189*

8. Acute Heart Failure Syndromes 200
Peter S Pang, Michel Komajda, Mihai Gheorghiade

- *Introduction 200*
- *Definition 200*
- *Epidemiology 201*
- *Patient's Characteristics 201*
- *Classification 203*
- *Pathophysiology 204*
- *Acute Heart Failure Syndromes Management 209*
- *Clinical Trials in Acute Heart Failure Syndromes 219*

9. Cardiopulmonary Exercise Testing and Training in Heart Failure **230**
Ileana L Piña

- *Introduction 230*
- *Normal Response to Exercise 230*
- *Exercise Response in Heart Failure 232*
- *Cardiopulmonary Exercise Testing 234*
- *Indications for CPX Testing in Heart Failure 240*
- *Exercise Training in Heart Failure 243*

10. Hibernating Myocardium **255**
Kanu Chatterjee

- *Introduction 255*
- *Historical Perspective 256*
- *Definition 256*
- *Pathophysiology 256*
- *Hibernation and Stunning: Clinical Prevalence 260*
- *Detection of Hibernating Myocardium 261*
- *Revascularization of Hibernating Myocardium and Changes in Ventricular Function 266*
- *Revascularization of Hibernating Myocardium and Changes in Prognosis 268*

11. Advanced Cardiac Therapies for End-stage Heart Failure: Cardiac Transplantation and Mechanical Circulatory Support **281**
Ashrith Guha, Frances Johnson

- *Introduction 281*
- *Identifying Candidates for Advanced Cardiac Therapies 283*
- *Heart Transplantation 290*
- *Mechanical Circulatory Support 305*

12. Palliative Medicine and End-of-life Care in Heart Failure **324**
KellyAnn Light-McGroary

- *Introduction 324*
- *Epidemiology of Heart Failure 325*
- *Economic Impact of Heart Failure 325*
- *History of Palliative Care/Definitions 328*
- *Feasibility of the Use of Palliative Care in Heart Failure 329*
- *Issues of Prognostication 331*
- *Communication and Patient's Understanding of their Disease 332*
- *Suffering in End-stage Heart Failure 334*
- *Symptom Management in Heart Failure 337*
- *Management of Implantable Cardiac Devices 342*

Index *357*

CHAPTER

Diastolic Heart Failure
(Heart Failure with Preserved Ejection Fraction)

Kanu Chatterjee

Chapter Outline

- Definition
- Epidemiology
- Pathophysiology
  - Ventricular Remodeling
  - Neurohormonal Changes
  - Functional Derangements
  - Hemodynamic Consequences
- Clinical Presentation
- Diagnosis
- Prognosis
- Treatment Strategies
- Future Directions

INTRODUCTION

Diastolic heart failure as a clinical entity was recognized over 70 years ago by Dr. Fishberg, who wrote in 1937 that "this form of cardiac insufficiency results from inadequate filling of the heart" and he termed this form of heart failure as "hypodiastolic failure".[1] However the syndrome of diastolic heart failure was not appreciated until about two decades ago when the cardiologists and the heart failure specialists recognized that diastolic heart failure is as common as systolic heart failure. The prevalence of diastolic heart failure appears to have also increased in recent years. In one community-based study, the prevalence of diastolic heart failure was approximately 35% in 1986, and it increased to over 50% in 2002.[2] It should also be appreciated that the recognition of this clinical subset of chronic heart failure by the physicians has also increased in recent years.

DEFINITION

There are not only confusions regarding the definition of diastolic heart failure but also for the timing of onset and duration of diastole. In the Webster Dictionary, diastole is defined as "the dilatation of the heart with blood: opposed to systole, or contraction".

The onset of diastole coincides with the closure of the aortic valve as it has been conventionally recognized as the onset of left ventricular relaxation.[3] However, as the left ventricular ejection influences its relaxation, it has been proposed that these phases should be regarded as part of systole and not of diastole.[4] In clinical practice the opening of the mitral valve is used as the beginning of diastole and the closure of the mitral valve as the end of diastole.[5] This phase is also called auxotonic relaxation phase.

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One of the pathophysiologic definitions, as proposed by Brutsaert et al.[4] is that it is "a condition resulting from an increased resistance to filling of one or both ventricles leading to symptoms of congestion due to an inappropriate upward shift of the diastolic-pressure-volume relation (i.e. during the terminal phase of the cardiac cycle). Another proposed pathophysiologic definition is that it is a condition in which the "ventricular chamber is unable to accept an adequate volume of blood during diastole at normal diastolic pressures and at volumes sufficient to maintain an appropriate stroke volume".[6] Although these definitions describe the pathophysiologic characteristics of diastolic heart failure, they cannot be used in clinical practice.

A number of clinical definitions have also been proposed. One definition is that the diastolic heart failure is a "clinical syndrome characterized by the symptoms and signs of heart failure, a preserved ejection fraction (EF), and abnormal diastolic function".[6] Other definitions, such as "heart failure with normal or near normal ejection fraction", have also been used. In clinical practice most commonly used definition of diastolic heart failure (HFNEF) is when "the symptoms and signs of heart failure are present and the ejection fraction is greater than 45%". It should be appreciated that EF is load dependent. A lower preload and a higher after load are associated with a lower EF. Thus, at the time of measurement of EF, it is desirable to consider the determinants of preload (e.g. end-diastolic volume) and of afterload (e.g. blood pressure).

EPIDEMIOLOGY

The incidence and prevalence of diastolic heart failure have been studied in a number of epidemiologic studies.[2,7-12] These studies estimated the prevalence of diastolic heart failure between 50% and 55%. The prevalence increases with age, and it is more common in women than in men. In the cardiovascular health study,[11a,12] the risk of developing heart failure was higher in elderly women. In women at age between 65 and 69 years, the incidence was 6.6%, and it increased to 14% in women older than 85 years.[7,11a,12] That diastolic heart failure is more common in women than in men has been recognized in many studies.[13,14] In the cardiovascular health study, the prevalence of diastolic heart failure in women was 67% and that in men 42%.[11a,12] Similar findings were observed in the Framingham study.[13,14] In the Candesartan in Heart Failure-Assessment of Reduction in Mortality and Morbidity-Preserved (CHARM-Preserved) trial, the incidence of diastolic heart failure was higher in women than in men.[15] In this study, left ventricular EF needed to be

of women was 40% in the CHARM-Preserved study. In the

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TABLE 1

Systolic vs diastolic heart failure

ADHERE—All enrolled discharges		
Profile	*SHF*	*DHF*
EF	(59,523)	(50,497)
EF	<40%	>40%
Age	69.9	74.2*
Female	39%	62.2%*
CAD	63%	54%*
Diabetes	42%	46%*
AF	29%	33%*
BNP	1486	925*

*<0.0001
(Abbreviations: EF: Ejection fraction; CAD: Coronary artery disease; AF: Atrial fibrillation; SHF: Systolic heart failure; DHF: Diastolic heart failure)

"Irbesartan in patients with heart failure and preserved ejection fraction (I-PRESERVE)" trial, the prevalence of women was 60%.[16] The left ventricular ejection needed to be greater than 45% to be enrolled to the study. In patients hospitalized for the treatment of heart failure the prevalence of women with diastolic heart failure was approximately 60%.[17] Normal EF in this study was defined when the ejection was 55% or higher.

The echocardiographic studies have reported that the prevalence of diastolic heart failure in females is between 1.7% and 9.5% and in males between 2.7% and 6.6%.[10] In this study the prevalence of diastolic heart failure in men and women were similar.

The risk factors for diastolic heart failure are similar to those of systolic heart failure **(Table 1)**.[17] Older age, female gender, diabetes, hypertension, obesity and the black race are the major risk factors for the development of heart failure. The incidence of coronary artery disease appears to be higher in systolic heart failure and obesity, diabetes and hypertension higher in diastolic heart failure. However, the incidence of coronary artery disease is still considerable in diastolic heart failure and diabetes and hypertension in systolic heart failure. In clinical practice therefore it is relevant to attempt to treat the modifiable risk factors in both diastolic and systolic heart failure.

PATHOPHYSIOLOGY

VENTRICULAR REMODELING

In diastolic heart failure, left ventricle is of normal size, and its wall thickness is increased. Left ventricular hypertrophy is of concentric type. Left ventricular end-diastolic and end-systolic volumes are normal or decreased. Thus, left ventricular cavity

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TABLE 2

Diastolic heart failure—remodeling

- Ventricular hypertrophy, usually concentric
- Increased ventricular mass
- Increased ventricular wall thickness
- Little or no increase in the cavity size
- Increased mass/cavity ratio
- Decreased wall stress
- Maintained ejection fraction
- Little or no change in ventricular shape
- Mechanical dyssynchrony with or without electrical dyssynchrony—present in approximately 1/3rd of patients

TABLE 3

Diastolic heart failure

	Controls	*DHF*
• LVEDV	102	87
• LVESV	46	37
• LVEF	54	60
• LVM	125	160
• LVM/V	1.49	2.12

(Abbreviations: LVEDV: Left ventricular end diastolic volume; LVESV: Left ventricular end systolic volume; LVEF: Left ventricular ejection fraction; LVM: Left ventricular mass; LVM/V: Left ventricular mass/volume ratio; DHF: Diastolic heart failure) (*Source:* Adapted from Kitzman DW, et al. JAMA. 2002;288:2144)

size remains normal or even may be decreased. Left ventricular mass is increased and the mass/cavity ratio is increased. Due to increased wall thickness without any change in the cavity size, left ventricular wall stress is decreased, which maintains normal EF.[18-23] Tissue Doppler imaging (TDI) studies have reported the prevalence of dyssynchrony in over 30% of patients with diastolic heart failure.[24,25]

left ventricular mechanical dyssynchrony may occur with or without electrical dyssynchrony.[26] The morphologic changes in diastolic heart failure are summarized in **Tables 2 and 3**.[23,27]

The distinctive features of left ventricular remodeling in isolated diastolic and systolic heart failures are illustrated in **Figure 1**. In this example, compared to a normal heart, in diastolic heart failure the left ventricular wall thickness is markedly increased and the cavity is small. A cross-sectional 2-dimensional echocardiographic view also demonstrates increased left ventricular wall thickness in diastolic heart failure. In contrast, in systolic heart failure the thickness of left ventricular wall is decreased and the cavity is dilated compared to normal wall. The cross-sectional 2-dimensional echocardiogram demonstrates similar features.[28]

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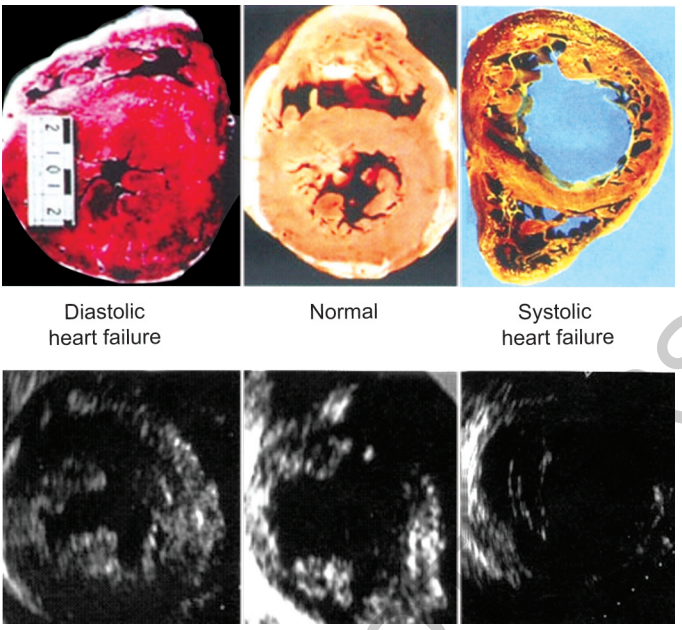

FIGURE 1: Autopsy examples of ventricular remodeling in diastolic and systolic heart failure are illustrated. Left-hand panel demonstrates gross pathology and right-hand panel 2-dimensional cross-sectional echocardiographic images (*Source*: MA Konstam. J Cardiac Failure. 2003;9:1-3)

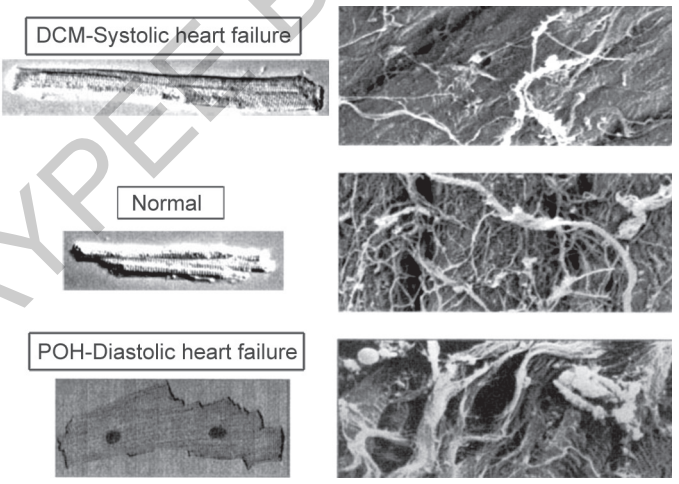

FIGURE 2: Changes in myocytes (left) and in extracellular matrix (right) are illustrated. (*Source*: Aurigemma et al. Circulation. 2006;113:296-304, with permission)

The characteristic changes in the myocytes and in the matrix in diastolic and systolic heart failure are illustrated in **Figure 2**.[19] Compared to normal myocyte, the myocyte in diastolic heart failure is thicker and there is increase in diameter without an increase in its length. The length/width ratio is

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TABLE 4

Myocardial structure and function in diastolic and systolic heart failure

	DHF	*SHF*	*P*
MYD (microM)	20.3	15.1	<0.001
CVF	+	+	NS
MFD (%)	46	36	<0.001

(Abbreviations: MYD: Myocyte diameter; CVF: Collagen volume fraction; MFD: Myofibrillar density; DHF: Diastolic heart failure) (*Source*: Heerbeek LV et al. Circulation. 2006;113:1966-73)

decreased. There is increased myocyte protein synthesis. In systolic heart failure the myocyte length is increased without changing its diameter. The length/width ratio is increased. Left ventricular endomyocardial biopsy in patients with symptomatic diastolic and systolic heart failure demonstrates distinctive features in myocardial structure **(Table 4)**.[23] Myocyte diameter in diastolic heart failure is greater than that in systolic heart failure. Myofibrillar density is less in systolic than in patients with diastolic heart failure. There is, however, no difference in collagen volume in diastolic and systolic heart failure.

In both diastolic and systolic heart failure the collagen volume is increased.[19,22,23] In diastolic heart failure, the thickness of the collagen bundles and the continuity of the fibrillar components of the extracellular matrix surrounding the myocytes are increased. In systolic heart failure there is degradation and disruption in collagen.[19] The collagen cross links are increased in diastolic heart failure. The matrix metalloproteinases (MMPs) are reduced and the endogenous tissue inhibitors of metalloproteinases (TIMPs) are increased. Thus the ratio of the MMPs/TIMPs is decreased in diastolic heart failure.[29,30]

Abnormal calcium regulation has been observed in diastolic heart failure. The sarcoplasmic reticular reuptake of cytosolic calcium is abnormal and is associated with slower myocyte relaxation.[31,32]

Titin is a large myocyte protein which has a recoil spring property. It also desensitizes the myofilaments to calcium. The stiff titin isoform protein N2B is over expressed in human cardiac myocytes isolated by myocardial biopsy from patients with diastolic heart failure. The N2BA titin isoform protein is more compliant and the N2BA/N2B ratio is lower in the myocytes of patients with diastolic heart failure.[23,33] In diastolic heart failure, the procollagen type I and III aminoterminal peptides concentrations are increased suggesting the abnormalities of collagen metabolism.

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TABLE 5

Diastolic heart failure

	DHF
• Myocyte hypertrophy	+
apoptosis	+
necrosis	+
• Fibrosis	+
• Ca regulation	–
• MMPs/TIMPs	–
• Collagen cross-links	+
• Titin isoforms N2BA/N2B	–

(Abbreviation: DHF: Diastolic heart failure)
+: Increased; –: Decreased

The changes in myocytes and extracellular matrix are summarized in **Table 5**.[34]

Myocardial ischemia impairs myocardial relaxation due to abnormality of calcium regulation. There is reduced reuptake of the cytosolic calcium by the sarcoplasmic reticulum.[35] Ischemia may induce myocyte necrosis and apoptosis.

NEUROHORMONAL CHANGES

A number of neurohormonal abnormalities have been observed in diastolic heart failure. The plasma norepinephrine levels are increased.[27] The circulating levels of interleukin-6 and interleukin-8 and of tumor necrosis factor-alpha (TNFα) are increased.[34,36] These neurohormonal abnormalities may contribute to myocyte hypertrophy and myocardial inflammatory changes which may be associated with myocyte necrosis and apoptosis. The counter regulatory hormone B-type natriuretic peptide (BNP) is also increased. Despite increased levels of BNP, ventricular remodeling continues.

The principal functional abnormality in diastolic heart failure is increased stiffness and decreased compliance of the left ventricle. The diastolic pressure-volume relation shifts upward and to the left. As a result, there is a disproportionate increase in left ventricular diastolic pressure for any increase in left ventricular diastolic volume **(Figs 3 and 4)**. Myocardial hypertrophy, fibrosis and ischemia contribute to increased left ventricular fibrosis.

The analysis of left ventricular pressure-volume relations allows delineation of hemodynamic changes and their mechanisms **(Fig. 4)**. Diastolic filling begins with

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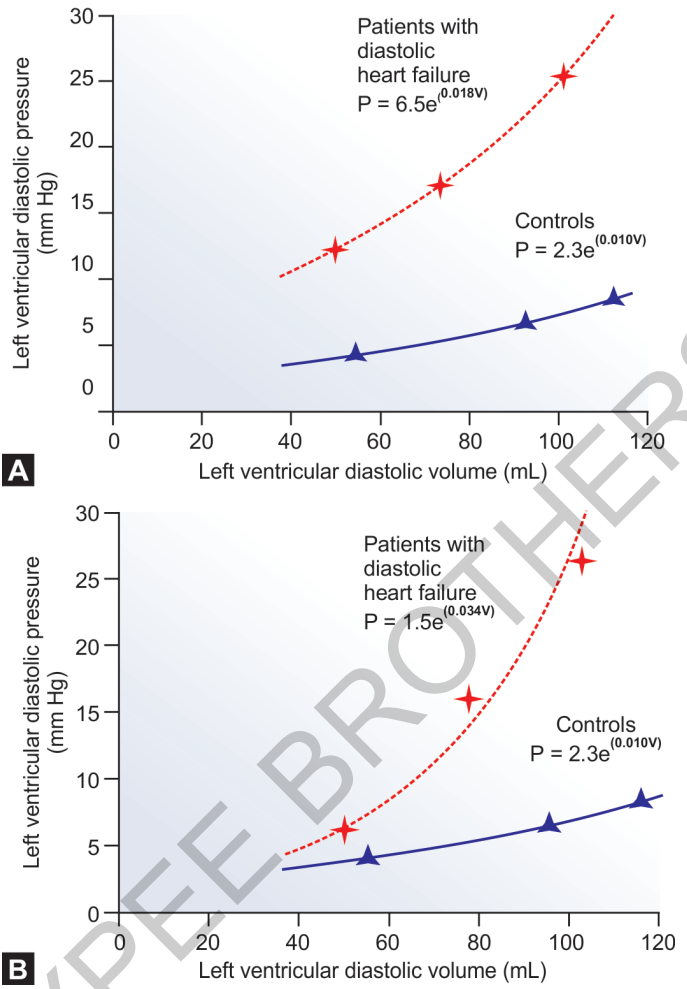

FIGURES 3A AND B: The changes in left ventricular diastolic pressure (mm Hg) (y-axis) and changes in left ventricular diastolic volume (mL) (x-axis) in groups of patients with diastolic heart failure and matched controls are illustrated. In patients with diastolic heart failure, the left ventricular-pressure volume relation is shifted upward and to the left. (*Source*: Zile et al. N Engl J Med. 2004;350:1953-9, with permission)

the opening of the mitral valve. Normally, 70–80% of left ventricular filling occurs during the rapid filling phase. It should be appreciated that after the opening of the mitral valve left ventricular pressure continues to decline into the rapid filling phase. The early diastolic pressure gradient between the left atrium and the left ventricle is maintained without any increase in left atrial pressure. During exercise which is associated with a need of a higher stroke volume, larger filling of the left ventricle is also required which is achieved with a more rapid decline and lower early diastolic pressure. The early diastolic pressure gradient is maintained

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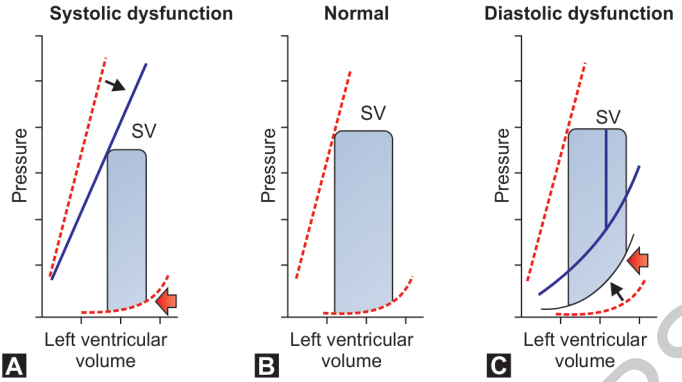

FIGURES 4A TO C: Schematic diagram of pressure-volume relations in Normal (B), Systolic dysfunction (A) and Diastolic dysfunction (C) are illustrated. In systolic heart failure, the end-systolic pressure-volume relation line (solid line) is shifted downward and to the right indicating reduced contractile function compared to normal (dashed line). In diastolic heart failure, there is no shift in the end systolic pressure-volume line indicating no change in contractile function. With a marked upward and leftward shift of the diastolic pressure-volume relation there is not only increased in left ventricular diastolic pressure but also a decrease in stroke volume (SV) (*Source*: Modified from GP Aurigemma. N Engl J Med. 2004;35:1097, with permission)

without any significant increase in left atrial pressure. Thus, in presence of normal diastolic function, pulmonary venous congestion does not occur. In the presence of significant diastolic dysfunction as in patients with diastolic heart failure, left ventricular relaxation is impaired and left ventricular early diastolic pressure is elevated. Consequently, left atrial pressure is also increased and there is a passive increase in pulmonary venous pressure which may be associated with signs and symptoms of pulmonary venous congestion. These hemodynamic abnormalities are accentuated during exercise. The rapid filling phase is not influenced by the increase in heart rate. During diastasis, that is the interval between the end of rapid filling phase and the beginning of the atrial filling phase, the relative amount of ventricular filling is small. During atrial filling phase, normally approximately 20% of ventricular filling occurs. In the presence of significant diastolic dysfunction as in patients with diastolic heart failure, the relative contribution of ventricular filling during atrial filling phase is greater as the degree of ventricular filling during the rapid filling phase is reduced. The atrial filling phase is influenced by heart rate, presence of sinus rhythm, and effective atrial contraction. The presence of sinus rhythm and effective atrial contraction is more important in diastolic than in systolic heart failure to maintain adequate stroke volume.

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After completion of ventricular filling with the closure of the mitral valve, the isovolumic systolic phase begins. During this phase, the left ventricular pressure increases without any change in ventricular volume. With the opening of the aortic valve left ventricular ejection begins and continues till the beginning of the hang-out time. The hangout time is the interval between the end of ejection and the closure of the aortic valve. With the closure of the aortic valve isovolumic relaxation phase begins and it ends with the opening of the mitral valve. At the end of ejection, the end-systolic pressure-volume relation remains unchanged, and the end-systolic pressure-volume relation is independent of afterload and preload. With decreased contractile state, the end-systolic pressure-volume line shifts downward and to the right as in patients with systolic heart failure. In patients with diastolic heart failure the contractile function is usually preserved, and there is no shift of the end-systolic pressure-volume line.

Due to the upward and leftward shift of the diastolic pressure-volume relation, left ventricular diastolic pressure is increased and there is a passive increase in pulmonary venous pressure with signs and symptoms of pulmonary venous congestion. With a further upward shift of the diastolic pressure-volume relation left ventricular filling is compromised which is associated with decreased stroke volume and cardiac output **(Fig. 4)**.

By definition diastolic heart failure is associated with normal or near normal left ventricular EF. However, controversy exists whether contractile function is entirely normal or not in patients with diastolic heart failure. The positive peak positive dp/dt, end-systolic elastance and endocardial stress-shortening relationships, which are indices of contractile function are not impaired in diastolic heart failure.[21] The TDI studies have reported that in some patients with diastolic heart failure the extent of midwall and long axis shortening is reduced indicating impaired contractile function. However, it occurs in less than 50% of patients.[19]

There is also controversy whether left ventricle dilates in patients with symptomatic diastolic heart failure in absence of ischemic myocardial necrosis. In a study of only ten patients with overt diastolic heart failure without coronary artery disease, during over 5 years of follow-up, there was not any significant increase in left ventricular end-diastolic volume between the initial and the follow-up study. However there was a significant increase in left ventricular end-diastolic pressure and increase in the indices of left ventricular stiffness **(Table 6)**.[37] This study indicates that left ventricle does not dilate in the absence of ischemic necrosis and the worsening symptoms are related to worsening left ventricular stiffness.

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TABLE 6

Changes in left ventricular volume and function in diastolic heart failure without coronary artery disease

	LVEDVI (mL/m²)	*LVEDP (mm Hg)*	*Stiffness-Mod (kn/m²)*	*LVEF (%)*
Init	68 ± 9	14 ± 3	3.4 ± 0.6	67 ± 3
End	76 ± 8	26 ± 2	6.3 ± 0.9	60 ± 4

(Abbreviations: LVEDVI: Left ventricular end diastolic volume index; LVEDP: Left ventricular end diastolic pressure; Stiffness-Mod: Stiffness modulus; Init: Initial study; End: End of follow-up of 64 ± 9 months). (*Source*: Handoko ML et al. Circulation. 2006;114:II-816)

HEMODYNAMIC CONSEQUENCES

The hemodynamic consequences of advanced diastolic heart failure are characterized by an increase in left ventricular diastolic pressure and a passive increase in left atrial and pulmonary venous pressures. There is an obligatory increase in pulmonary arterial pressure which is associated with increased right ventricular afterload. Thus, right ventricular failure ensues with clinical manifestations such as dependant peripheral edema. The prevalence, severity and the hemodynamic mechanisms of pulmonary arterial hypertension have been investigated in a community based population study.[38] Transthoracic echocardiography and Doppler echocardiography was used to assess pulmonary artery systolic and pulmonary capillary wedge pressures. In 244 patients with diastolic heart failure, pulmonary arterial hypertension was detected in 83% of patients. The mechanism of pulmonary artery hypertension was due to both increase in pulmonary capillary wedge pressure and pulmonary vascular resistance. Pulmonary hypertension was a strong predictor of mortality.

CLINICAL PRESENTATION

Asymptomatic left ventricular diastolic dysfunction is far more common than symptomatic diastolic heart failure.[8] However, in symptomatic patients, the signs and symptoms of overt diastolic heart failure are similar to those of systolic heart failure **(Table 7)**.[6] Exertional dyspnea is the most common presenting symptom and was present in 85% of patients. In more than 50% of patients, paroxysmal nocturnal dyspnea and orthopnea were present. Reduced exercise tolerance is also frequent in patients with diastolic heart failure.[27] The hemodynamic mechanisms of impaired exercise tolerance may be caused by disproportionate increase in pulmonary venous pressure and decreased cardiac output. Diastolic dysfunction was found to be an independent

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TABLE 7

Prevalence of specific symptoms and signs in systolic vs diastolic heart failure

	Diastolic HF (EF > 50%)	*Systolic HF (EF < 50%)*
Symptoms		
Dyspnea on exertion	85%	96%
Paroxysmal nocturnal dyspnea	55%	50%
Orthopnea	60%	73%
Physical examination		
Jugular venous distension	35%	46%
Rale	72%	70%
Displaced apical impulse	50%	60%
S3	45%	65%
S4	45%	66%
Hepatomegaly	15%	16%
Edema	30%	40%
Chest radiograph		
Cardiomegaly	90%	90%
Pulmonary venous hypertension	75%	80%

(Abbreviation: HF: Heart failure) (*Source*: Zile MR, Brutsaert DL. Circulation. 2002;105:1387-93)

predictor of impaired exercise tolerance in a study of 2,867 patients undergoing exercise echocardiography.[39]

Physical findings indicative of right heart failure, such as elevated jugular venous pressure and peripheral edema, are present in approximately 30% of patients **(Table 7)**.[6]

Postcapillary and mixed type of pulmonary arterial hypertension is the principal mechanism of right ventricular failure.

Evidence of left heart failure, such as signs of pulmonary venous congestion, left ventricular hypertrophy, left sided S_3 and S_4 gallops which indicate elevated left ventricular end-diastolic pressure are present in approximately 50% of patients with overt diastolic heart failure.

Cardiomegaly and signs of pulmonary venous hypertension are detected frequently in plain chest X-ray.

Sleep-disordered breathing has been reported to occur frequently in patients with overt diastolic heart failure.[40a] In a study of 244 consecutive patients sleep-disordered breathing was observed in approximately 70% of patients. About 40% of patients had obstructive and 30% central sleep apnea. The patients with sleep-disordered breathing had impaired exercise tolerance, and higher left ventricular end diastolic, pulmonary capillary wedge and pulmonary artery pressures.

DIAGNOSIS

The diagnosis of heart failure is made by the clinical evaluations, and the Framingham criteria are used for diagnosis. Once the

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Manual of
Heart Failure

Manual of Heart Failure is an up-to-date, concise and comprehensive book in cardiology. The book is containing 12 chapters. Advances in the management of systolic and diastolic heart failure, hibernating myocardium and advances in the use of implantable cardiac devices are unique features of this book. Chapters have been written by internationally recognized experts in their respective fields. Acute heart failure syndromes, cardiopulmonary exercise testing and training in heart failure, palliative medicine and end of life care in heart failure are also other highlighted features of this book.



Kanu Chatterjee is currently Professor of Medicine, the Carver College of Medicine, University of Iowa and Emeritus Professor of Medicine, University of California, San Francisco, USA. He is an internationally recognized expert in heart failure and coronary artery disease. He has received numerous awards including "Gifted Teacher Award" of the American College of Cardiology; the Legacy Award, University of California San Francisco; Melvin Marcus Distinguished Fellowship Award, International Academy of Cardiology; Lewis E January Clinical Teaching Award, University of Iowa; Cardiovascular Distinguished Scientist Award, University of Iowa and American Heart Association Professional Education Award. He is a Fellow of the Royal College of Physicians of London and of Edinburgh, American Heart Association, American College of Cardiology and a Master of the American College of Physicians. He is listed in the "Best Doctors in America" and in "America's Top Cardiologists".



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