

Congestive Heart Failure (CHF) in Elderly

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Geriatric growth is a reality; by the year 2035, nearly one in four individuals will be 65 years of age or older.¹ Congestive heart failure (CHF) is a common problem in elderly, which accounts for about half of deaths in over 65 years. The prevalence of CHF rises from 1% in 50-59 years age group to 10% in 80-89 years age group according to Framingham study^{2,3} which excluded mild heart failure. Heart failure represents a final common pathway for most cardiovascular disorders, their management increases the survival albeit with increased risk for heart failure. In a study⁴, 5% of total admissions in a hospital were due to CHF and majority (80%) were patients over 65 years of age. More than 90% of CHF deaths occur in adults above 65 year of age⁵. Among elderly persons hospitalized for heart failure, the 5 years mortality is more than 7% with similar incidence in blacks and whites. CHF in elderly poses challenge because of atypical symptoms, age related physiological changes and co-morbidities that influence management strategies.

Age associated changes in heart structure and function in normal elderly

There is a continuum of cardiac structural and functional alterations with age in healthy human, and these age associated cardiac changes appear to have relevance to the steep increase in heart failure with increasing age.

With aging, the large elastic arteries become dilated with a reduction in compliance. Progressive thickening of the aortic media and intima are associated with aortic enlargement. There is an age-associated increase in arterial stiffness resulting from changes in

the arterial media such as thickening of the smooth muscle layers, increased fragmentation of elastin, an increase in the amount of collagen, and increased calcification. Arterial stiffening leading to an increase in pulse wave velocity is associated with degeneration of the vascular media independent of atherosclerosis. Arterial stiffening causes earlier occurrence of wave reflection from peripheral sites to the ascending aorta. With aging, there is an increase in systolic blood pressure and a widened pulse pressure. A slight reduction in diastolic blood pressure occurs after the sixth decade.

With aging, left ventricular stiffness is increased, left ventricular compliance is decreased, left ventricular relaxation is impaired, and left ventricular early diastolic filling is decreased. This may result in hypotension if preload is decreased. Left ventricular filling during early diastole decreases 50 percent from age 20 to 80 years⁶. In the Framingham Heart Study⁷, age was the most powerful independent variable for left ventricular filling in healthy persons. Age was inversely associated with the E wave (Peak early diastolic filling velocity) and was directly associated with the A wave (peak late diastolic filling velocity), but due to aging per se E/A ratio is never less than 0.5 (E/A ratio less than 0.5 is indicative of diastolic dysfunction).

Since left atrial contraction can contribute up to 50 percent of left ventricular filling in a poorly compliant left ventricle, development of atrial fibrillation may cause a marked reduction in cardiac output due to the loss of left atrial contribution to left ventricular late diastolic filling. A rapid ventricular rate associated with atrial fibrillation will also reduce the time for diastolic filling of the left ventricle.

The intrinsic ability of the heart to generate force does not change with age in healthy people. However, the duration of contraction and relaxation is prolonged in senescent animals. Prolongation of left ventricular ejection time and of the pre-ejection period with aging, in healthy persons, indicates that prolongation of contraction occurs with aging.

There is no reduction in resting left ventricular

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ejection fraction or circumferential fiber shortening but systolic function with exercise is impaired with aging. In the Baltimore study of aging, elderly showed less exercise induced increase in left ventricular ejection fraction when compared with young individuals because of an age related increase in left ventricular end systolic volume⁸.

Aging is associated with prolongation of isovolumic relaxation time, a reduction in early diastolic filling of the left ventricle, and augmentation of late diastolic filling of the left ventricle. Normal aging changes affecting left ventricular diastolic function include increase in systolic blood pressure, increase in left ventricular wall thickness (Fig. 1). Above described changes in heart structure and function due to aging are summarized in Table 1 and Box 1-2.

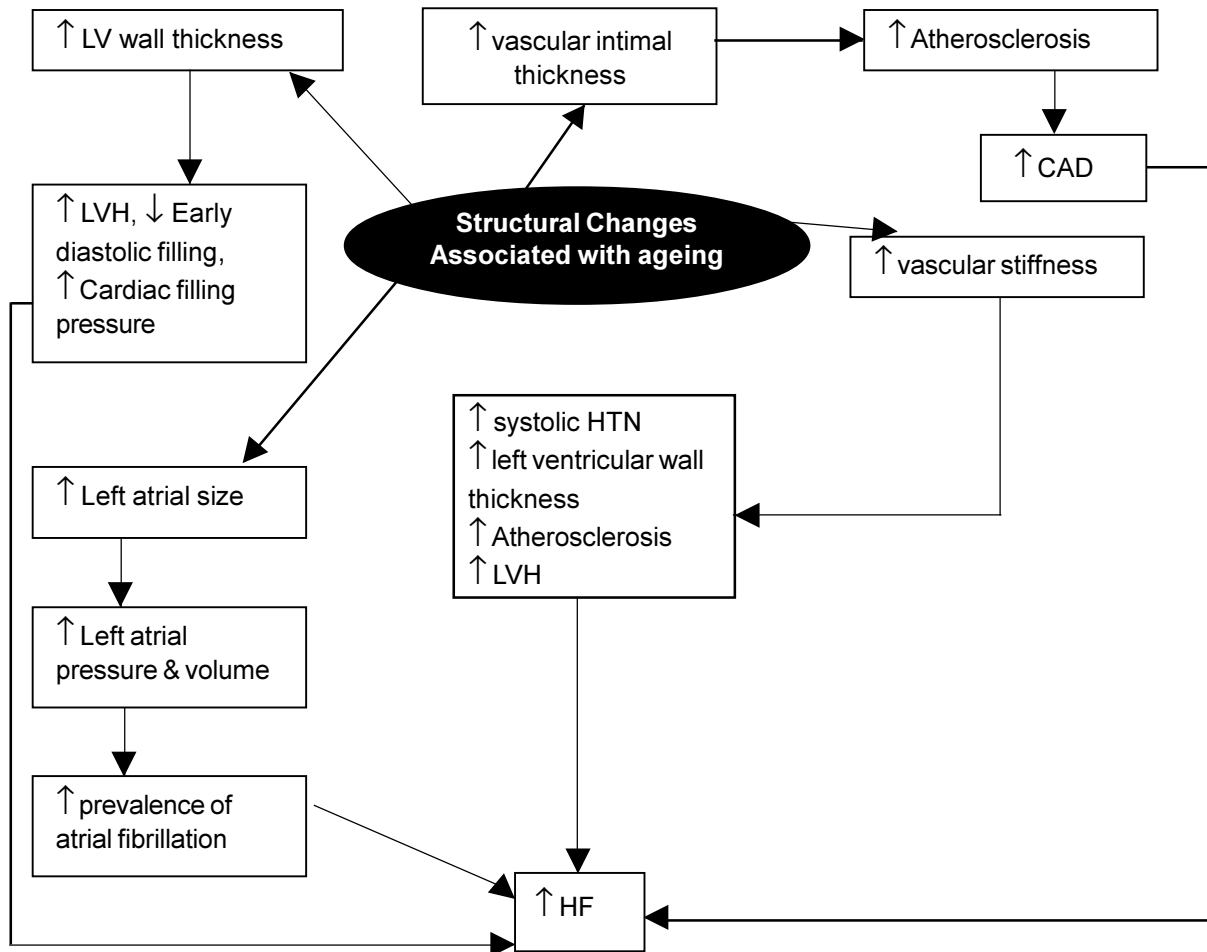


Fig. 1 : Age associated changes in cardiovascular function in elderly relevant to heart failure

Table 1: Myocardial changes with adult aging.

Changes in structures	Physiological effects
Increase in myocyte size	• Prolonged contraction
Decrease in myocyte number	• Prolonged action potential
	• Diminished contraction velocity
	• Diminished beta-adrenergic contractile response
Increase in matrix connective tissue	• Increased myocardial tissue stiffness

Box 1 : Age related alterations in cardiovascular function in elderly

1. Resting preload does not change with age
2. Contractility at rest does not change with age
3. Decrease in arterial compliance largely responsible for increase in afterload in elderly
4. Prolongation of left ventricular contraction and relaxation time with age
5. Left ventricular early diastolic filling is decreased with aging

Box 2 : Cardiovascular response to exercise in healthy elderly

1. Peak exercise LVEF decreases with aging
2. LVEDV and LVESV ↑ during peak exercise with aging
3. Maximum heart rate decreases with aging
4. Exercise stroke volume increases with aging to maintain cardiac output
5. Systemic arteriovenous oxygen difference increases with aging at peak exercise
6. VO₂ max at peak exercise decreases with aging

It is important to note that, age associated cardiovascular changes themselves are unlikely to result in heart failure but they increase the likelihood in presence of cardiac diseases.

Pathophysiology of CHF

Congestive heart failure may be predominantly systolic or diastolic. CHF with normal systolic function and diastolic dysfunction is common and has been seen in 40% of patients of 70 years as compared to 6% of those less than 60 years.⁹ Diastolic dysfunction is more common with hypertension and is characterized by decreased early diastolic filling rates, increased end diastolic volume and pressure, and increased dependence on atrial contribution.

Systolic dysfunction is more common with ischaemic heart disease. Aging heart with modest hypertrophy is more sensitive to ischaemic injury. Impedance to LV ejection dominate the picture in CHF in elderly and vasodilation is the most important corrective measure. Aged heart is slower or may have decreased capacity to modify structures in response to long term changes in load hence, decreased ability to tolerate aortic stenosis or alteration in LV functions like acute myocardial infarction. These physiological changes with aging results in decrease in maximal work capacity and increased oxygen consumption.

Aetiology of CHF

Hypertension, coronary heart disease and cor pulmonale are the commonest causes of CHF (Box-3). In the Framingham study², hypertension was found in 76% men and 79% of women with heart failure, coronary artery disease was prevalent in 46% of men and 27% of women with CHF. Cor pulmonale may be present alone or along with coronary artery disease. Calcified aortic stenosis, mitral valve calcification, mitral regurgitation secondary to mitral valve prolapse are the commonest valvular diseases in elderly. However,

valvular heart diseases are uncommon sole cause of CHF. Multiple causes may contribute to CHF in elderly like coexisting hypertension, coronary artery disease and calcified aortic stenosis.

Box 3: Common aetiology of CHF in elderly

- Hypertension
- Coronary heart disease
- Cor pulmonale
- Valvular heart disease
- Myocarditis
- Cardiomyopathy (Amyloid or Hemochromatosis)
- Chemotherapy induced (Adriamycin)

Congestive heart failure is precipitated by any condition placing extra strain on cardiovascular system which is otherwise in a compensated state. In elderly, myocardial ischaemia, atrial fibrillation, digitalis withdrawal or administration of NSAIDs, noncompliance with drugs, diet or fluid restrictions are common precipitating factors (Box 4).

Box 4 : Precipitating factors for CHF

- Anaemia
- Arrhythmias (atrial fibrillation)
- Myocardial ischaemia
- Thyrotoxicosis
- Exacerbation of COPD
- Drugs (eg. NSAIDs)
- Infection/sepsis
- Hypoxia
- Renal failure
- IV fluid overload
- Pulmonary embolism
- Dietary or medication non-compliance

Clinical Features

The elderly may present as in the young with classical symptoms of heart failure. However, due to decreased work and coexistent diseases they may present late and often in atypical manner¹⁰ (Box 5).

Box 5: Atypical features of heart failure in elderly

Lethargy	Confusion
Falls	Dizziness
Weakness	Syncope
Immobility	Insomnia
Anorexia	Fatigue

History may be lacking or symptoms like cough may be attributed to aging. Dyspnea may not be noticed due to lack of activities with co-morbidities like osteoarthritis. Symptoms of low cardiac output e.g., weakness, tiredness, fatigue will also appear late due to low level of activities or may be masked by co-morbidities like depression. Low cardiac output is more likely to present as acute confusion, falls, blackouts and even fits due to poor cerebral perfusion. Falls may be due to skeletal muscle wasting associated with CHF, which may be due to undernutrition (cardiac cachexia)¹¹.

Physical Signs: Most of the physical signs of heart failure are nonspecific, thus they should be interpreted with caution as they may be attributable to other diseases. While the Framingham criteria for CHF have been frequently cited, they have not been validated in a purely elderly population and their applicability to clinical practice and research trials has not been tested extensively.

In a series of Cocchi et al¹² from Italian aged patients, hospitalized with heart failure, the most common signs and symptoms were pulmonary rales (77%), orthopnea (73%), edema (62%), paroxysmal nocturnal dyspnea (56%), gallop rhythm (45%) and jugular venous distention (32%). Elevation of jugular venous pressure or hepato-jugular reflux are excellent signs of right sided failure. Hepatomegaly and ascites if present are helpful but may have other causes as well. S₃ gallop is an excellent sign of heart failure but may be difficult to hear. Pedal edema is most commonly gravitational due to impaired mobility, varicose veins or hypoalbuminemia. Bilateral crackles may be present commonly in elderly due to fibrotic changes in lung. Commonly present S₄ in healthy elderly makes it a nonspecific finding. Thus pedal edema, pulmonary crackles and S₄ may be non specific in elderly for CHF.

The ability to discriminate clinically between those with primarily systolic or diastolic function is difficult, diabetes, coronary artery disease, prior myocardial infarction is high in both groups; moreover there is no difference in presenting symptoms and signs, precipitating factors or historical factors.

The differences in heart failure in elderly as compared to younger population is summarized in (Table 2).

Table 2: Heart failure in elderly versus middle aged patients

Characteristics	Elderly	Middle aged
Prevalence	4 to 15%	<12
Gender	M < F	
Etiology	Hypertension	CAD
Type of LV dysfunctions	Diastolic > systolic	Systolic > Diastolic
Co-morbidity	Multiple	Few or None

Investigations

A good quality X-ray chest may be difficult to obtain. It can be performed with the patient sitting upright in wheelchair. Key findings are an enlarged heart, enlarged hila with indistinct margins and prominence of veins draining the upperlobes.

B type Natriuretic Peptide (BNP) is a 32 aminoacid polypeptide containing 17 amino acid ring structure

Box 6: Investigations

- Chest X-ray
- Electrocardiogram
- Blood tests (full blood count, urea and electrolytes, random blood sugar, thyroid function tests)
- Echocardiograph
- Exercise testing
- Invasive tests (for selected elderly patients)

common to all natriuretic peptides. BNP release appears to be directly proportional to ventricular volume expansion and pressure overload. BNP is an independent predictor of high left ventricular end diastolic pressure.¹² In a study by Maisel et al¹², all patients with a known history of CHF and previously documented LV dysfunction (n=102), had abnormal findings with elevated BNP levels (545±45 pg/ml). BNP levels are elevated in both systolic and diastolic dysfunction, with higher values in systolic dysfunction.

Accumulating data suggest that BNP will be of immense help in emergency room for differentiating cardiac dyspnea from breathlessness due to other causes (cutoff value of BNP 100 pg/ml). Additionally, BNP might serve as a screening test for patients referred for echocardiography. A low BNP level makes echocardiographic indices of LV dysfunction (both systolic and diastolic) highly unlikely. BNP is a simple blood test that aids in the diagnosis and management of patients with congestive heart failure (CHF) and would clearly have a favourable impact in future.¹³

ECG is helpful in establishing the etiological diagnosis but it may be normal. Echocardiography is essential not only to diagnose reversible causes e.g., pericardial and valvular diseases but also to differentiate between predominant systolic and diastolic dysfunction (Table 3), influencing treatment and prognosis. Exercise testing and invasive investigations should be reserved for patients intended to undergo cardiac surgery.

Table 3: Echocardiographic differences in Systolic and Diastolic CHF

Echocardiographic findings	Systolic dysfunction	Diastolic dysfunction
Cavity size	dilated	Normal
LV wall	thin	thick
Ejection fraction	decreased	normal or increased
Early diastolic filling	normal	decreased
Diastolic pressure	normal	increased

Treatment

Principles of treating heart failure in elderly are similar to young but age related pharmacokinetic changes and coexistent morbidities require due diligence. Both general measures and specific measures are to be taken simultaneously.

General Measures

Salt restriction between 3-4 gms/day and control of precipitating factors like anemia, atrial fibrillation are as important in elderly as in young. All efforts should be done to prevent cardiac cachexia and ensure a proper diet to prevent undernutrition which is so prevalent in elderly. Alcohol should be omitted and discouraged. Prevention of deconditioning of elderly by early mobilization and exercises to strengthen muscles after acute symptoms have been controlled, are important measures. Aerobic exercises like walking, cycling increases functional capacity and quality of life. Education of patient and social support group is important to achieve compliance and early medical intervention.

Specific measures

These measure are to achieve increased quality of life and improved survival. In CHF diuretics, ACE inhibitors and beta blockers are the corner stone of treatment. CHF with predominant systolic dysfunction is commonly seen in patients of ischaemic heart disease. Vasodilators decreases preload and afterload

and improves systolic function. Diuretics alone or in conjunction with vasodilators and digitalis improve clinical outcome in this group.

Diuretics

The goal of treatment is to induce gentle diuresis and to avoid hypotension. As glomerular filtration rate in elderly is often below 30ml/min, thiazides alone are usually ineffective. Loop diuretics (Frusemide, Bumetamide, Ethacrynic acid) are used most frequently; however due to their decreased efficacy, higher doses are needed (40-160 mg of frusemide daily). Ethacrynic acid is rarely used due to ototoxicity. First dose of frusemide should not be more than 20 mg because occasional exaggerated first dose diuresis causes hypovolemia. Loop diuretics may be used in conjunction with thiazides.

Major side effects of loop diuretics are hypotension, hypokalemia, hypomagnesemia, hyponatremia and incontinence. Hypotension can be avoided by monitoring orthostatic hypotension, BUN, Serum creatinine and reducing the dose of loop diuretic at the first sign of volume depletion. Effort to remove the last trace of peripheral edema in elderly may decrease preload and decrease cardiac output. Serum potassium and magnesium should be measured routinely and replaced as indicated. Magnesium oxide supplements 400 mg 1-2 times daily for several days are usually sufficient. Hyponatremia due to excessive diuresis is usually associated with hypovolemia and is corrected by reducing the dose of diuretics; hyponatremia secondary to free water retention should be treated with fluid restrictions. Major drug interactions include other drugs with hypotensive potential, nephrotoxic drugs and NSAIDs which by their action on GFR can completely inhibit brisk loop diuretic induced diuresis.

Spirolactone

In RALES study¹⁴, spironolactone 25 mg daily was shown to reduce mortality by 27% in patients with NYHA class III & IV CHF with ejection fraction of less than 30% and on full dose of ACE inhibitors and diuretic therapy with or without digoxin. The mortality benefit was similar in younger and older patients, which was thought to be independent of its diuretic effect. This drug is contraindicated in hyperkalemia and renal failure. Painful gynecomastia occurs in 10% of patients but is lower in women. New aldosterone antagonists, eplerenone appears to have lower incidence of gynecomastia.

ACE inhibitors

ACE inhibitors have been shown to reduce mortality, reduce hospitalization, reduce symptoms and increase exercise capacity in patients with heart failure and large heart (low ejection fraction). The CONSENSUS study¹⁵ (mean age 70 years) showed that in patients with severe (NYHA grade IV) heart failure, enalapril reduced the one year mortality by nearly one-third. ACE inhibitors increase myocardial oxygen consumption and increase cardiac output by decreasing afterload and reducing pulmonary venous congestion by decreasing preload. All patients with left ventricular systolic dysfunction characterized by LVEF less than 40% should be given a trial of ACE inhibitors unless contraindicated (Box-7); reducing the dose or even its stoppage is indicated if the serum potassium rises to 6 meq/L, creatinine increases by 0.75 mg to 1 mg/dl, patient develops orthostatic hypotension or severe cough, dyspnea or wheezing develops. The recommended

Box 7 : Guidelines for use of ACE inhibitors in the elderly

- Preclude bilateral renal artery stenosis, s. creatinine > 3 mg/dl
- Hospitalise if at risk of hypotension e.g., severe heart failure, systolic blood pressure < 120 mmHg
- Test dose of ACE inhibitors at night
- Increase dose slowly over weeks
- Stop potassium supplements and potassium-sparing diuretics
- Reduce dose of diuretic for 24-48 hours at the start of treatment
- Check serum K⁺ and creatinine two week after starting the treatment

maintenance dose may not be achieved in elderly but benefits occur even at lower doses.¹⁴⁻¹⁶

Co-prescription with loop diuretics not only improve symptoms or signs of all grades of heart failure but also reduce hospitalization and mortality.¹⁴⁻¹⁷ Concurrent use of NSAIDs shall be avoided as it contributes to renal dysfunction. Hydralazine and isosorbide dinitrate are inferior to ACE inhibitors in efficacy and patient acceptance¹⁸ and should be used only if ACE inhibitors are not tolerated. If cough is the troubling side effect of ACE inhibitors, Angiotensin II receptor blockers may be used.

Angiotensin receptor blockers

Attention recently has been directed towards angiotensin receptor blockade as a means of modifying

the symptoms of heart failure. There are two angiotensin receptors - AT II type 1 and AT II type 2. Stimulation of the former results in vasoconstriction, while AT II type 2 results in vasodilation. Angiotensin II type 1 receptor antagonists are effective in hypertension and CHF.

The effects of these agents in elderly patients with heart failure has been clarified recently in the ELITE^{19,20} and Val-HeFT studies²¹. Although ELITE-1¹⁹ suggested a survival advantage of the losartan over the captopril in patients >65 years of age, the larger ELITE-2²⁰ study failed to confirm this finding; one year mortality was 11.7% with losartan, and 10.4% with captopril, a statistically non significant difference. ARBs are generally well tolerated and have lower incidence of cough hence for patients who are unable to tolerate ACE inhibitors, ARB is a suitable alternative.

The addition of ARBs to patients who remain symptomatic despite maximal doses of ACE inhibitors, may improve symptoms and exercise tolerance. This combination was associated with more favorable LV remodeling than either agent alone in the RESOLVD²² trial. However, in Val-HeFT²¹ trial, the combination of ACE inhibitor and ARB (Valsartan) did not reduce the primary endpoint of all-cause mortality though it effected a 28% reduction in hospitalization and improved quality of life. This effect, however was not seen in persons who were also taking beta blockers. Considering above facts, at this point of time combination therapy cannot be recommended.

Digoxin

Digoxin is useful in management of CHF with systolic dysfunction; it is the only orally active inotropic agent available. In a group of 20 elderly patients (over 74 years) with CHF, digoxin has been shown to increase ejection fraction.²³ Digoxin is also useful in patients of CHF with atrial fibrillation.

Digoxin pharmacokinetics is altered in elderly because of diminished volume of distribution and decreased renal clearance. In non-emergency situations loading dose of digoxin should be avoided; for creatinine clearance between 10-30 ml/min, 0.125 mg daily and for creatinine clearance below 10 ml/min, 0.125 mg every 2nd or 3rd day should be given.

Digoxin has a narrow therapeutic toxic window and in elderly there is decreased inotropic effect whereas arrhythmogenic potential remains the same. Digitalis toxicity may present atypically in elderly with confusion, fatigue and irritability.

Beta blockers

There is strong evidence that beta-blockers produce important beneficial effects in patients of chronic CHF as it blocks the potentially harmful effects of circulating catecholamines. Studies with bisoprolol²⁴, metoprolol²⁵ and carvedilol²⁶ (beta-blocker with vasodilator property) have shown benefit during chronic therapy, in form of decrease in hospitalization rates and improved survival. Carvedilol has been evaluated in stable outpatients with mild to moderate chronic CHF (NYHA classification I-III) receiving usual doses of diuretics and ACE inhibitors. Carvedilol was given 3.125 mg twice daily and increased at intervals of 2 weeks to the tolerated dose (max. 50 mg). Patients need close monitoring as 10% of patients may deteriorate during initiation and up-titration. This may be managed by increasing the dose of diuretics. There were no separate trials of their use in elderly, though few trials have included elderly subjects.

Other pharmacotherapies

Calcium channel blockers have not been found to be beneficial in chronic CHF; on the other hand they may be detrimental by producing reflex tachycardia. However the PRAISE²⁷ study showed that amlodipine has a neutral effect on mortality.

Pacing in heart failure

The high morbidity and mortality associated with chronic heart failure despite optimized medical therapy has led to the development of additional interventions, such as pacemakers and defibrillators. Recently a study demonstrated that atrial synchronized biventricular pacing can improve LV function and improve functional capacity in heart failure patients with prolonged QRS (>120 milliseconds). The MIRACLE²⁸ study confirmed these findings and also demonstrated a 40% reduction in death or hospitalization for worsening of heart failure in a randomized double blind trial of 453 patients, the mean age of 64 years and the absence of an upper age limit make these findings relevant to the elderly.

Defibrillators in heart failures

Sudden cardiac death is responsible for 40% deaths in heart failure patients. Considering this fact it appears to be logical to use implantable cardiac defibrillators (ICD) in heart failure patients. Recently MADIT II²⁹ study demonstrated that prophylactic implantation of an ICD in patients with a prior MI and ejection fraction <30% reduced all cause mortality from 20% to 14% over a 20 month mean follow up. The benefit in patients 70 year old was similar to younger

adults. Thus ICD should be considered prophylactically in patients with LVEF less than 30%.

Cardiac transplantation

Cardiac transplantation is treatment of choice in younger patients suffering from end stage heart failure despite optimum medical therapy. Recently cardiac transplantation has been successfully employed to manage end-stage heart failure in selected patients in their seventh decade. In one study,³⁰ the 1 year actuarial survival was 84%, only 4% developed a serious infection and the incidence of rejection was 2.2 episode per patient. These results compared favourably with those in younger individuals. At this stage, cardiac transplantation can't be recommended as treatment of choice in end stage heart failure patients in elderly.

Drug treatment for diastolic heart failure

Evidenced based data regarding the management of diastolic heart failure is scanty. A recent update of the joint ACC / AHA³¹ Guidelines for Evaluation and Management of Heart Failure noted the absence of published trial data to guide the management of diastolic dysfunction. Diastolic dysfunction in CHF patients is associated with a better prognosis than systolic dysfunction in terms of mortality.

The general measures discussed above for systolic heart failure are also applicable to diastolic heart failure. Treating the etiology of diastolic dysfunction may completely reverse the dysfunction. Diuretics should be used for initial control of congestion and edema but should be used carefully because heart in diastolic dysfunction is already operating on low preload. Thus, aggressive use of diuretic will reduce forward cardiac output significantly.

In diastolic heart failure, the left ventricle is highly dependent on preload by atrial contraction. Thus in atrial fibrillation, loss of atrial contribution impairs LV filling significantly. Sinus rhythm should be achieved and maintained, although this is difficult in elderly where the rate of atrial fibrillation is high.

As far as the role of ACE inhibitors, ARB and beta-blockers in diastolic dysfunction is concerned, no concrete data is available on which recommendations can be made. Drugs increasing the contractility such as digoxin and sympathomimetics are of little benefit.

Summary

Heart failure is a major cause of morbidity and mortality in elderly. Hypertension, coronary artery

disease and cor pulmonale are common causes of CHF in elderly. Diastolic dysfunction contributes 40% of heart failure in elderly. Anemia, atrial fibrillation, thyrotoxicosis, and myocardial ischemia are the common precipitants of heart failure, clinical presentation of which may be atypical or masked by comorbidities. Pedal edema, pulmonary rales and fourth heart sound may be non specific in elderly. BNP has the promise to differentiate between cardiogenic and non-cardiogenic dyspnea. Negative BNP test obviates the need for 2D-Echo study. Close supervision of treatment is required. ACE inhibitors, beta blockers, low dose spironolactone are the mainstay of treatment. When there is evidence of fluid retention, patients should be treated with a diuretic. Pacing and ICD may be important therapeutic considerations in patients with prolonged QRS (>120 milliseconds) and reduced LV ejection fraction (<30%) respectively. In spite of best treatment, the overall prognosis for chronic heart failure is poor.

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The suggestions must be accompanied with reasons for recommending the particular person showing the value of his/her research and work in the field of Geriatrics. The relevant papers in connection with suggestions such as Bio-data, List of Publications, etc., should be submitted by the proposer to the secretary Indian Academy of Geriatrics.

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