# A STUDY ON PREVALENCE AND ASSOCIATED FACTORS OF DIASTOLIC HEART FAILURE IN ELDERLY WITH CONGESTIVE HEART FAILURE

*Dissertation submitted to*

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY,

CHENNAI, INDIA.

*in partial fulfillment of*

*requirements for*

**M.D DEGREE IN GERIATRICS**

**BRANCH-XVI**


## MADRAS MEDICAL COLLEGE,

**THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY,**

**CHENNAI 600003**

**APRIL 2011**

# CERTIFICATE

This is to certify that the dissertation entitled **"PREVALENCE AND ASSOCIATED FACTORS OF DIASTOLIC HEART FAILURE IN ELDERLY WITH CONGESTIVE HEART FAILURE"** is a bonafide work done by Dr.P.RANJITH at Madras Medical College, Chennai in partial fulfillment of the university rules and regulations for award of M.D.,Degree in Geriatrics(Branch XVI) under my guidance and supervision during the Academic Year 2008 - 2011.

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# DECLARATION

I solemnly declare that this dissertation entitled **"PREVALENCE AND ASSOCIATED FACTORS OF DIASTOLIC HEART FAILURE IN ELDERLY WITH CONGESTIVE HEART FAILURE"** was done by me at Madras Medical College Government General Hospital, during academic years 2008-2011under the guidance and supervision of Prof. B.KRISHNASWAMY, M.D. This dissertation is submitted to the Tamil Nadu Dr.M.G.R Medical University towards the partial fulfillment of requirements for the award of M.D.Degree in Geriatrics(Branch XVI) ,examination to be held in April 2011

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# ACKNOWLEDGEMENT

At the outset I thank **Prof.J.MOHANASUNDARAM M.D.Ph.D,** Dean Madras Medical College , Chennai-3 for permitting me to use hospital data for the study.

I am grateful to my chief, **Prof.B.KRISHNASWAMY M.D.,** Head of Department of Geriatric Medicine, Madras Medical College and Government General Hospital, Chennai-3 for his valuable guidance and encouragement during the course of stud

I also express my gratitude to **Dr.S.SIVAKUMAR M.D.** Reader in Department of Geriatric Medicine, **Dr.G.USHA, M.D.** Assistant Professor in Department of Geriatric Medicine, Madras Medical College and Government General Hospital, Chennai-3.

I wish to express my sincere thanks to **Dr.Geetha Subramaniyam., MD., DM,** Prof and Head of Department of cardiology, Madras Medical College and Government General Hospital, Chennai-3.

My sincere thanks to all the patients who participated in this study.

Lastly, I thank all my professional colleagues for their support and valuable criticisms.

# CONTENTS

# INTRODUCTION

Diastolic Heart failure (DHF) is characterized by signs and /or symptoms of heart failure in presence of a normal ejection fraction (EF).Prognosis of DHF is as poor as that of systolic failure, and the number of cases is likely to increase within aging populations. Heart failure with normal systolic function has been equated to diastolic heart failure (DHF). DHF appears to be quite common in the elderly, especially in elderly women with hypertension. Recent epidemiologic studies suggest that 30%-50% of patients with heart failure may have DHF. Morbidity for DHF is comparable to that of heart failure with left ventricular systolic dysfunction. Both groups of patients have similar rates of recurrent hospitalization and cost of care. Long-term mortality also appears to be similar in the two groups of patients. With the aging of the population, the number of patients with DHF will continue to rise and are likely to contribute significantly to the burden of disease caused by heart failure. Unfortunately, as yet, no reliable definition has been found for DHF. Currently the diagnosis of DHF is often made by exclusion, and treatment is empirical and unsatisfactory because of the lack of large-scale, randomized, controlled trials in this area; however, several large and other smaller trials are currently in progress that will hopefully provide some answers.

# AIM OF THE STUDY

- To assess the the prevalence of diastolic heart failure in elderly with congestive heart failure
- To study the association with demographic factors like

  1) Age
  2) Sex

  and comorbid conditions like

  1) Hypertension
  2) Diabetes mellitus
  3) Coronary artery disease
  4) Atrial Fibrillation
  5) Chronic kidney disease
  6) Hypothyroidism
  7) Obesity

# MATERIALS AND METHODS

**STUDY CENTRE:**

Department of Geriatric medicine, Madras Medical College & Government General Hospital Chennai-600003

**STUDY DESIGN:**

Cross sectional, descriptive study

**SAMPLE SIZE:**

Ninety

**STUDY DURATION:**

August 2008 - September 2011

**SELECTION OF PATIENTS:**

**INCLUSION CRITERIA:**

90 elderly patients age ≥60 admitted with typical signs and symptoms of congestive heart failure are included in this study.

**EXCLUSION CRITERIA:**

Patients with evidence of

1) Congenital heart disease,
2) Valvular heart disease,
3) Pericardial disease and
4) High output heart failure (severe anemia, Thyrotoxicosis, AV fistula) are excluded from this study

**METHODOLOGY:**

The study enrolled 90 patients admitted in Govt general hospital with features of congestive heart failure according to Framingham criteria. Age group selected for this study are ≥60yrs, individuals were characterized by detailed medical history, clinical examination, ECG,Chest Xray,echocardiography,and following lab tests:Hb,blood sugar,urea,serum creatinine,serum electrolytes,serum albumin and LDL. DHF was defined as

(1) Clinical signs of heart failure,
(2) Ejection fraction ≥50,
(3) Echocardiographic findings of diastolic dysfunction.

**1. Calculation of Ejection Fraction by two methods**

a) Teicholtz formula

b) Simpson's rule

2) **Dimension**

a) LA b) LV c)IVS d)RA e)RV

**3) Doppler Echocardiographic Finding Of diastolic Dysfuntion**

**TRANS MITRAL FLOW**

a) Assessment of Transmitral flow velocity during early diastole(E) and during atrial contraction(A)

b) E/A

c) Deceleration Time(DT)

d) Isovolumetric relaxation time(IVRT)

**PULMONARY VENOUS FLOW**

a) Systolic velocity(S)

b) Diastolic velocity(D)

c) Atrium to pulmonary veins during atrial contraction(AR)

4) Pulmonary Hypertension

5) Regional wall motion abnormality

6) Right ventricular Function

7) Valvular heart disease

**ANALYSIS**

Data analysed using statistical package-SPSS Software

# REVIEW OF LITERATURE

## Cardiovascular Changes Related to Aging

Knowledge of cardiovascular changes that occur with aging are important for several reasons:

- Recognition of these changes allow us to better distinguish normal cardiovascular aging from disease states in elderly.
- Manifestation of various cardiovascular disease vary between young and old patient, e.g. hypertension manifest as elevated diastolic pressure in young and systolic in elderly.
- Ability for an individual to compensate for their cardiovascular illness may be age dependent and thus an elderly patient may be more symptomatic for any burden of disease.
- Response to appropriate cardiovascular therapy may be age dependent, such as Beta adrenoreceptor blockade.

There have not been clear cut distinction between when an age related phenomenon becomes a disease related phenomenon, e.g, Left ventricular mass increase with increasing age and increasing left ventricular mass predicts future cardiovascular morbidity and mortality.

- Age-related changes in the cardiovascular system parallel age-related changes elsewhere in the body. However, age-related changes in a single organ or group of organs, such as the brain, lungs, or kidneys, may predominate, while, other organs remain unaffected.
- Age-related changes in the cardiovascular system are specific and enumerable. First, elderly hearts experience myocyte hypertrophy along with an increase in the connective tissue matrix[1]. On the cellular level, total number of myocytes decreases and remaining myocytes hypertrophy[2]. The weight of the heart increases 1.5 g/year between 30 and 90 years of age because of this hypertrophy and connective tissue deposition[3-4].
- With aging, ventricular chamber dimensions decrease with septal hypertrophy because of increased ventricular septal thickness and decreased base-to-apex dimensions. The sigmoid septum of aging is another morphologic manifestation of the senescent heart resulting from the reduction in the ventricular cavity size and rightward shift of the ascending aorta[5]. Although not hemodynamically significant, this curved septum simulates asymmetric hypertrophic cardiomyopathy.

- Despite structural changes, systolic function of the heart at rest remains essentially normal. However, alterations that impair peak systolic function do occur at the subcellular level of myocardial function. These include availability of energy stores, intracellular calcium handling, and transmembrane action potential[6].

## AGE-RELATED CHANGES IN CARDIAC ANATOMY

| | |
|---|---|
| **MYOCARDIUM** | Increased heart weight, LV mass, LV wall thickness Increased myocyte size, decreased myocyte number Fibrosis with deposition of less distensible form of collagen. |
| **CHAMBERS** | Decreased LV cavity size, shortened long axis Rightward shift and dilatation of the aorta Dilatation of left atrium, senile septum. |
| **VALVES** | Calcific and fatty degeneration of valve leaflets and annuli |
| **CORONARY ARTERIES** | Atherosclerosis Dilation, tortuosity, and Monckeberg's calcific arteriosclerosis. |
| **CONDUCTION SYSTEM** | Fibrosis of atrioventricular node and left anterior fascicle Loss of specialized cells and fibers. |

## AGE-RELATED CHANGES IN CARDIOVASCULAR PHYSIOLOGY

| | |
|---|---|
| **VASCULAR IMPEDANCE INCREASES IN LARGE AND MEDIUM-SIZE ARTERIES** | Pulse wave velocity and pulse pressure increases Systolic blood pressure increases Impaired endothelial function. |
| **MYOCARDIAL RELAXATION DECREASES** | Diastolic dysfunction develops Peak EF maintained by a larger diastolic volume Cardiac output and stroke volume are preserved at rest. |
| **ELECTRICAL CONDUCTION AND HEART RATE RESPONSE TO STIMULI IS IMPAIRED** | PR, QRS, and QT are prolonged Peak exercise HR declines Sensitivity to Beta agonists is decreased Reactivity to chemoreceptors and baroreceptors is diminished. |
| **INTEGRATED PERFORMANCE DURING CARDIOVASCULAR EXERCISE IS IMPAIRED** | Decrease peripheral muscle mass; increased adipose tissue Diminished respiratory capacity Diminished $VO_2$ max reserves |

- In contrast to systolic function, which is preserved at rest, diastolic function is impaired. Connective tissue matrix becomes replaced with a less distensible form of collagen. This causes greater stiffness of the senescent heart, requiring greater filling pressures to adapt via the Frank-Starling mechanism[9].

- Progressive cellular disarray, myocyte asynchrony, and abnormal calcium handling further affect the compliance and filling parameters during diastole. From a study of senescent animal models, the most predictable change in cardiac muscle function is longer duration of relaxation. This impaired relaxation with senescence is attributable to slower intracellular handling of calcium and longer action potentials, in addition to the stiffness from altered collagen. Echocardiographic Doppler studies in humans confirm prolonged relaxation and slower early diastolic filling with aging[10].

- However, end-systolic volumes are usually maintained by augmentation of late diastolic filling evidenced by exaggerated A wave and altered E:A ratio through the mitral valve[11]. Diastolic function of the aging heart may be worsened by coexisting structural changes, such as mitral or aortic valvular disease,

hypertension, atrial arrhythmias, or senile amyloidosis, which further alter hemodynamic conditions.

- Age-related changes in the arterial system begin in the 30s and accelerate through midlife. Increased collagen deposition and weakened vascular elastin result in altered elasticity, distensibility, and dilatation. These changes, particularly in the intima, appear to resemble those that occur during atherosclerosis[12-13]. Within the vascular media, there is progressive growth of smooth muscle, as well as deposition of lipids and calcium in the elastic lamella. Stiffening of the central arteries results in higher pulse wave velocities and augmentation in systolic arterial pressure, and whereas the lower elasticity results in a diminished contribution of arterial recoil to forward arterial perfusion. As arterial distensibility decreases, the speed of travel of the pulse along an arterial segment, referred to as the pulse wave velocity, increases. The forward cardiac ejection wave travels through central compliance arteries until it meets forward resistance. The pulse wave is then reflected (reflection wave), where it sums with continuing forward cardiac ejection increasing systolic pressures. Less compliant vasculature returns the

reflection wave sooner, making a greater contribution to systolic pressure. Cyclic fatiguing and elastase activity also result in a reduction and fragmentation of vascular elastin[14].

- Vascular remodeling therefore takes place and results in dilatation and elongation of the aorta and major arteries. These changes are accompanied by impaired endothelial function owing to reduced prostacyclin production by cells which remain. With age, the endothelium undergoes apoptosis, progressive irregularity in cell size and shape, and increased multinucleated giant cells. Endothelial-dependent responses to agonists such as acetylcholine are therefore impaired[15-16].

- Age-related changes in the conduction system result from apoptosis and the deposition of collagenous and fatty tissue. Fat accumulates around the sinoatrial node, sometimes producing partial or complete separation of the node from the atrial musculature. There is also a pronounced decrease in the number of pacemaker cells in the sinoatrial node beginning at age 60. At age 75, less than 10% of the cell number found in the young adult remains.

- Calcification of the atrioventricular node and left and right bundle branches also occur. Thus, older patients often have modest increases in electrocardiographic PR and QT intervals, increased QRS duration and bundle branch blocks, and decreased T-wave amplitude[17]. The maximum predicted heart rate (HR) in an octogenarian is 30 beats per minute lower than it was at age 50, and HR in older individuals is also less responsive to Beta-adrenergic stimulation.

- In the Framingham cohort, variability in RR intervals declines by 38% between age 40 and 70, reflecting the lesser contribution of autonomic tone to cardiac function with aging[18]. Altered autonomic regulation is also demonstrated by reduced heart rate variability (HRV) to head-up tilt testing and impaired baroreflex[19]. The Framingham study demonstrated the gradual increase in the prevalence of atrial fibrillation in the population between age 50 and 80[20]. In addition, there is an increase in ambient rate of premature atrial and ventricular contractions as evidenced by holter monitors in healthy adults. Short runs of supraventricular tachycardia occur in up to 33% of healthy individuals over age 60[21].

- Aerobic capacity ($VO_2$ max) declines with normal aging due to diminished cardiac reserve. Age-related changes in heart function must be differentiated from those resulting from a sedentary lifestyle or other disease processes. Many older patients become inactive, both physically and mentally, which accelerates deterioration and loss of function[22]. Early studies found a steady decline in overall cardiovascular performance with aging as judged from exercise training[23].

**DEFINITION**

Heart failure is defined as the pathologic state in which the heart is unable to pump blood at a rate required by the metabolizing tissues or can do so only with an elevated filling pressure. Inability of the heart to pump blood sufficiently to meet the needs of the body tissues may be attributable to the inability of the LV to fill(diastolic performance) or eject(systolic performance).When the heart failure is associated with a reduced EF, the pathologic state may be called systolic heart failure. In contrast, when the heart failure is associated with diastolic dysfunction in the absence of a reduced EF, the pathologic state is diastolic heart failure.

## EPIDEMIOLOGY

Thirteen epidemiological studies have defined the prevalence of DHF in various HF populations and have documented a prevalence of 50 to 55 percent. [10] [14] [15] [16] Importantly, the prevalence of DHF among patients with HF varies dramatically with age and gender. The prevalence of HF increases with age and is similar in men and women . The prevalence of HF with a depressed EF increases with age but is more common in men than in women at any age whereas the prevalence of DHF increases even more dramatically with age (more than HF with a reduced EF) and is much more common in women than in men at any age . More data are needed concerning variation in the prevalence of DHF in different regional and ethnic groups.

## NATURAL HISTORY

## MORTALITY

Most large contemporary studies have now suggested that the mortality for DHF is similar to that of HF with a reduced EF. [25] [29] [30] Differences in survival between the two forms of HF are minimal. Although survival has improved over time for patients with HF with a reduced EF, it has not changed for patients with DHF.[24] In the Digitalis

Investigation Group (DIG) study, which included patients with DHF,[25] the cause of death was somewhat different in patients with DHF, in whom deaths caused by non cardiovascular and non-HF cardiovascular mechanisms were more frequent.

## MORBIDITY

Patients with DHF have comparable morbidity to those with HF with a reduced EF, with similar or minimal differences in HF readmission rates.[30] Rates of progressive functional decline after an admission for HF are also similar in patients with preserved or reduced EF.[26]

## CAUSES OF DIASTOLIC DYSFUNCTION AND HEART FAILURE

### Common causes

Cardiac ischemia

Hypertension

Aging

Obesity

Aortic stenosis

**Uncommon causes**

Myocardial disorders

Myocardial diseases

Infiltrative disease (e.g., amyloidosis, sarcoidosis, fatty infiltration)

Noninfiltrative diseases (e.g., idiopathic and hypertrophic cardiomyopathy)

Endomyocardial diseases

Hypereosinophilic syndrome

Storage diseases

Glycogen storage disease

Hemochromatosis

## CLINICAL FEATURES

Patients with DHF were shown to have similar pathophysiological characteristics compared with HF patients with a reduced EF including severely reduced exercise capacity, neuroendocrine activation, and impaired quality of life despite normal EF, normal left ventricular (LV) volume, and an increased LV mass-to-volume ratio.[27]

## Clinical Features of Diastolic Heart Failure

| Parameter | Features |
|---|---|
| Framingham criteria for diagnosis of heart failure[*] | |
| Major criteria | Paroxysmal nocturnal dyspnea or orthopnea |
| | Jugular venous distention (or CVP > 16 mm Hg) |
| | Rales or acute pulmonary edema |
| | Cardiomegaly |
| | Hepatojugular reflex |
| | Response to diuretic (weight loss >4.5 kg in 5 days) |
| Minor criteria | Ankle edema |
| | Nocturnal cough |
| | Exertional dyspnea |
| | Pleural effusion |
| | Vital capacity < two thirds of normal |
| | Hepatomegaly |
| | Tachycardia (>120 bpm) |
| Demographic features | Elderly; female > male |
| Underlying CV disease | Hypertension, coronary disease, diabetes, atrial fibrillation |
| Comorbidities | Obesity, renal dysfunction |
| Doppler echocardiography results | |
| LV size | Normal to ↓ (small subset with↑) |
| LV mass | LVH common but *frequently* absent; ↑ relative wall thickness (> 0.45) |
| Left atrium | Enlarged |
| Diastolic dysfunction | Grade I-IV (∞ diastolic dysfunction severity, BP, volume status) |
| Other features | PH, wall motion abnormality, RV enlargement |
| Pertinent negatives | Rule out valve disease, pericardial disease, ASD |
| BNP or NT-proBNP | ↑ but HFnlEF < HFrEF |
| Exercise testing | ↓ $VO_2$ peak |
| | Exaggerated hypertensive response in many |
| | Chronotropic incompetence in subset |
| Chest radiogram | Similar to HFrEF, cardiomegaly, pulmonary venous hypertension, edema, pleural effusion |
| Electrocardiogram | Variable |

## RISK FACTORS

Patients with DHF are generally older than age 65 years, with many older than 80, and are predominantly women (60 to 70 percent). A history of hypertension is present in most (60 to 80 percent) and may have developed only later in life. Obesity is seen in 30 to 50 percent of patients, diabetes in 30 to 50 percent, and atrial fibrillation in up to 20 to 40 percent. The prevalence of renal disease is high and similar to that noted in patients with HF and a reduced EF. The reported medications at diagnosis in patients with DHF have included diuretics, digoxin, angiotensin-converting enzyme (ACE) inhibitors, beta blockers, calcium channel blockers and various other vasodilators, and antihypertensive and antiarrhythmic drugs. [25] [29] [30] [16] The reported prevalence of coronary artery disease varies widely.[29]

## AGING

Although cardiovascular disease may contribute to diastolic dysfunction in older people, studies have also suggested that diastolic function deteriorates with normal aging.[30] The speed of LV relaxation declines with age in men and women, even in the absence of cardiovascular disease.[30] It also appears that vascular, LV systolic, and

LV diastolic stiffness increase with aging. Increases in vascular stiffness have been shown to be related to effort intolerance in patients with DHF. Structural cardiac changes with aging (e.g., increased cardiomyocyte size, increased apoptosis with decreased myocyte number, altered growth factor regulation, focal collagen deposition) and functional changes at the cellular level involving blunted beta-adrenergic responsiveness, excitation-contraction coupling, and altered calcium-handling proteins may contribute to diastolic dysfunction with normal aging. Some studies have suggested that prolonged, sustained endurance training may preserve LV compliance with aging and help prevent HF in the elderly.[18]

**GENDER**

Along with age, female gender is a potent risk factor for DHF. Indeed, there appears to be important age-gender interactions, such that the prevalence of DHF increases more sharply with age in women than the prevalence of HF with a reduced EF . The reasons for the female predominance in DHF are not entirely clear, but women have higher vascular and LV systolic and diastolic stiffness than men, and vascular and ventricular stiffness increases more dramatically with age in

women.[30] Emerging evidence of unique coronary vascular functional changes in women may play a role in DHF pathophysiology.

## HYPERTENSION

Hypertension is the most commonly associated cardiac condition in patients with DHF. Chronically increased blood pressure is an important stimulus for cardiac structural remodeling and functional changes. The resultant hypertensive heart disease is characterized by LV hypertrophy (LVH), increasing vascular and ventricular systolic stiffness, impaired relaxation, and increased diastolic stiffness, all factors linked to the pathogenesis of DHF. In the presence of hypertensive heart disease, ischemia produces exaggerated increases in filling pressures, and hypertensive and ischemic heart disease are often present in combination in patients with DHF. Elucidating which factors mediate transition to DHF in persons with hypertensive heart disease is an area of active investigation. [29-30]

## CORONARY ARTERY DISEASE.

The reported prevalence of coronary artery disease or myocardial ischemia in patients with DHF varies widely. [29-30] Although acute ischemia is known to cause diastolic dysfunction , the role of coronary

artery disease and ischemia in contributing to chronic diastolic dysfunction and symptoms in patients with DHF remains speculative. Despite uncertainty regarding the role of ischemia in the pathophysiology of DHF and a lack of data documenting that revascularization improves outcomes in patients with DHF, HF management guidelines recommend revascularization in those DHF patients in whom ischemia is felt to contribute to diastolic dysfunction. [26-27] Importantly, emerging evidence suggests that unique coronary vascular functional changes are present in women.

## ATRIAL FIBRILLATION AND OTHER RHYTHM DISTURBANCES

Atrial fibrillation is recognized as a frequent precipitant of acute decompensation in patients with DHF. Whereas atrial fibrillation may cause decompensated HF in patients with diastolic dysfunction, diastolic dysfunction (in the absence of HF) is also a risk factor for atrial fibrillation. Thus, diastolic dysfunction, atrial fibrillation, and DHF are common and related conditions that probably share common pathogenic mechanisms in the elderly. The prevalence of ventricular arrhythmias in DHF is poorly defined. Although tachycardia caused by atrial arrhythmias is a recognized precipitant of acute decompensation in

DHF, bradycardia and adverse atrioventricular timing caused by first-degree heart block may also adversely affect LV filling in some patients.

## OBESITY

Obesity is associated with an increased risk for HF. In general, patients with DHF are more often obese than patients with HF with a reduced EF, [29-30]and the prevalence of diastolic dysfunction is increased in obese persons. Increased adiposity not only imposes an adverse hemodynamic load on the heart but is also a source of a large number of biologically active peptide and nonpeptide mediators, many linked to chronic inflammation by various pathways. Increased body mass index (BMI) is a risk factor for hypertension, diabetes mellitus, coronary artery disease, and atrial fibrillation, all of which are associated with DHF. Studies using tissue Doppler imaging or invasive LV pressure measurement have reported an association between diastolic dysfunction, elevated filling pressures, and obesity.

## DIABETES MELLITUS

Diabetes is a potent risk factor for HF, and the prevalence of diabetes is similar in patients with HF and reduced or preserved EF, suggesting that diabetes contributes to the pathophysiology of both

forms of HF. Although diabetes predisposes to coronary artery disease, renal dysfunction, and hypertension, numerous direct effects of diabetes and hyperglycemia on myocardial structure and function have been described. The morphological changes in the diabetic heart include myocyte hypertrophy, increased extracellular matrix (fibrosis), and intramyocardial microangiopathy. Functional changes, which may represent a continuum, include endothelial-dependent and endothelial-independent microvascular dysfunction, impaired relaxation, and increased passive diastolic stiffness and contractile dysfunction. Mechanisms contributing to structural and functional coronary vascular and myocardial changes are diverse and include metabolic disturbances, activation of proinflammatory and profibrotic mediators, cardiac autonomic neuropathy, and increases in advanced glycation end-products (AGE), which promote increased collagen accumulation and increased collagen stiffness. AGE accumulation may also play a role in age-related cardiovascular stiffening.

**RENAL DYSFUNCTION**

The critical impact of renal function on morbidity and mortality in HF is well established. Studies have shown no difference between the severity of renal dysfunction in patients with reduced

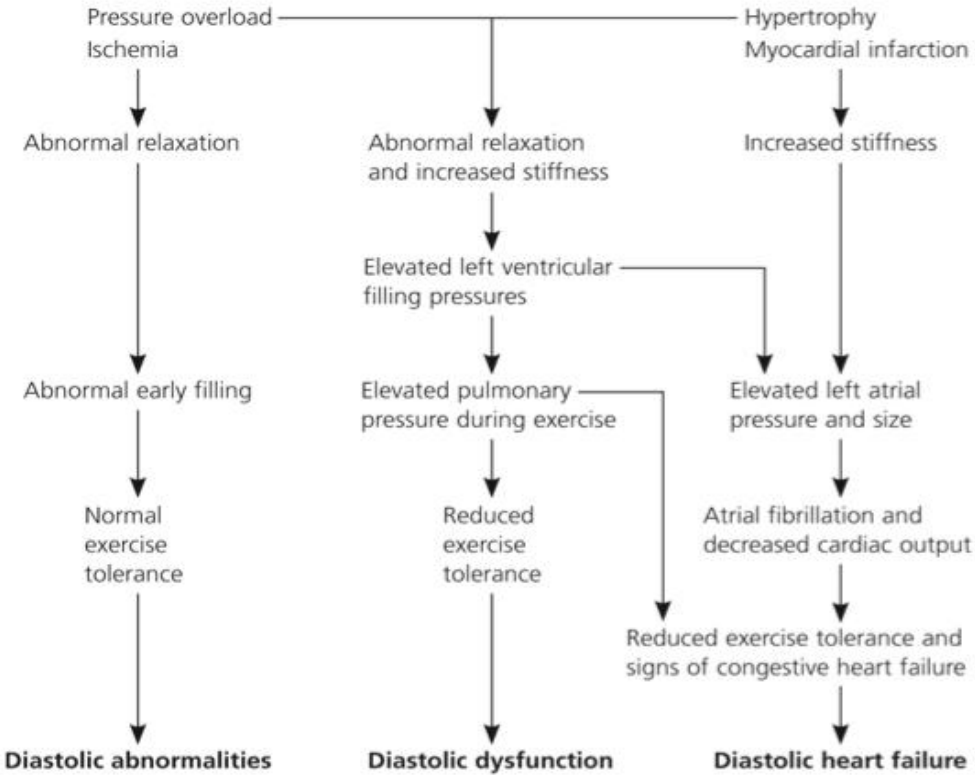
or preserved EF.[29-30] Furthermore, the incidence of worsening renal function during HF therapy is similar in patients with preserved or reduced EF.Although the prevalence of renal vascular disease in HF has been poorly delineated, it is probably high, and bilateral renal artery stenosis with rapid-onset or flash pulmonary edema is a well-recognized cause of DHF. Evaluation of the renal arteries should be considered in patients presenting with the triad of hypertension, renal dysfunction, and DHF.

**RARER CAUSES OF DIASTOLIC HEART FAILURE**

Hypertrophic cardiomyopathy, infiltrative cardiomyopathies such as amyloidosis, valvular disease, and constrictive pericarditis should always be considered in young patients with DHF or patients with other suggestive features. However, these diseases account for a minority of patients with DHF. Idiopathic restrictive cardiomyopathy in young persons without the above mentioned factors may represent a distinct group, particularly if a family history is present. However, the clinical presentation and echocardiographic appearance in older persons with DHF may be identical to those of patients previously labeled as having restrictive cardiomyopathy. An important consideration in patients with previous malignancy treated with mediastinal radiation is radiation heart

disease. Radiation can cause pericardial and concomitant myocardial damage, and outcomes after pericardiectomy are frequently poor because of concomitant restrictive myocardial disease. Concomitant valvular disease and premature coronary artery disease are also common in patients with previous mediastinal radiation and may contribute to the pathophysiology of DHF in patients with radiation heart disease.

## PATHOPHYSIOLOGY OF DIASTOLIC HEART FAILURE




Algorithm for pathophysiology of diastolic heart failure.

In the clinical definition most often used, diastole is delineated by the onset of isovolumic relaxation and closure of the mitral valve. Applying this definition, ventricular relaxation is included in diastole, which is understandable since active myocardial relaxation influences ventricular pressures during early and mid-diastole. Diastole is traditionally divided into four phases,i.e. isovolumic relaxation, early diastolic filling, diastasis and atrial contraction. In all phases, a multitude of factors determines LV filling with a varying relative importance. These factors overlap in time[31], and are influenced by each other, by LV systolic function, heart rate, and by the cardiac conduction system. Their final combined effect is on the transmitral pressure gradient, which actually determines LV filling.

**Isovolumic relaxation**

Myocardial relaxation requires dissolution of the force-generating crossbridges between myosin and actin. Myocardial sarcomeres generate force under the direct control of calcium[32], and prompt relaxation demands the reduction of myocyte cytosolic calcium to its physiologically low concentrations. This is achieved by a sarcoplasmic reticulum calcium transporting ATPase that pumps calcium ions against a concentration gradient back into storage sites within the sarcoplasmic reticulum.In addition, sodium–calcium exchangers and sarcolemmal

calcium pump transport calcium outward across the sarcolemma. Since myofilaments may sustain active cross bridge connections beyond the time that cytosolic free calcium has been reduced to its diastolic concentration in the normal myocardium, mechanical relaxation will be limited by the myofilaments per se, not the calcium exchange rate. In failing human heart, both myofilaments and calcium exchange pumps act more slowly, i.e. an attenuation of calcium sensitivity develops. The relative timing of crossbridge dissociation and calcium uptake may alter and the removal of calcium may become rate limiting, causing LV relaxation to decelerate[33]. Since calcium reuptake is an energydependent process, relaxation may thus become abnormal early in several cardiac disease states; ischemia,hypertrophy and heart failure will all lead to slowing of LV relaxation[34-36].

**Early diastolic filling**

Early rapid filling of the LV starts with the first reversal of the atrioventricular pressure gradient opening the mitral valve and accelerating the blood into the ventricle. An explosive period of filling ensues during which the largest proportion (80–85%) of total LV filling occurs under physiological conditions. Filling velocity reaches its maximum when the atrioventricular pressure gradient reverses again, although fluid inertia will cause LV filling to continue. Finally, LV

filling decelerates to a minimum until a third pressure gradient reversal occurs at the beginning of diastasis[37]. Thus, the instantaneous atrioventricular pressure gradient determines flow during the rapid filling phase. The acceleration of early flow will be determined by the rate of ventricular relaxation and left atrial pressures resulting from loading conditions and atrial and pulmonary vein compliance[38].

**Diastasis and Atrial Contraction**

Diastole further encompasses diastasis and the atrial contraction phase. On a time scale, their duration mainly depends on heart rate and on the duration of systole,and at normal heart rates, this will correspond to approximately 50% of the total duration of the cardiac cycle. On a volume scale however, only the last 5–15% of ventricular filling occurs during diastole. Once deceleration of early rapid filling flow is complete, LV filling enters a phase during which only a small additional volume is slowly added to the LV, accompanied by a very gradual rise in LV pressure[39]. In young volunteers, only 6% of total LV filling volume is added during diastasis in approximately 180ms, and presently, mechanisms operating during diastasis are not considered important in overall diastolic function.

The left atrium acts as a reservoir during systole, storing blood at a certain pressure which is determined by left atrial compliance, blood

that is subsequently supplied to the ventricle during diastole. The atrial contraction has a booster effect on LV filling, contributing approximately 15% of the total LV filling volume. The effectiveness of the atrial contraction is dependent on LV compliance (i.e. atrial afterload), but also atrial preload, heart rate, atrial contractility and atrial geometry. Particularly at increased heart rates, during exercise, or in case of impaired LV function, atrial contribution may be increased, thus augmenting LV filling and, through the Frank–Starling mechanism, increasing cardiac output[40]. Increase of atrial reservoir and pumping capacity will act as a first mechanism in response to impairment of LV filling in the failing heart. Eventually, left atrial dilatation and atrial afterload mismatch will result in ineffective atrial systolic function, rendering the atrium to a mere conduit for flow.

**Diastolic heart failure**

Conceptually, the causes of diastolic dysfunction may be subdivided into a decrease in passive myocardial diastolic compliance, and an impairment in active LV relaxation. A variety of myocardial and pericardial disorders may provoke an upward shift of the diastolic portion of the LV pressure–volume or pressure–dimension relation, resulting in relatively large changes in LV diastolic pressures for relatively small volume changes. It is the shift in LV pressure volume relation that causes the symptoms of pulmonary congestion

(i.e. dyspnea), first occurring during exercise, later also in rest. Patients with diastolic dysfunc tion may be unable to normally augment stroke volume e.g. during exercise, even in the setting of increased ventricular filling pressure[41].

In systolic heart failure, a downward and rightward shift of the end-systolic pressure-volume line indicates decreased contractile function, which is the principal cause of reduced ejection fraction and forward stroke volume. In primary diastolic heart failure, diastolic pressure-volume relation (dashed line) shifts upward and to the left, indicating a disproportionate and a greater increase in diastolic pressure for any increase in diastolic volumes. If there is also a decrease in end-diastolic volume, then a decrease in stroke volume also occurs

## METHODS OF DIAGNOSING DIASTOLIC HEART FAILURE

### Findings on Doppler Echocardiography

Comprehensive Doppler echocardiography is invaluable in the evaluation of HF patients. Although other cardiovascular imaging techniques may provide as good or even more accurate assessment of cardiac structure or function, the widespread availability and comprehensive functional information provided by echocardiography make it the test of choice for patients with HF.

## LV Size

Most patients with DHF have normal chamber dimensions, although a subset will have variable degrees of LV enlargement.[44]

## LV Hypertrophy

Although DHF has been thought to occur primarily in patients with LVH, studies that have carefully quantified LV mass report that echocardiographic criteria for LVH are met in less than 50 percent of patients.[46] Patients with DHF have, on average, increased relative wall thickness and an increased mass-to-volume ratio,[44] but these findings often occur in the setting of normal LV mass. Thus, LVH is not invariably present in DHF, in which the cardiac phenotype is variable. There is some evidence that the prevalence of LVH may be higher in African American DHF patients.

## Doppler Echocardiographic Assessment of Diastolic Function and Filling Pressures

Early studies focused on the transmitral flow velocity profile. Decreases in the ratio of early to late diastolic filling (E/A), increases in the deceleration time (DT) of the early diastolic filling velocity profile, or increases in the isovolumic relaxation time (IVRT) indicate impaired relaxation. However, in the presence of impaired relaxation, increases in

filling pressure progressively modify the transmitral gradient and mitral inflow pattern. A comprehensive Doppler assessment is used to determine diastolic function and filling pressures. Patients studied at various times during their presentation (e.g., acutely decompensated, after initial treatment of an acute decompensation episode, or as stable outpatients) will display a spectrum of filling patterns, including abnormal relaxation and pseudonormal or restrictive patterns. Such a spectrum has also been reported in patients with HF with a depressed EF and reflects the potent effect of filling pressures, blood pressure, and their interaction with underlying diastolic dysfunction on the Doppler patterns.

**Left Atrial Enlargement**

Increases in left atrial dimension or volume is commonly, if not almost uniformly, present in patients with DHF[46]

**Pulmonary Hypertension**

Just as chronic pulmonary venous hypertension leads to pulmonary arterial hypertension in HF with depressed systolic function, the same can occur in DHF, and an elevated tricuspid regurgitant velocity indicative of pulmonary hypertension is common in DHF. Chronic pulmonary venous hypertension in HF has a direct

hemodynamic effect on pulmonary pressure and causes reactive pulmonary hypertension, which can be slow to resolve after normalization of LV filling pressures. With time, chronic pulmonary venous hypertension causes pulmonary vascular remodeling (congestive pulmonary vasculopathy) and irreversible pulmonary hypertension. These changes are well described in HF with a reduced EF and mitral valvular disease but can occur in DHF as well. The prevalence of significant pulmonary hypertension in DHF is as high as 50 percent.[42]

**Other Doppler Echocardiographic Findings**

Regional wall motion abnormalities (with preserved EF) and right ventricular dilation, either because of ischemic disease or pulmonary hypertension, can also be present at echocardiography in patients with DHF.[26] Additional important negative findings to be considered at echocardiography include the absence of valvular disease important enough to cause the HF symptoms, pericardial tamponade or constriction, or the presence of congenital heart diseases such as atrial septal defect or other more extensive structural abnormalities.

Proposed progression of diastolic function abnormalities as assessed with comprehensive Doppler echocardiography, with correlation of invasively measured diastolic properties.
A comprehensive Doppler assessment can yield useful information

regarding relaxation, filling pressures, and (indirectly) diastolic stiffness in most patients but requires careful data acquisition and informed interpretation.

**Natriuretic peptides**

BNP is produced in the myocardium in response to an increase in ventricular diastolic stretch, and its secretion results in natriuresis, vasodilatation, and improved ventricular relaxation. In patients with DHF, BNP values correlate with indices that evaluate the early and late LV diastolic relaxation. High values have been observed in patients with late or abnormal relaxation. Since BNP levels may be influenced by different conditions (sepsis, liver failure, kidney failure, COPD, obesity), high BNP values do not provide sufficient evidence for the diagnosis of diastolic dysfunction, requiring additional tests for the diagnosis of DHF, a high positive predictive value was determined when the BNP cut-off point was chosen (200 pg/ml). For the exclusion of HFNEF, a high negative predictive value was determined for the choice of the BNP cut-off point (<100 pg/ml)5.

Therefore, natriuretic peptides are mainly recommended for exclusion, and not for diagnosis of DHF. Since BNP alone does not provide evidence for the diagnosis of DHF, it should always be used with other non-invasive tests.

## Exercise Testing

Cardiopulmonary exercise testing (CPET) is useful in the diagnostic evaluation of exercise intolerance of unclear cause and thus, in select patients with suspected DHF in whom concern over pulmonary limitations also exist, CPET may play a role in the diagnostic evaluation. Objective measures of exercise tolerance are similarly impaired in HF (of similar clinical severity) with reduced and preserved EF.[12] Although the prognostic features of metabolic stress testing are less well described and less relevant in the DHF population, few of whom are considered for cardiac transplantation, standard exercise stress testing without metabolic measurements may be useful in the evaluation and management of patients with DHF. Exaggerated hypertensive responses to exercise causes load-dependent diastolic dysfunction and recognition, and treatment of exercise-induced hypertension is important in the treatment of DHF. Importantly, stress testing also allows assessment of the heart rate response to exercise. Whereas past recommendations have focused on the potential use of negative chronotropic agents to allow a longer diastolic filling period in patients with impaired relaxation, recent studies have suggested that chronotropic incompetence is also relatively common (higher than 20 percent) in patients with DHF(although not more common than in HF with a reduced EF), even in the absence of beta blocker therapy.

## DIAGNOSTIC CRITERIA FOR DIASTOLIC HEART FAILURE

### Previous and ongoing studies

Vasan et al. reviewed 31 studies of CHF with normal LV systolicfunction published between 1970 and 1995,mostly clinical comparative studies, mostly hospital based. Apart from a striking lack of uniformity in the criteria for congestive heart failure that were applied in these studies, they found that only three of 31 studies provided details regarding the exclusion of alternative explanations for dyspnea, which is essential, since the signs and symptoms of congestive heart failure are nonspecific. In addition, only six of 31 studies assessed ventricular diastolic function in a satisfactory manner, whereas in most studies the mere presence of a normal LV systolic function was considered synonymous with diastolic heart failure. In these studies, a LV ejection fraction above a certain cut-off point (e.g. 45%) was used as inclusion criterion. Vasan et al. concluded, that the lack of consensus and subsequent heterogeneity in previous studies emphasize the need for prospective and uniform evaluation of patients with diastolic heart failure to better characterize epidemiology and natural history, as well as optimal treatment. In two ongoing studies of ACE inhibitors in diastolic heart failure, the investigators have already provided specified reports of

the in- and exclusion criteria, which will be applied to patients to allow for participation. In the PEP-CHF study, perindopril will be compared to placebo in elderly patients with chronic heart failure in the absence of any major LV systolic dysfunction[47].

The investigators aim to recruit one thousand patients over the age of 70 years into their study, and will follow-up on these patients for at least one year. Primary end-point of the study is the time to first occurrence of a combined end-point of total mortality and unplanned heart failure related hospital admission. The latter includes hospitalization due to an increase in severity of the heart failure signs or symptoms, hospitalization for declining renal function or acute myocardial ischemic events or arrhythmias that are associated with worsening of heart failure, as well as admissions because of other problems, such as infections, that lead to an exacerbation of congestive heart failure. Secondary outcome measures in this study include death, an increase in diuretic treatment of over 40 mg furosemide per day (or equivalent) or new initiation of combination therapy with thiazide diuretics.

**PRIMARY DIASTOLIC HEART FAILURE CRITERIA PROPOSED BY EUROPEAN STUDY GROUP ON DIASTOLIC HEART FAILURE**

**1. Presence of signs or symptoms of congestive heart failure**

Exertional dyspnoea if possible objectivated by reduced peak exercise oxygen consumption (< 25 ml/kg/min), orthopnoea, gallop sounds, pulmonary edema

**2. Presence of normal or mildly reduced left ventricular (LV) systolic function**

Baseline LV ejection fraction >/=45% in the presence of LV end-diastolic internal dimension index of < 3.2 cm/m2 or LV end-diastolic volume index <102 ml/m2

**3. Evidence of abnormal LV relaxation, filling, diastolic distensibility and stiffness**

(a) Slow IVRT

LV d*p*/d*t(*min)<1100 mmHg/s and/or

IVRT (<30yrs)>92 ms, IVRT(30-50yrs)>100 ms,

IVRT(>50yr)>105 ms and/or t>48 ms and/or

(b) Slow early ventricular filling PFR<160 ml/s/m2 and/or PFR (<30y)< 2.0 EDV/s, PFR(30-50yrs)< 1.8 EDV/s, PFR(>50yrs)<1.6 EDV/s and/or E/A (<50yrs)< 1.0 and DT(<50yr)>220 ms, E/A

(>50yr)< 0.5 and DT(>50yr)>280 ms and/or S/D(<50yrs)>1.5, S/D(>50yr)>2.5 and/or

(c) Reduced LV diastolic distensibility

LVEDP>16 mmHg or

Mean PCWP>12 mmHg and/or

PV A flow>35 cm/s and/or

PV A flow velocity duration>MV A flow velocity duration+30 ms And/or A/H>0.20

(d) Increased LV chamber or muscle stiffness

*b*>0.27 and/or *b*'>16

LV, left ventricular; IVRT, iso-volumic relaxation time; PFR, peak filling rate; DT, deceleration time; S/D, ratio of pulmonary vein systolic and diastolic flow velocities; EDV, end-diastolic volume; EDP, end-diastolic pressure; PCWP, pulmonary capillary wedge pressure; PV A flow, pulmonary venous atrial flow; MV A flow, mitral atrial flow; A/H, ratio of atrial wave to total signal wave excurion on the apex cardiogram; *b*, constant of LV chamber stiffness; *b*9, constant of muscle stiffness. in symptom score, quality of life or NYHA heart failure score.

The PEP-CHF study requires clinical and echocardiographic evidence of cardiac dysfunction rather than the mere absence of systolic dysfunction for inclusion. A first report on the ongoing screening process reports of considerable numbers of patients excluded because of comorbidity and significant systolic dysfunction. This suggests, that the use of positive echo criteria for diastolic dysfunction may lead to a lower proportion of patients with a diagnosis of diastolicheart failure than in previously reported series. However, as the research group comments, the need for studies of the elderly heart failure population with its comorbidity and functional impairments remains irrefutable[48].

The Hong Kong Diastolic Heart Failure study has already started in May 1999, and includes patients who have clinical evidence of heart failure documented by typical symptoms and signs, and radiological evidence of pulmonary venous congestion, combined with a LV ejection fraction over 45% as measured by echocardiography[49]. Patients are randomized to diuretics alone, diuretics plus ramipril, or diuretics plus irbesartan. The investigators also plan to recruit approximately 1000 patients, 300 in each group. The primary end-point is again a combination of mortality and episodes of hospitalization, with secondary end-points being quality of life and exercise capacity as measured by a validated questionnaire and 6-min walking tests, respectively.

## DIASTOLIC HEART FAILURE CRITERIA PROPOSAL

**1. Definite diastolic heart failure is**

(a) Definite evidence of congestive heart failure

Clinical symptoms and signs, supporting chest X-ray, typical clinical response to diuretics With or without elevated LV filling pressure or low cardiac index and

(b) Objective evidence of normal LV systolic function in proximity of event LV EF>/=50% within 72 h of event and

c) Objective evidence of LV diastolic dysfunction

Abnormal LV relaxation/filling/distensibility indices on catheterization

**2. Probable diastolic heart failure is**

(a) As definite and

(b) As definite and

(c) No conclusive information on LV diastolic function Patients fullfilling criteria 1 and 2 are accepted as having probable diastolic heart failure, provided that mitral valve disease, corpulmonale, primary volume overload and non-cardiac causes are excluded.

**3. Possible diastolic heart failure is**

(a) As definite and

(b) LV EF>/=50%, not measured within 72 h of event and

(c) No conclusive information on LV diastolic function

The diagnostic probability for diastolic heart failure may increase if the clinical setting is typical for the presence of LV diastolic dysfunction. According to the authors, this would apply to a patient with a markedly elevated blood pressure during congestion, or a patient with moderate concentric hypertrophy without concomitant wall motion abnormalities on echocardiography, or if tachy-arrhythmia such as atrial fibrillation accompany congestive heart failure, or if congestive heart failure occurs after the administration of small amounts of intravenous fluid in a patient with normal LV ejection fraction. Diastolic heart failure may also become more likely if the heart failure improves with treatment directed at the underlying cause of diastolic dysfunction, such as lowering blood pressure, controlling a rapid heart rate, or restoration of atrioventricular synchrony.

**TREATMENT**

In general, the management of DHF has two objectives. The first is to treat the presenting syndrome of HF—relieve venous congestion and eliminate precipitating factors. The second is to reverse the factors responsible for diastolic dysfunction or other perturbations

that lead to DHF. Both nonpharmacological and pharmacological strategies may be used to achieve these objectives. Present treatment strategies for DHF are largely based on assumptions regarding its pathophysiological mechanisms and on extrapolations from proven strategies used in HF with a reduced EF.

**Goals for Treating Diastolic Heart Failure**

Treat precipitating factors and underlying disease.

Prevent and treat hypertension and ischemic heart disease.

Surgically remove diseased pericardium.

Improve left ventricular relaxation.

ACE inhibitors

Calcium channel blockers

Regress left ventricular hypertrophy (decrease wall thickness and remove excess collagen).

ACE inhibitors and ARBs

Aldosterone antagonists

Beta blockers

Calcium channel blockers

Maintain atrioventricular synchrony by managing tachycardia (tachyarrhythmia).

Beta blockers (preferred)

Calcium channel blockers (second-line agents)

Digoxin (controversial)

Atrioventricular node ablation (rare cases)

Optimize circulating volume (hemodynamics).

ACE inhibitors

Aldosterone antagonists (theoretical benefit)

Salt and water restriction

Diuresis, dialysis, or plasmapheresis

Improve survival.

Beta blockers

ACE inhibitors

Prevent relapse by intensifying outpatient follow-up.

Control blood pressure.

Dietary counseling (sodium)

Monitoring volume status (daily weights and diuretic adjustment)

Institute exercise program.

**Nonpharmacological Therapy**

General measures that may be used in the management of patients with chronic DHF are not different from those pursued in patients with HF with a reduced EF. They include daily monitoring of weight, attention to diet and life style, patient education, and close medical follow-up. In patients with DHF, aggressive control of

hypertension, tachycardia, and other potential precipitants for HF decompensation should be emphasized.[52]Although there are no adequate clinical trials with appropriate outcome endpoints, such as increased longevity, decreased symptoms, or improved quality of life, to prove the benefits of exercise training in patients with DHF definitively, several clinical and experimental studies have suggested that exercise training would be beneficial for such patients. [11] [12] [27]

**Medical and Surgical Therapy**

In contrast to the treatment of HF with reduced EF, information to guide the pharmacological therapy of patients with DHF are lacking.

**CLINICAL STUDIES**

Small controlled studies have been performed using various standard HF drugs in patients with DHF. The drugs used have included ACE inhibitors, angiotensin receptor antagonists, beta blockers, and calcium channel blockers. These trials have, however, been small or have produced inconclusive results.[53]

## RANDOMIZED CONTROLLED CLINICAL TRIALS

The Digitalis Investigators Group (DIG) Trial included a small subgroup of patients with DHF. In the DHF group, digoxin did not alter the primary endpoint of HF hospitalization or cardiovascular mortality but did reduce HF hospitalizations.[54]

In the CHARM-Preserved Trial,[55] HF patients with an EF higher than 40 percent were randomized to candesartan (an angiotensin receptor antagonist) or placebo in addition to standard therapy. Fewer patients in the candesartan group than in the placebo group reached the primary endpoint of cardiovascular death or HF hospitalization, a finding that reached statistical significance only after adjusting for nonsignificant differences in baseline characteristics

In the PEP-CHF trial, patients older than 70 years with chronic HF and normal or near-normal EF were randomized to perindopril (an ACE inhibitor) or placebo.[56] The primary endpoint was a composite of all-cause mortality or unplanned HF-related hospitalization. Both enrollment and event rates were lower than anticipated and there was a high rate of cessation of blinded therapy, with crossover to open-label ACE inhibitor use in both groups. These factors limited the strength of

the study, which did not show significant reduction in the primary endpoint. Some trends toward benefit, primarily driven by reduction in HF-related hospitalizations, were observed at 1 year, when crossover therapy rates were lower.

The SENIORS trial tested the effect of the $beta_1$-selective blocker nebivolol in patients with HF.[57] Nebivolol also has vasodilator properties thought to be related to its effects on nitric oxide release. This trial was not restricted to those with normal EF. There was a modest but significant reduction in the primary endpoint of all-cause mortality or cardiovascular hospitalizations which was driven primarily by the effect on hospitalizations. Prespecified subgroup analysis in patients with EF > vs < 35 percent did not detect any trends toward reduced benefit in those with higher EF. Unfortunately, there were very few patients with EF > 50 percent in the trial.

Ongoing clinical trials in DHF are testing the efficacy of the endothelin antagonist, sitaxsentan sodium, irbesartan (I-PRESERVE trial), combinations of diuretics, ramipril, and irbesartan (Hong Kong Diastolic Heart Failure study), aldosterone antagonists (TOPCAT trial), and nesiritide.

## CURRENT THERAPEUTIC RECOMMENDATIONS

It is important to treat other contributing comorbidities and risk factors aggressively, such as diabetes, hyperlipidemia, renal dysfunction, and renal vascular disease. One retrospective study has shown that statin use, but not the use of beta blockers, ACE inhibitors, or calcium channel blockers, is associated with improved survival in patients with DHF.[58] Until more clinical trials are performed in patients with DHF, the empirical nature of therapeutic recommendations and their uncertain benefit must be recognized.

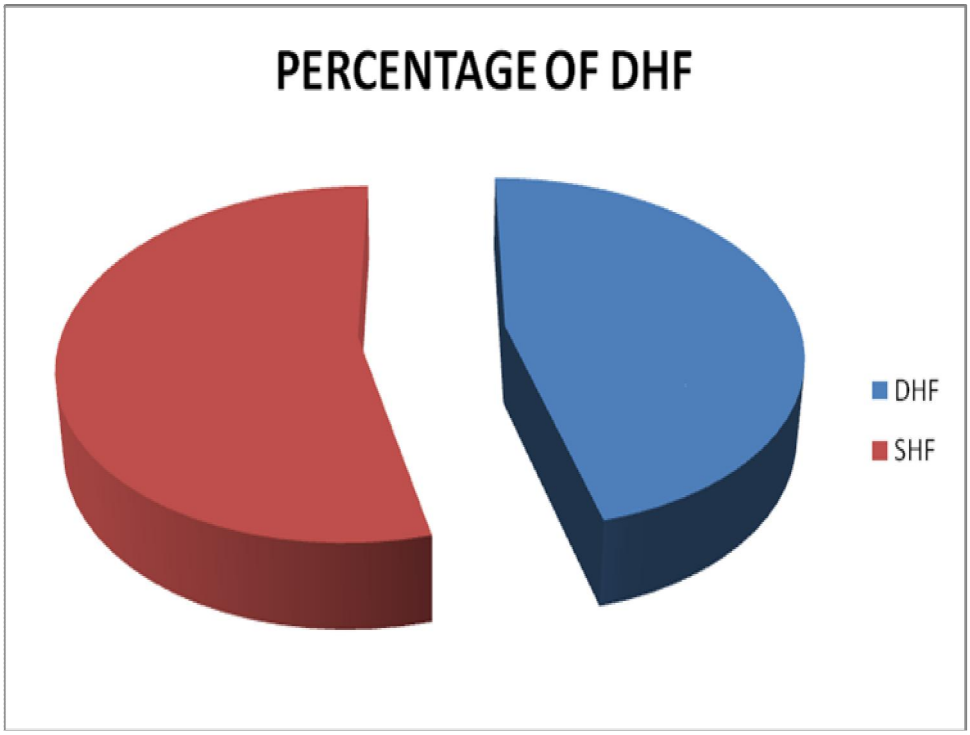
**Recommendations for Treatment of Patients with Heart Failure with Normal Ejection Fraction**

| Class | Recommendation | Level of Evidence |
|---|---|---|
| I | Physicians should control systolic and diastolic hypertension, in accordance with published guidelines. | A |
| I | Physicians should control ventricular rate in patients with atrial fibrillation. | C |
| I | Physicians should use diuretics to control pulmonary congestion and peripheral edema. | C |
| IIa | Coronary revascularization is reasonable in patients with coronary artery disease in whom symptomatic or demonstrable myocardial ischemia is judged to be having an adverse effect on cardiac function. | C |
| IIa | Restoration and maintenance of sinus rhythm in patients with atrial fibrillation might be useful to improve symptoms. | C |
| IIb | The use of digitalis to minimize symptoms of heart failure is not well established. | C |

# RESULTS AND OBSERVATION

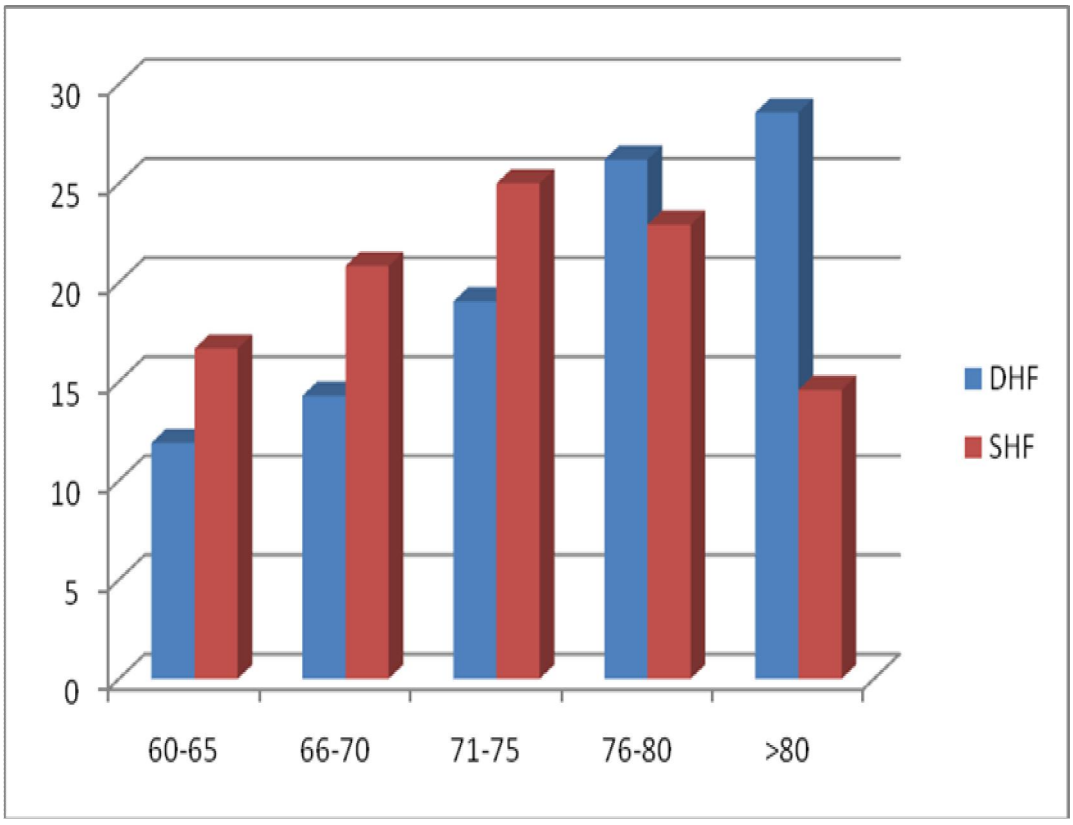
## PERCENTAGE OF DIASTOLIC HEART FAILURE IN ELDERLY CONGESTIVE CARDIAC FAILURE

| TOTAL POPULATION | DIASTOLIC HF | SYSTOLIC HF |
|---|---|---|
| 90 | 42(46.66%) | 48(53.34%) |



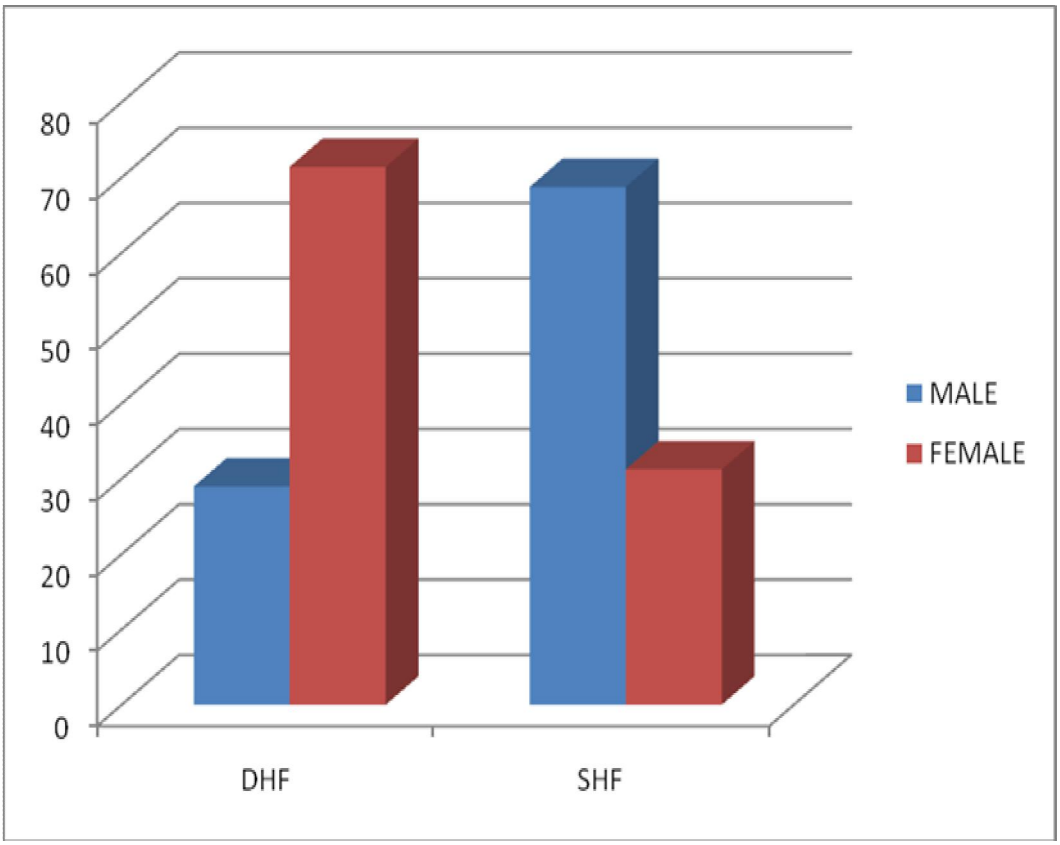
## AGE DISTRIBUTION

| AGE GROUP | DIASTOLIC HF | SYSTOLIC HF |
|---|---|---|
| 60-65 | 5(11.90%) | 8(16.66%) |
| 66-70 | 6(14.28%) | 10(20.83%) |
| 71-75 | 8(19.04%) | 12(25%) |
| 76-80 | 11(26.19%) | 11(22.91%) |
| >80 | 12(28.57%) | 7(14.58%) |



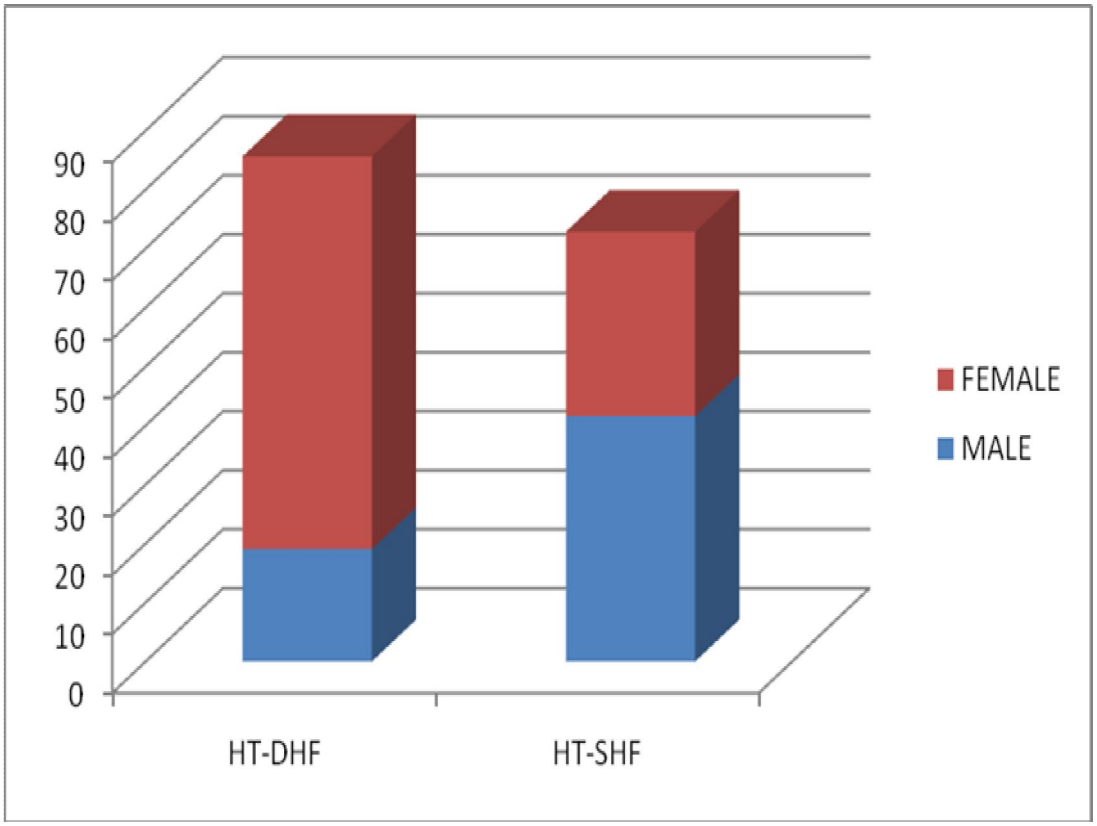
**SEX DISTRIBUTION**

| STUDY POPULATION | DHF | SHF | TOTAL |
|---|---|---|---|
| MALE | 12(28.97%) | 33(68.75%) | 45 |
| FEMALE | 30(71.42%) | 15(31.25%) | 45 |



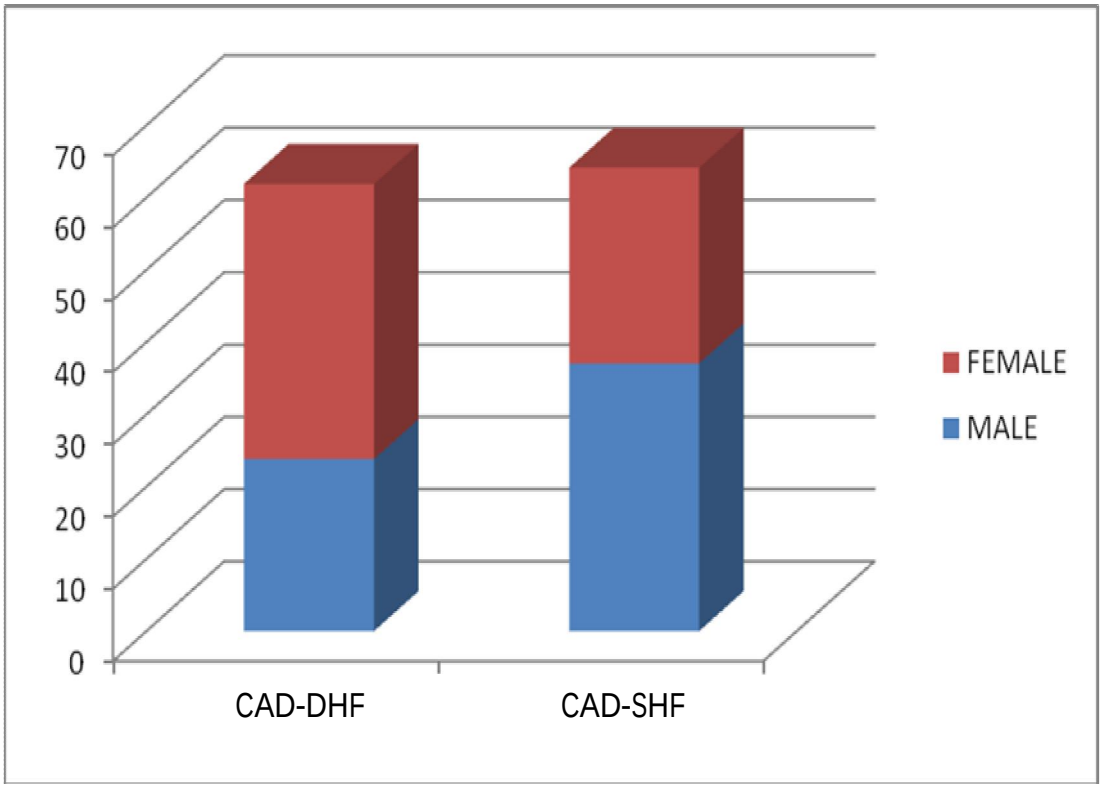
## DIASTOLIC HEART FAILURE WITH HYPERTENSION

| STUDY POPULATION | TOTAL | MALE | FEMALE | PERCENTAGE |
|---|---|---|---|---|
| DHF | 36 | 8(19.04%) | 28(66.6%) | 85.7% |
| SHF | 35 | 20(41.6%) | 15(31.25%) | 72.9% |



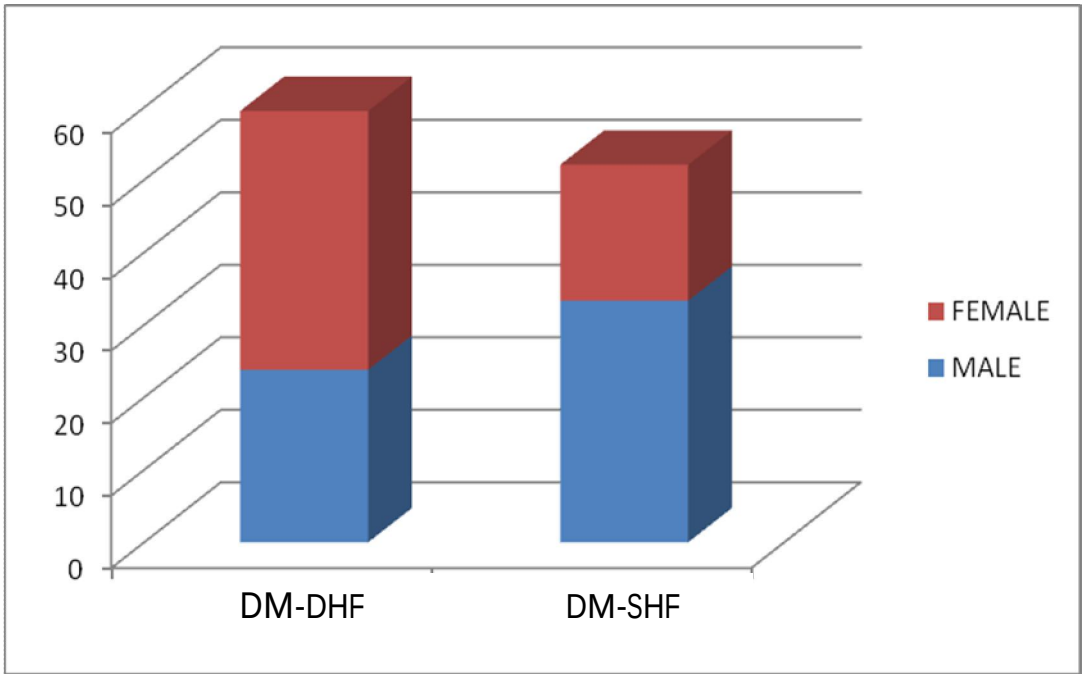
**DIASTOLIC HEART FAILURE WITH CORONARY ARTERY DISEASE**

| STUDY POPULATION | TOTAL | MALE | FEMALE | PERCENTAGE |
|---|---|---|---|---|
| DHF | 28 | 12(28.57%) | 16(38.09%) | 59.52% |
| SHF | 35 | 23(47.91%) | 12(25%) | 72.91% |



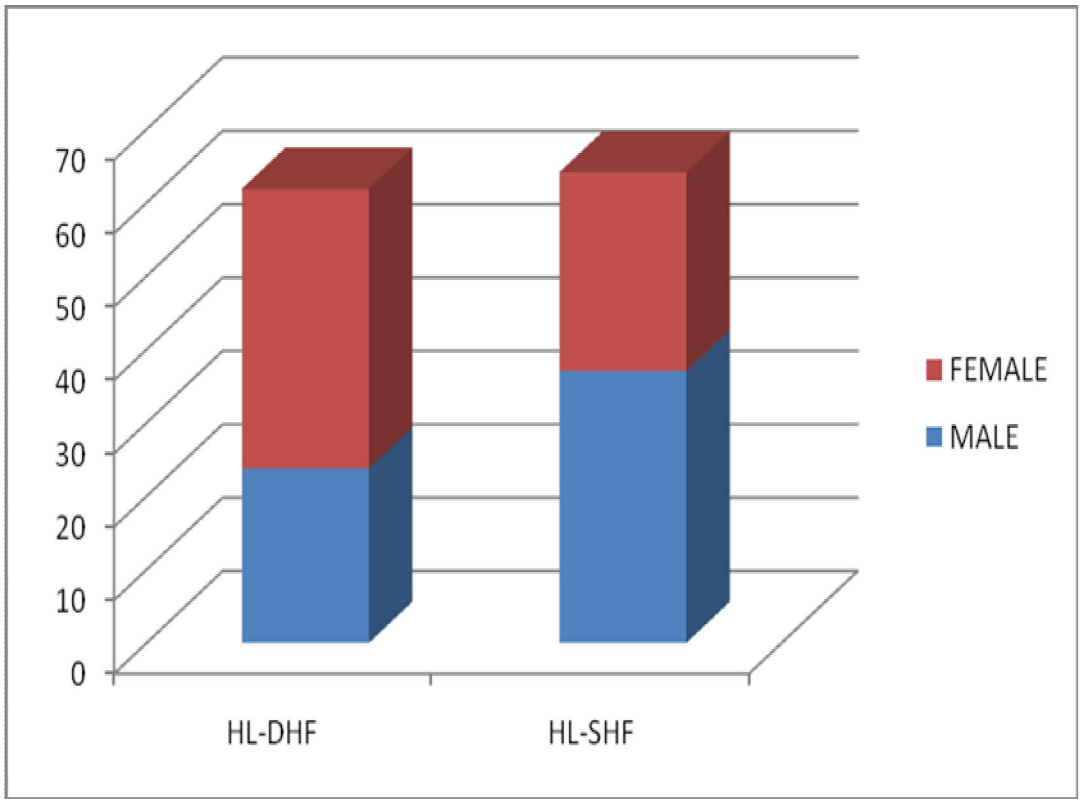
## DIASTOLIC HEART FAILURE WITH DIABETES MELLITUS

| STUDY POPULATION | TOTAL NO DM | MALE | FEMALE | PERCENTAGE |
|---|---|---|---|---|
| DHF | 25 | 10(23.80%) | 15(35.71%) | 59.52% |
| SHF | 26 | 16(33.33%) | 10(20.83%) | 54.16% |



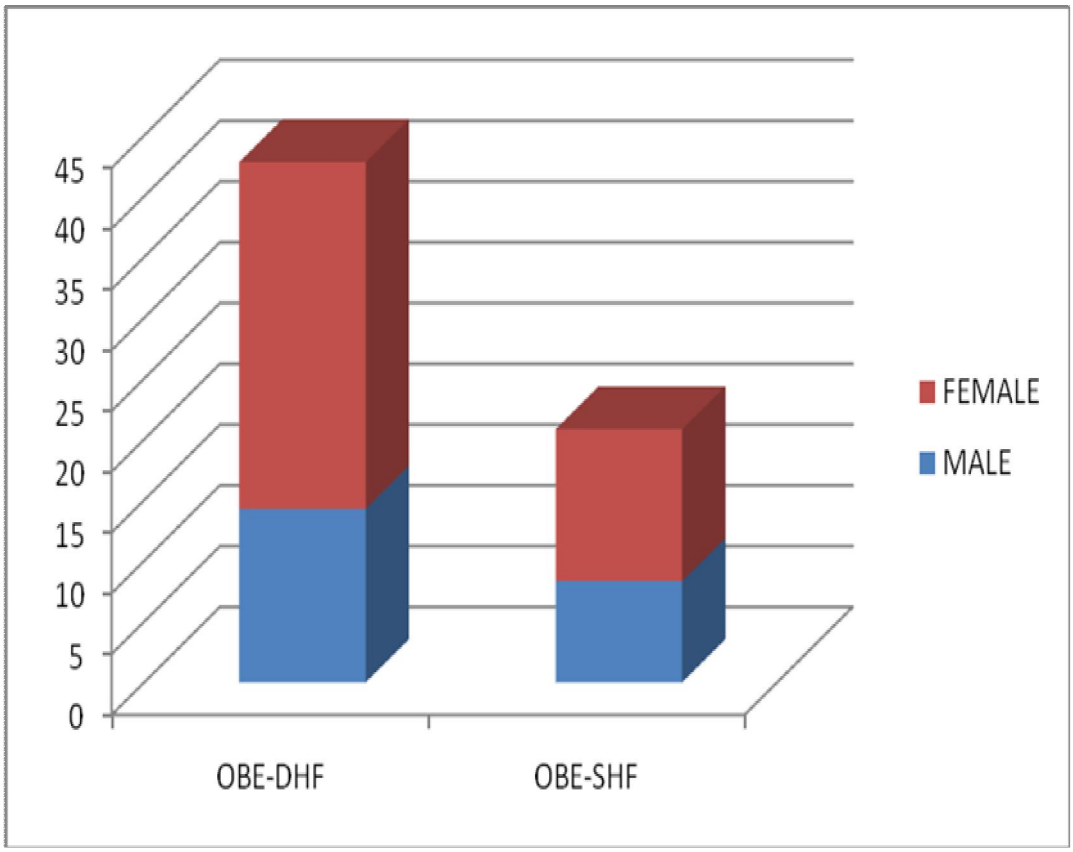
## DIASTOLIC HEART FAILURE WITH HYPERLIPEDIMIA

| STUDY POPULATION | TOTAL | MALE | FEMALE | PERCENTAGE |
|---|---|---|---|---|
| DHF | 26 | 10(23.80%) | 16(38.09%) | 61.90% |
| SHF | 31 | 18(37.5%) | 13(27.08%) | 64.58% |



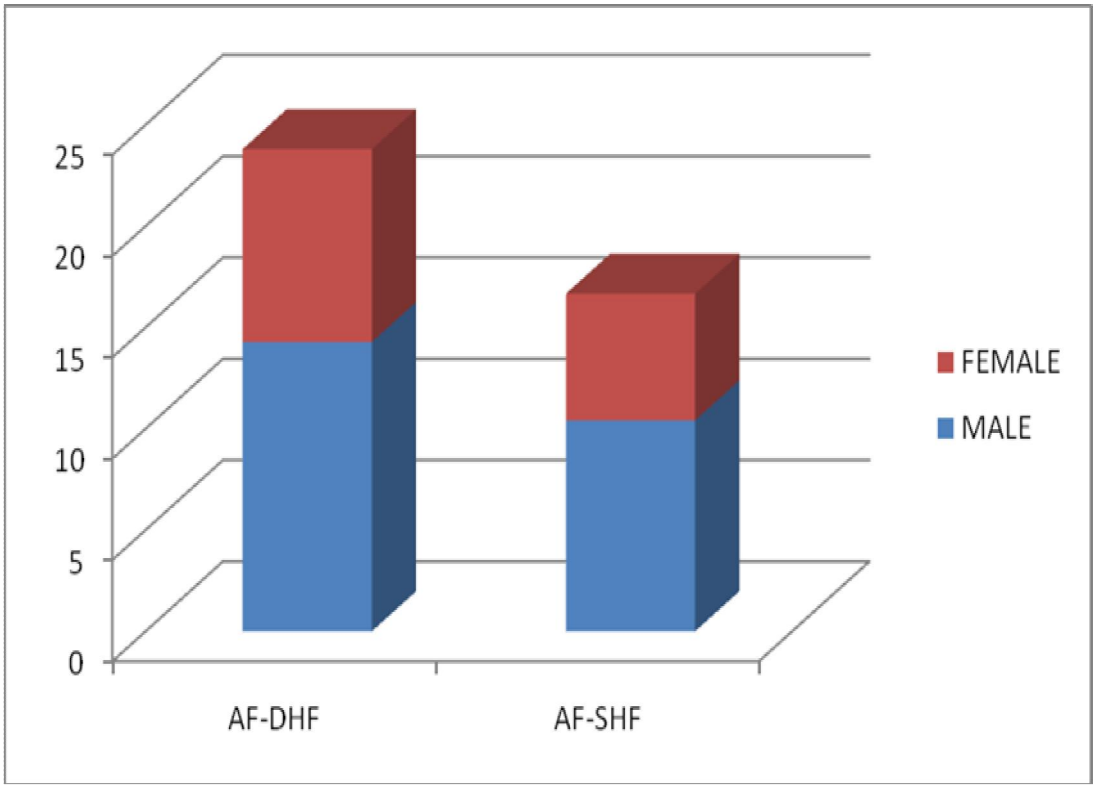
## DIASTOLIC HEART FAILURE WITH OBESITY

| STUDY POPULATION | TOTAL | MALE | FEMALE | PERCENTAGE |
|---|---|---|---|---|
| DHF | 18 | 6(14.28%) | 12(28.57%) | 42.85% |
| SHF | 10 | 4(8.33%) | 6(12.5%) | 20.83% |



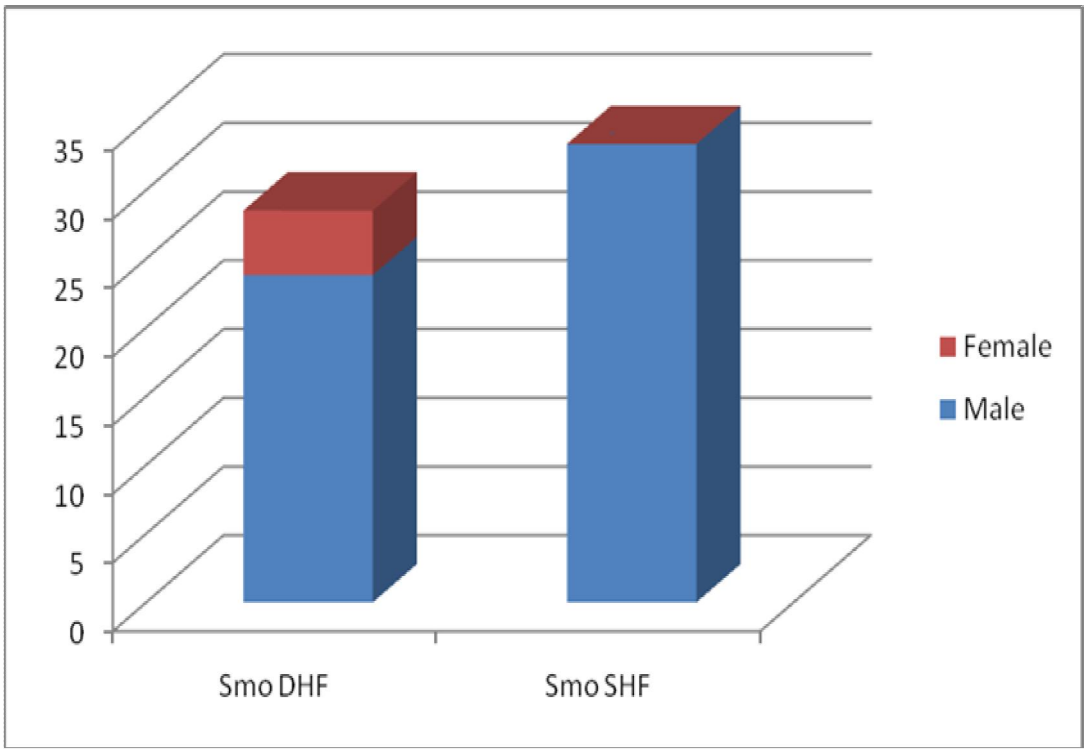
**DIASTLIC HEART FAILURE WITH ATRIAL FIBRILLATION**

| STUDY POPULATION | TOTAL NO | MALE | FEMALE | PERCENTAGE |
|---|---|---|---|---|
| DHF | 10 | 6(14.28%) | 4(9.52%) | 23% |
| SHF | 8 | 5(10.41%) | 3(6.25%) | 16.66% |



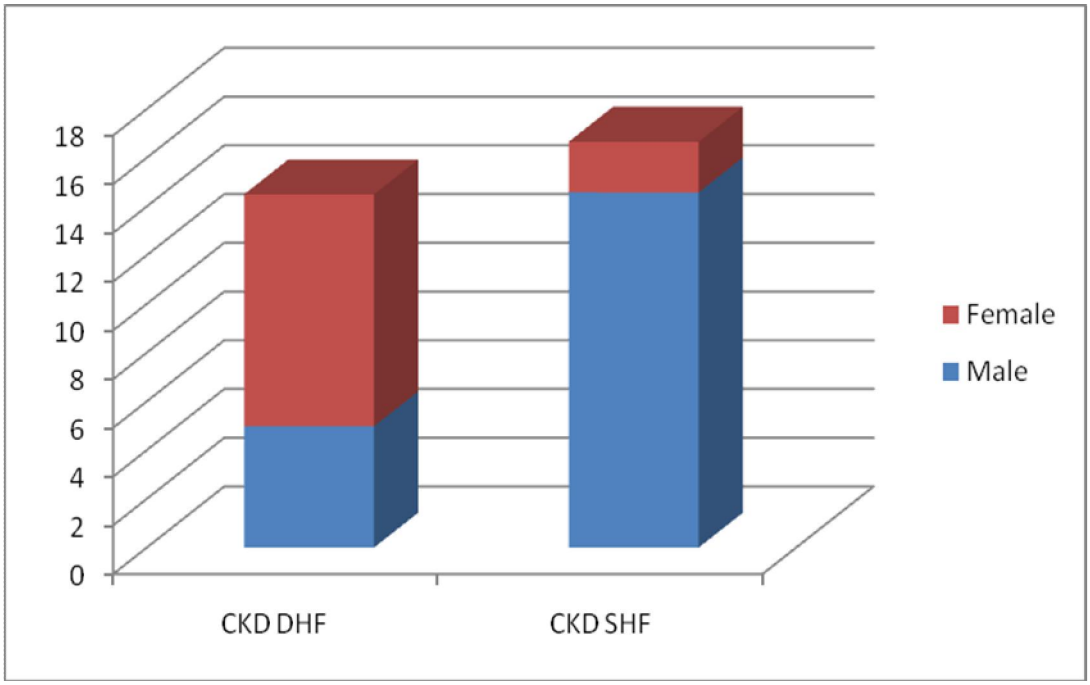
## DIASTOLIC HEART FAILURE WITH SMOKING

| STUDY POPULATION | TOTAL | MALE | FEMALE | PERCENTAGE |
|---|---|---|---|---|
| DHF | 12 | 10(23.80%) | 2 (4.76%) | 28.57% |
| SHF | 16 | 16(33.33%) | 0(0%) | 33.33% |



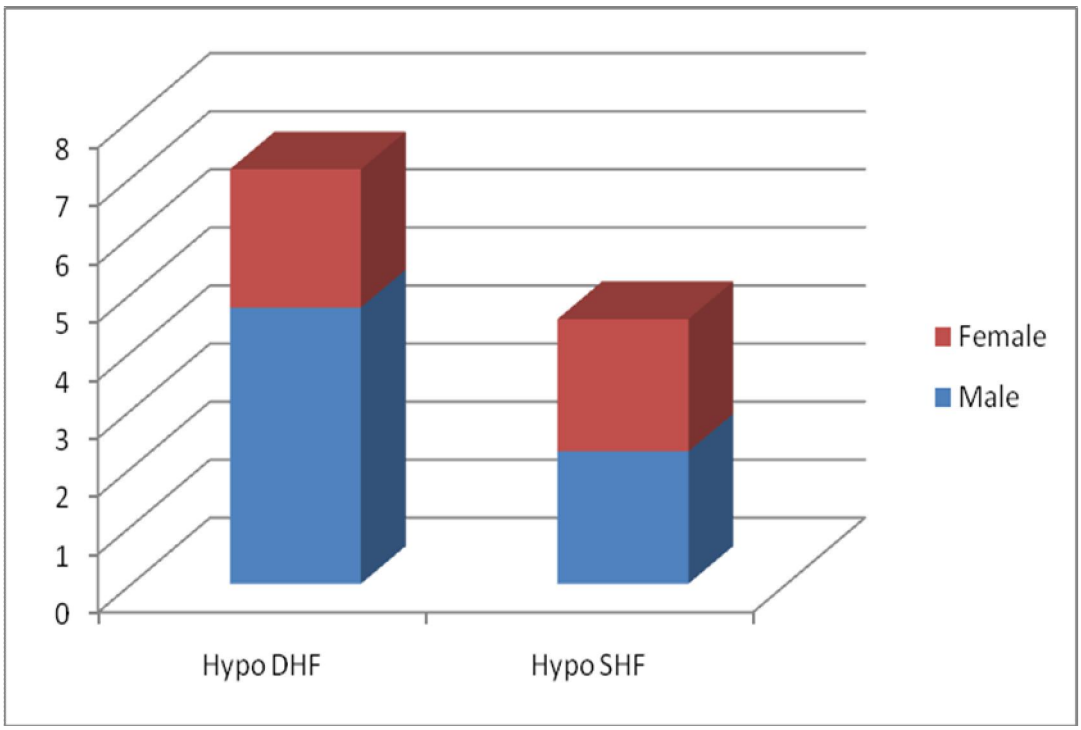
**DIASTOLIC HEART FAILURE WITH CHRONIC KIDNEY DISEASE**

| STUDY POPULATION | TOTAL | MALE | FEMALE | PERCENTAGE |
|---|---|---|---|---|
| DHF | 6 | 2 (4.98%) | 4 (9.52%) | 14.28% |
| SHF | 8 | 7 (14.58%) | 1( 2.08%) | 16.66% |



## DIASTOLIC HEART FAILURE WITH HYPOTHYROIDISM

| STUDY POPULATION | TOTAL | MALE | FEMALE | PERCENTAGE |
|---|---|---|---|---|
| DHF | 3 | 2(4.76%) | 1(2.38%) | 7.14% |
| SHF | 2 | 1(2.08%) | 1(2.08%) | 4.16% |



# CLINICAL MANIFESTATIONS

| S.NO | CLINICAL PRESENTATION | NO | PERCENTAGE |
|---|---|---|---|
| 1 | EXERTIONAL DYSPNEA | 42 | 100% |
| 2 | PND | 38 | 90.47% |
| 3 | ORTHOPNEA | 36 | 85.71% |
| 4 | NOCTORNAL COUGH | 30 | 71.42% |
| 5 | RALES | 40 | 95.23% |
| 6 | ANKLE EDEMA | 34 | 80.95% |
| 7 | PLEURAL EFFUSION | 10 | 23.80% |
| 8 | HEPATOMEGALY | 6 | 14.28% |
| 9 | TACHYCARDIA | 18 | 42.85% |
| 10 | JUGULAR VENOUS PULSE | 8 | 19.04% |

# DISCUSSION

- In this study 90 elderly people with congestive cardiac failure from general hospital were studied for prevalence of Diastolic Heart Failure with symptoms and Echocardiographic analysis. Of this 90 persons 45 were male,45 were female

- The overall prevalence of DHF among the elderly congestive cardiac patients was found to be 46% Prevalence of DHF increasing with age was noted 11.9% of DHF patients were found to be in the age group of60-65 yrs,14.28% among 65-70yrs, 19.04%in 71-75 yrs,26.19%in 76-80,**28.57%** in age group above 80yrs. Although cardiovascular disease may contribute to diastolic dysfunction in older people, study shown that diastolic function deteriorates with normal aging. The speed of LV relaxation declines with age in men and women, even in the absence of cardiovascular disease. It also appears that vascular, LV systolic, and LV diastolic stiffness increase with aging. Increases in vascular stiffness have been shown to be related to effort intolerance in patients with DHF. Structural cardiac changes with aging (e.g., increased cardiomyocyte size, increased apoptosis with decreased myocyte number, altered growth factor

regulation, focal collagen deposition) and functional changes at the cellular level involving blunted beta-adrenergic responsiveness, excitation-contraction coupling, and altered calcium handling proteins may contribute to diastolic dysfunction with normal aging

- Prevalence of DHF in the male population with CHF was found to be 37.7% whereas in **female** population was found to be 66.6% Along with age, female gender is a potent risk factor for DHF,. Indeed, there appears to be important age-gender interactions, such that the prevalence of DHF increases more sharply with age in women than the prevalence of HF with a reduced EF. The reasons for the female predominance in DHF are not entirely clear, but women have higher vascular and LV systolic and diastolic stiffness than men, and vascular and ventricular stiffness increases more dramatically with age in women..

- Among patients with DHF major association was found to be with Systemic hypertension.85.7% of patients with DHF were found to be hypertensive and 72.9% of patients with SHF had hypertension. Chronically increased blood pressure is an important stimulus for cardiac structural remodeling and functional changes. The resultant hypertensive heart disease is

characterized by LV hypertrophy (LVH), increasing vascular and ventricular systolic stiffness, impaired relaxation, and increased diastolic stiffness, all factors linked to the pathogenesis of DHF. In the presence of hypertensive heart disease, ischemia produces exaggerated increases in filling pressures, and hypertensive and ischemic heart disease are often present in combination inpatients with DHF.

- In patients with DHF 66.6% was associated with coronary artery disease whereas 72.9% of patients with SHF had coronary artery disease. prevalence of coronary artery disease or myocardial ischemia in patients with DHF varies widely. Although acute ischemia is known to cause diastolic dysfunction, the role of coronary artery disease and ischemia in contributing to chronic diastolic dysfunction and symptoms in patients with DHF remains speculative. Despite uncertainty regarding the role of ischemia in the pathophysiology of DHF and a lack of data documenting that revascularization improves outcomes in patients with DHF, HF management guidelines recommend revascularization in those DHF patients in whom “ischemia is felt to contribute to diastolic dysfunction.”

- 59.52% of patients with DHF had diabetes and 54.16% with SHF were diabetic. the prevalence of diabetes is similar in patients with HF and reduced or preserved EF, suggesting that diabetes contributes to the pathophysiology of both forms of HF. Although diabetes predisposes to coronary artery disease, renal dysfunction, and hypertension, numerous direct effects of diabetes and hyperglycemia on myocardial structure and function have been described

- 61.9%of patients with DHF had high lipid levels and 64.58% of SHF patients had hyperlipidemia.

- In 23% of patients with DHF atrial fibrillation was noticed and 16.66% of patients with SHF had associated AF. Atrial fibrillation is recognized as a frequent precipitant of acute decompensation in patients with DHF. Potential mechanisms responsible for this frequent presentation are discussed more fully later. Whereas atrial fibrillation may cause decompensated HF in patients with diastolic dysfunction, diastolic dysfunction (in the absence of HF) is also a risk factor for atrial fibrillation. Thus, diastolic dysfunction, atrial fibrillation, and DHF are common and related conditions that probably share common pathogenic mechanisms in the elderly.

- 42.85% of patients with DHF fell into the obese category and 20.83% with SHF were found to be obese. the prevalence of diastolic dysfunction is increased in obese persons. Increased adiposity not only imposes an adverse hemodynamic load on the heart but is also a source of a large number of biologically active peptide and nonpeptide mediators, many linked to chronic inflammation by various pathways. Increased body mass index (BMI) is a risk factor for hypertension, diabetes mellitus, coronary artery disease, and atrial fibrillation, all of which are associated with DHF.

- 14.28% of patients with DHF had associated chronic renal disease and 16.66% of SHF patients had renal disease.

- 47.61% patients with DHF had smoking as risk factor and 33.33% of SHF patients were found to smoke.

- 7.14% of DHF patients were hypothyroid and 4.16% of patients with SHF had associated hypothyroidism.

- The most common presentation of patients with DHF was found to be exertional dyspnea and the most common finding on examination of patients with DHF was found to be basal crackles and ankle edema.

# CONCLUSION

- 40- 50% of elderly with CHF have DHF.
- The prevalence of DHF has increased with age and appears to be quite common in elderly women with hypertension
- T he diagnosis of DHF is generally based on typical symptoms and signs of HF, preserved or normal LVEF, DD and no valvular abnormalities.
- The goal of therapy is the relief of symptoms, improvement in quality of life (improved exercise tolerance and reduced hospital admissions), and prolongation of survival. In the elderly, relief of symptoms and improvement of quality of life are crucial
- The outcomes of pharmacological therapy in patients with DHF are frequently neutral in clinical trials. Further trials are necessary to outline a specific line of therapy.
- Prognosis of DHF is as poor as that of systolic failure and if left undetected the mortality is as high as in systolic heart failure.

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NAME: ADDRESS:

AGE:

SEX:

OCCUPATION:

OP NO: IP NO: CONTACT NO:

FRAMINGHAM CRITERIA: 2 MAJOR or 1MAJOR+2 MINOR

| MAJOR CRITERIA | MINOR CRITERIA |
|---|---|
| Paroxysmal nocturnal dyspnea or orthopnea | Nocturnal cough |
| Jugular venous distention (or CVP > 16 mm Hg) | Exertional dyspnea |
| Rales or acute pulmonary edema | Pleural effusion |
| Cardiomegaly | Ankle edema |
| Hepatojugular reflex | Hepatomegaly |
| Response to diuretics(wt loss 4.5kg in 5days) | Tachycardia (>120 bpm) |

PULSE RATE:

BP:

BMI:

RISK FACTORS: Hypertension, coronary disease, diabetes, atrial fibrillation,Hyperlipedimia,smoking

CO MORBID CONDITIONS: Obesity, renal dysfunction,Hypothyroidism,COPD

HB% SUGAR(R): UREA : CREATININE: Na: K:

T.CHO: TGL: S.Alb:

ECG:

CHEST XRAY:

TREATMENT GIVEN:

NAME :

AGE :

SEX :

OP NO :

IP NO :

ECHOCARDIOGRAM

- VALVULAR HEART DISEASE/PERICARDIAL DISEASE/ASD :
- WALL MOTION ABNORMALITY :
- PULMONARY HYPERTENSION :
- LA LV IVS
  RA RV
- EJECTION FRACTION

| | |
|---|---|
| TEICHHOLZ FORMULA | |
| SIMPSON'S RULE | |

- DIASTOLIC FUNCTION

TRANS MITRAL

| E | A | E/A | IVRT | DT |
|---|---|---|---|---|
| | | | | |

PULMONARY VENOUS FLOW

| S | D | AR |
|---|---|---|
| | | |

DD GRADE: 1 2 3 4

| S NO | AGE | SEX | HT | DM | CAD | AF | CKD | OB | SMK | HYPO | HPL | SUGAR | UREA | Cr | LDL | HB | PR | BP | ECG | CXR | EF | GR DD | DIAG |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 62 | F | Y | Y | N | N | N | Y | Y | Y | N | 160 | 32 | 1.2 | 90 | 10 | 72 | 150/92 | LVH | CM | 52 | 2 | DHF |
| 2 | 65 | F | Y | Y | N | N | N | Y | Y | N | N | 200 | 34 | 1.1 | 78 | 11 | 76 | 148/90 | PRWP | N | 56 | 2 | DHF |
| 3 | 64 | F | Y | Y | N | N | N | Y | N | N | N | 184 | 29 | 1.3 | 88 | 12 | 78 | 150/88 | LAD | CM | 70 | 1 | DHF |
| 4 | 62 | F | Y | Y | N | N | N | Y | N | N | N | 163 | 28 | 0.9 | 80 | 13.5 | 70 | 150/90 | LVH | CM | 68 | 1 | DHF |
| 5 | 60 | F | Y | Y | N | N | N | Y | N | N | N | 174 | 26 | 0.8 | 86 | 12 | 68 | 146/94 | LAD | PF | 65 | 1 | DHF |
| 6 | 66 | M | Y | Y | Y | N | Y | Y | Y | N | N | 156 | 70 | 2.2 | 100 | 14 | 71 | 150/98 | LVH,LAD | CM | 64 | 1 | DHF |
| 7 | 70 | M | Y | Y | Y | N | Y | Y | Y | Y | N | 198 | 96 | 2.8 | 98 | 12 | 73 | 160/86 | LVH | CM | 68 | 1 | DHF |
| 8 | 68 | M | Y | Y | Y | N | N | Y | Y | Y | Y | 178 | 29 | 1.1 | 134 | 13 | 75 | 154/86 | LVH | CM | 72 | 1 | DHF |
| 9 | 67 | M | Y | Y | Y | N | N | Y | Y | N | Y | 168 | 31 | 1.2 | 140 | 11 | 73 | 146/90 | NSR | N | 70 | 1 | DHF |
| 10 | 66 | M | Y | Y | Y | N | N | Y | Y | N | Y | 159 | 30 | 1.3 | 150 | 10 | 72 | 144/90 | LAD | N | 68 | 1 | DHF |
| 11 | 69 | M | Y | Y | Y | N | N | Y | Y | N | Y | 160 | 32 | 1.1 | 148 | 11 | 71 | 142/86 | PRWP | N | 66 | 1 | DHF |
| 12 | 71 | F | Y | Y | Y | N | Y | Y | N | N | N | 176 | 66 | 2 | 78 | 13 | 70 | 144/92 | PRWP | PF | 54 | 2 | DHF |
| 13 | 73 | F | Y | Y | Y | N | Y | Y | N | N | N | 149 | 58 | 1.8 | 88 | 12 | 69 | 146/90 | LAD | N | 60 | 1 | DHF |
| 14 | 75 | F | Y | Y | Y | N | Y | Y | N | N | N | 178 | 76 | 1.9 | 76 | 11 | 68 | 148/92 | LVH | CM | 62 | 1 | DHF |
| 15 | 74 | F | Y | Y | Y | N | Y | Y | N | N | N | 148 | 80 | 2 | 86 | 12 | 72 | 150/92 | LVH | CM | 63 | 1 | DHF |
| 16 | 72 | F | Y | Y | Y | N | N | Y | N | N | N | 198 | 29 | 1.1 | 90 | 13 | 74 | 150/90 | ST-T | CM | 64 | 1 | DHF |
| 17 | 75 | M | Y | Y | Y | Y | N | N | Y | N | Y | 196 | 30 | 1.1 | 146 | 12 | 78 | 148/90 | LAD | N | 60 | 2 | DHF |
| 18 | 73 | M | Y | Y | Y | Y | N | N | Y | N | Y | 214 | 27 | 1.1 | 138 | 13 | 70 | 146/88 | PRWP | N | 70 | 1 | DHF |
| 19 | 71 | F | Y | Y | Y | N | N | Y | N | N | N | 206 | 30 | 1.1 | 78 | 11 | 68 | 148/90 | LVH | CM | 68 | 1 | DHF |
| 20 | 76 | F | Y | Y | Y | N | N | Y | N | N | N | 158 | 28 | 0.8 | 76 | 13 | 71 | 154/90 | LVH | CM | 66 | 1 | DHF |
| 21 | 78 | F | Y | Y | Y | N | N | N | N | N | N | 165 | 38 | 0.9 | 74 | 12 | 72 | 156/90 | LAD | N | 65 | 1 | DHF |
| 22 | 77 | F | Y | Y | Y | N | N | N | N | N | Y | 157 | 28 | 0.7 | 146 | 11 | 73 | 140/96 | PRWP | N | 52 | 2 | DHF |
| 23 | 76 | F | Y | Y | Y | N | N | N | N | N | Y | 168 | 40 | 0.8 | 156 | 12 | 74 | 146/90 | ST-T | CM | 60 | 1 | DHF |
| 24 | 79 | F | Y | N | Y | Y | N | N | N | N | Y | 84 | 32 | 0.9 | 146 | 12 | 76 | 150/90 | LVH | CM | 64 | 1 | DHF |
| 25 | 80 | F | Y | N | Y | Y | N | N | N | N | Y | 108 | 32 | 0.9 | 130 | 13.5 | 72 | 140/90 | PRWP | N | 64 | 1 | DHF |
| 26 | 79 | F | Y | N | Y | Y | N | N | N | N | Y | 96 | 32 | 0.8 | 156 | 10 | 72 | 160/100 | LVH | CM | 68 | 1 | DHF |
| 27 | 77 | F | Y | N | Y | Y | N | N | N | N | Y | 78 | 34 | 0.8 | 146 | 12 | 75 | 150/90 | LAD | N | 70 | 1 | DHF |
| 28 | 78 | F | Y | N | Y | N | N | N | N | N | Y | 88 | 26 | 1 | 156 | 13 | 82 | 156/92 | LVH | CM | 66 | 1 | DHF |
| 29 | 76 | F | Y | N | Y | N | N | N | N | N | Y | 86 | 29 | 0.9 | 148 | 12 | 76 | 158/90 | LVH | CM | 58 | 2 | DHF |
| 30 | 77 | F | Y | N | N | N | N | N | N | N | Y | 110 | 31 | 1 | 138 | 11 | 73 | 148/92 | PRWP | N | 65 | 1 | DHF |
| 31 | 81 | F | Y | N | N | N | N | N | N | N | Y | 86 | 35 | 0.8 | 142 | 12 | 71 | 144/90 | ST-T | CM | 67 | 1 | DHF |

| S NO | AGE | SEX | HT | DM | CAD | AF | CKD | OB | SMK | HYPO | HPL | SUGAR | UREA | Cr | LDL | HB | PR | BP | ECG | CXR | EF | GR DD | DIAG |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 32 | 82 | F | Y | N | N | N | N | N | N | N | Y | 98 | 32 | 0.9 | 154 | 13 | 69 | 148/90 | LAD | CM | 69 | 1 | DHF |
| 33 | 84 | F | Y | N | N | N | N | N | N | N | Y | 78 | 27 | 0.8 | 152 | 12 | 71 | 146/94 | LVH | CM | 62 | 1 | DHF |
| 34 | 86 | F | Y | N | N | N | N | N | N | N | Y | 76 | 28 | 0.8 | 164 | 11 | 68 | 152/94 | LAD | N | 63 | 1 | DHF |
| 35 | 85 | F | Y | N | N | N | N | N | N | N | Y | 84 | 36 | 0.9 | 158 | 10 | 70 | 158/90 | LVH | CM | 65 | 1 | DHF |
| 36 | 82 | F | Y | N | N | N | N | N | N | N | Y | 86 | 34 | 1 | 160 | 11 | 71 | 150/90 | LAD | CM | 61 | 1 | DHF |
| 37 | 81 | F | N | N | N | N | N | N | N | N | Y | 78 | 29 | 1.1 | 146 | 12 | 73 | 110/80 | NSR | N | 66 | 1 | DHF |
| 38 | 82 | M | N | Y | Y | Y | N | N | Y | N | Y | 68 | 32 | 0.8 | 140 | 11 | 72 | 120/84 | PRWP | N | 58 | 2 | DHF |
| 39 | 84 | M | N | Y | Y | Y | N | N | Y | N | Y | 98 | 30 | 0.9 | 138 | 13 | 76 | 130/70 | NSR | N | 65 | 1 | DHF |
| 40 | 83 | M | N | N | Y | Y | N | N | N | N | Y | 88 | 30 | 1 | 136 | 12 | 75 | 128/80 | NSR | N | 70 | 1 | DHF |
| 41 | 86 | M | N | N | Y | N | N | N | N | N | Y | 98 | 32 | 1.2 | 134 | 11 | 76 | 136/80 | WNL | N | 58 | 2 | DHF |
| 42 | 86 | F | Y | Y | Y | N | Y | Y | N | Y | N | 214 | 28 | 1 | 78 | 12 | 68 | 150/94 | LVH | CM | 30 | N | SHF |
| 43 | 60 | F | Y | Y | Y | N | N | Y | N | N | N | 204 | 32 | 0.8 | 90 | 13 | 66 | 156/90 | LVH | CM | 36 | N | SHF |
| 44 | 64 | F | Y | Y | Y | N | N | Y | N | N | N | 186 | 31 | 0.8 | 78 | 12 | 82 | 158/92 | LVH | CM | 37 | N | SHF |
| 45 | 65 | F | Y | Y | Y | N | N | Y | N | N | Y | 198 | 30 | 0.9 | 146 | 111 | 72 | 156/90 | LVH | CM | 38 | N | SHF |
| 46 | 63 | F | Y | Y | Y | N | N | Y | N | N | Y | 176 | 29 | 0.8 | 134 | 13 | 86 | 152/92 | LVH | CM | 34 | N | SHF |
| 47 | 61 | F | Y | Y | Y | N | N | Y | N | N | Y | 168 | 26 | 1 | 128 | 10 | 68 | 148/90 | AWMI | PF | 36 | N | SHF |
| 48 | 62 | M | N | Y | Y | N | Y | Y | Y | N | N | 158 | 98 | 3 | 100 | 11 | 72 | 120/80 | WNL | N | 35 | N | SHF |
| 49 | 64 | M | N | Y | Y | N | Y | Y | Y | Y | N | 198 | 70 | 2.6 | 86 | 13 | 70 | 110/70 | WNL | N | 40 | N | SHF |
| 50 | 65 | M | N | Y | Y | N | Y | Y | Y | N | N | 169 | 86 | 2.4 | 86 | 12 | 72 | 120/84 | WNL | N | 42 | N | SHF |
| 51 | 66 | M | N | Y | Y | N | Y | Y | Y | N | N | 159 | 68 | 2.1 | 89 | 13 | 71 | 116/70 | WNL | N | 34 | N | SHF |
| 52 | 67 | M | N | Y | Y | N | Y | N | Y | N | N | 178 | 78 | 1.9 | 96 | 12 | 69 | 120/80 | WNL | N | 45 | N | SHF |
| 53 | 69 | M | N | Y | Y | N | Y | N | Y | N | N | 168 | 76 | 1.8 | 80 | 13 | 75 | 122/76 | WNL | N | 40 | N | SHF |
| 54 | 70 | M | N | Y | Y | N | Y | N | Y | N | N | 198 | 72 | 1.8 | 82 | 11 | 68 | 128/80 | WNL | N | 38 | N | SHF |
| 55 | 66 | M | N | Y | Y | N | N | N | Y | N | N | 194 | 32 | 1.1 | 78 | 12 | 76 | 130/80 | IWMI | PF | 27 | N | SHF |
| 56 | 67 | M | N | Y | Y | N | N | N | Y | N | N | 174 | 31 | 1 | 76 | 13 | 76 | 132/80 | WNL | N | 30 | 1 | SHF |
| 57 | 68 | M | N | Y | Y | N | N | N | Y | N | N | 164 | 30 | 1 | 82 | 12 | 82 | 132/80 | WNL | N | 32 | N | SHF |
| 58 | 69 | F | Y | Y | Y | N | N | N | N | N | Y | 196 | 28 | 0.9 | 80 | 13 | 80 | 120/82 | WNL | N | 38 | N | SHF |
| 59 | 70 | F | Y | Y | Y | N | N | N | N | N | Y | 186 | 26 | 0.8 | 88 | 11 | 71 | 124/80 | WNL | N | 40 | N | SHF |
| 60 | 69 | F | Y | Y | Y | N | N | N | N | N | Y | 176 | 24 | 0.8 | 78 | 13 | 74 | 134/86 | WNL | N | 42 | N | SHF |
| 61 | 71 | F | Y | Y | Y | N | N | N | N | N | Y | 156 | 22 | 0.9 | 76 | 12 | 72 | 130/90 | WNL | N | 36 | N | SHF |
| 62 | 73 | F | Y | N | Y | N | N | N | N | N | Y | 98 | 23 | 0.8 | 86 | 11 | 76 | 140/90 | PRWP | PF | 35 | N | SHF |

| S NO | AGE | SEX | HT | DM | CAD | AF | CKD | OB | SMK | HYPO | HPL | SUGAR | UREA | Cr | LDL | HB | PR | BP | ECG | CXR | EF | GR DD | DIAG |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 63 | 75 | M | N | Y | Y | N | N | N | Y | N | N | 188 | 33 | 0.8 | 82 | 12 | 78 | 156/96 | LVH | CM | 37 | N | SHF |
| 64 | 73 | M | N | Y | Y | N | N | N | Y | N | N | 166 | 35 | 1 | 84 | 12 | 72 | 120/80 | WNL | N | 37 | N | SHF |
| 65 | 74 | M | Y | Y | Y | N | N | N | Y | N | N | 176 | 37 | 0.9 | 72 | 13 | 70 | 120/86 | WNL | N | 28 | 1 | SHF |
| 66 | 72 | M | Y | Y | Y | N | N | N | Y | N | N | 198 | 27 | 0.8 | 79 | 11 | 74 | 140/80 | WNL | N | 30 | N | SHF |
| 67 | 71 | M | Y | Y | Y | N | N | N | Y | N | Y | 186 | 26 | 0.8 | 140 | 12.5 | 72 | 136/40 | WNL | N | 29 | N | SHF |
| 68 | 72 | M | Y | Y | Y | N | N | N | Y | N | Y | 196 | 25 | 0.8 | 136 | 11 | 70 | 124/80 | WNL | N | 36 | N | SHF |
| 69 | 73 | M | Y | N | Y | N | N | N | N | N | Y | 110 | 32 | 1 | 132 | 10 | 69 | 134/90 | WNL | N | 40 | N | SHF |
| 70 | 74 | M | Y | N | Y | N | N | N | N | N | Y | 74 | 31 | 1.1 | 150 | 12 | 72 | 134/86 | WNL | N | 42 | N | SHF |
| 71 | 75 | M | Y | N | Y | N | N | N | N | N | Y | 68 | 33 | 1.1 | 142 | 13 | 66 | 136/86 | WNL | N | 43 | N | SHF |
| 72 | 71 | M | Y | N | Y | N | N | N | N | N | Y | 78 | 32 | 1 | 129 | 12 | 71 | 146/86 | WNL | N | 39 | N | SHF |
| 73 | 76 | F | Y | N | Y | N | N | N | N | N | Y | 58 | 34 | 1.2 | 1142 | 11 | 69 | 170/90 | LVH | CM | 40 | N | SHF |
| 74 | 77 | F | Y | N | N | N | N | N | N | N | Y | 98 | 32 | 0.9 | 148 | 13 | 70 | 140/80 | LAD | N | 48 | N | SHF |
| 75 | 79 | F | Y | N | N | Y | N | N | N | N | Y | 76 | 31 | 1 | 138 | 12 | 82 | 126/88 | AWMI | PF | 25 | N | SHF |
| 76 | 78 | F | Y | N | N | Y | N | N | N | N | Y | 86 | 34 | 0.9 | 136 | 10 | 86 | 140/90 | NSR | N | 42 | 1 | SHF |
| 77 | 80 | F | Y | N | N | Y | N | N | N | N | Y | 78 | 35 | 1.1 | 142 | 11 | 79 | 150/90 | LVH | CM | 32 | N | SHF |
| 78 | 76 | M | Y | N | Y | N | N | N | N | N | Y | 98 | 36 | 1.1 | 156 | 12 | 75 | 146/90 | LAD | CM | 30 | N | SHF |
| 79 | 79 | M | Y | N | Y | N | N | N | N | N | Y | 112 | 37 | 1.2 | 142 | 13 | 73 | 150/86 | LVH | CM | 34 | N | SHF |
| 80 | 78 | M | Y | N | Y | N | N | N | N | N | Y | 121 | 31 | 1 | 142 | 12 | 72 | 144/84 | PRWP | PF | 27 | N | SHF |
| 81 | 80 | M | Y | N | N | N | N | N | N | N | Y | 124 | 29 | 0.9 | 134 | 13 | 68 | 166/90 | LVH | CM | 30 | N | SHF |
| 82 | 76 | M | Y | N | N | N | N | N | N | N | Y | 116 | 27 | 0.8 | 136 | 12 | 81 | 146/90 | LAD | CM | 34 | N | SHF |
| 83 | 77 | M | Y | N | N | N | N | N | N | N | Y | 118 | 23 | 0.7 | 140 | 13 | 76 | 168/96 | LVH | CM | 36 | N | SHF |
| 84 | 81 | M | Y | N | N | N | N | N | N | N | Y | 108 | 25 | 0.8 | 134 | 12 | 80 | 160/90 | LVH | CM | 31 | N | SHF |
| 85 | 85 | M | Y | N | N | Y | N | N | N | N | Y | 106 | 21 | 0.8 | 130 | 13 | 76 | 156/88 | LAD | N | 36 | N | SHF |
| 86 | 86 | M | Y | N | N | Y | N | N | N | N | Y | 112 | 28 | 0.9 | 168 | 12 | 72 | 184/90 | LVH | CM | 30 | N | SHF |
| 87 | 83 | M | Y | N | N | Y | N | N | N | N | Y | 116 | 22 | 0.8 | 178 | 13 | 75 | 168/90 | LVH | CM | 35 | N | SHF |
| 88 | 82 | M | Y | N | N | Y | N | N | N | N | Y | 98 | 28 | 0.9 | 168 | 12 | 73 | 144/88 | LAD | N | 31 | N | SHF |
| 89 | 81 | M | Y | N | N | Y | N | N | N | N | Y | 118 | 32 | 1 | 142 | 11.8 | 70 | 134/86 | PRWP | PF | 28 | N | SHF |
| 90 | 83 | M | N | Y | Y | N | Y | Y | N | Y | N | 218 | 90 | 3 | 132 | 11.4 | 90 | 120/80 | WNL | N | 30 | N | SHF |

# INSTITUTIONAL ETHICAL COMMITTEE
# MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No: 04425305301
Fax : 044 25363970

## CERTIFICATE OF APPROVAL

To
Dr. P. Ranjith
PG in MD Geriatrics
Madras Medical College, Chennai -3

Dear Dr. P. Ranjith

The Institutional Ethical Committee of Madras Medical College reviewed and discussed your application for approval of the project / proposal / clinical trail entitled **"Prevalance and Associated factors of Diastolic heart failure in elderly patients with Congestive heart failure "No 10092010.**

The following members of Ethical committee were present in the meeting held on 14.09.2010 conducted at Madras Medical College, Chennai -3.

| | |
|---|---|
| 1. Prof. S.K. Rajan, MD | -- Chairperson |
| 2. Prof. J. Mohanasundaram, MD,Ph.D,DNB<br>Dean, Madras Medical College, Chennai -3 | -- Deputy Chairman |
| 3. Prof. A. Sundaram, MD<br>Vice Principal , MMC, Chennai -3 | -- Member Secretary |
| 4. Prof. R. Sathianathan, MD<br>Director, Institute of Psychiatry | -- Member |
| 5. Prof R. Nandhini, MD<br>Director, Institute of Pharmacology, MMC, Ch-3 | -- Member |
| 6. Prof. C. Rajendran , MD<br>Director, Institute of Internal Medicine, MMC, Ch-3 | -- Member |
| 7. Prof. Geetha Subramanian, MD,DM<br>Professor & Head , Dept. Of Cardiology | -- Member |
| 8. Prof. V. Shruti Kamal, MS<br>Professor of Surgery, MMC, Ch-3 | -- Member |
| 9. Prof. Md. Ali, MD, DM<br>Professor & Head ,,Dept. of MGE, MMC, Ch-3 | -- Member |
| 10. Tmt.Arnold Saulina,<br>Social Scientist | -- Member |

**We approve the proposal to be conducted in its presented form.**

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee