21 Current Concepts in Management of Congestive Heart Failure

Abstract: Congestive heart failure is a clinical syndrome resulting from inability of the heart to pump adequate amount of blood to meet the demands of the body. Main causes include CAD, HT, DM, VHD, anemia, and alcohol. LV injury causes LV remodeling, reduced EF, arrhythmias, sudden cardiac deaths and increased mortality. The ACC/AHA classification focuses on pathophysiology and is complimentary to NYHA classification emphasizing on the aggressive management of risk factors. Main drugs are ACEIs, BBs, ARBs, diuretics, aldosterone blockers and digoxin. ACEI and BBs reduce mortality, prevent, delay or reverse LV remodeling. ACEIs are recommended in all patients as first-line therapy, with or without symptoms to improve survival, symptoms, functional capacity, and reduction of hospitalizations. BBs are the second most important drugs even in concomitant DM, PVD. ARBs can be an alternative to an ACEI, if patients cannot tolerate the latter. Diuretics are not the first line drug treatment in early stages if there are no signs of fluid retention. Aldosterone blockers are important to improve morbidity and mortality; they also reduce ventricular remodeling and myocardial fibrosis. Digoxin is indicated if HF persists despite optimal therapy with ACEI, BBs, and diuretics especially if patients have arrhythmias. CRT is recommended in advanced HF and wide QRS complex, already optimized on medical therapy. ICDs are indicated in CHF with EF less than 30%, on optimal medical therapy and serious arrhythmia. VADs originally intended for short-term use before transplantation, now with recent improvements might become more accessible to the class IV patients ineligible for cardiac transplantation. Treatment should also focus on lifestyle modifications, low sodium, low cholesterol diet, fluid restriction, compliance with medications, regular follow-up, weight management, regular exercise, supplemental oxygen, treatment of co-morbidities, aggressive treatment of HT, myocardial ischemia and lipid disorders, and avoidance of precipitating factors like infection, arrhythmias, physical or emotional stress, alcohol, NSAIDs, most CCBs and anti-arrhythmic agents. Long-term administration of positive inotropes is discouraged except for palliative reasons.

INTRODUCTION

Congestive heart failure is a clinical syndrome resulting from the inability of the heart to pump adequate amount of blood to meet the demands of the body. It can be caused by an impaired left ventricular (LV) systolic function (systolic failure) or a reduced ability of the heart to relax despite normal systolic function (diastolic failure). However, this division is arbitrary as patients with systolic failure can have diastolic failure and vice versa.[1] Clinical studies have shown that left ventricular diastolic function declines with age, whereas there is no age-related decline in contractile performance.[2,3]

CHF results from several potential injuries to the myocardium[4,5] including CAD, hypertension, valvular heart disease, diabetes, congenital heart defects, anemia, and alcoholism. Injury to the LV leads to LV remodeling defined as stretching and dilatation of the LV cavity size with subsequent reduction in ejection fraction (EF). LV remodeling following injury is an attempt to reduce wall stress and increase cardiac output by hypertrophy of viable myocytes (increase in cell length more than width). However, this process leads to an increase in mass-to-volume ratio and subsequently the vicious cycle of further increase in wall stress and premature myocyte cell death occurs.[6] The reduction in EF leads to the symptoms of CHF including dyspnea, orthopnea, paroxysmal nocturnal dyspnea, chest pain, fatigue, and overall reduced functional capacity. Also the reduction in EF is a prognostic indicator of increased mortality, arrhythmias, and sudden cardiac death.[7,8]

FORMS OF HEART FAILURE (HF)

1. **Left-sided HF** is predominant abnormality, is inadequate output by left ventricle. **Right-sided HF** is dysfunction of right ventricle secondary to left-sided heart failure or pulmonary disease.
2. **Systolic HF** is caused by impaired LV systolic function. LV remodeling occurs following injury, to reduce wall stress and to increase the cardiac output. Renin Angiotensin system and sympathetic system contributes to ventricular remodeling. As IHD is the most common cause with left ventricular systolic dysfunction, although diastolic impairment at rest is a common if not universal accompaniment. **Diastolic HF** is caused by reduced ability of heart to relax despite normal systolic function. Higher filling pressures are required to maintain cardiac output. Diastolic heart failure is often diagnosed when symptoms and signs of heart failure occur in the presence of preserved left ventricular ejection fraction at rest.
3. **In chronic HF** myocardial cells die from cytotoxic mechanisms leading to necrosis, or from the acceleration of apoptosis or programmed cell death. Necrosis stimulates fibroblast proliferation, which results in the replacement of myocardial cells with collagen, cardiac dilation and an increased afterload and wall tension, which results in further systolic dysfunction. In a**cute HF,** in previously normal individual there develops a sudden serious anatomical or functional abnormality of cardiac function, such as a massive myocardial infarction, tachyarrhythmia with a very rapid rate, or rupture of a valve secondary to infective endocarditis $\rightarrow$ a marked reduction in cardiac output occurs, with inadequate organ perfusion or acute congestion or both.
4. **High output HF** occurs in a variety of high-cardiac output states, including thyrotoxicosis, arteriovenous fistulas, beriberi, Paget disease of bone, and anemia may lead to heart failure. **Low output HF** occurs at rest or during exertion causing heart failure as it occurs in disease like congenital, valvular, rheumatic, hypertensive, coronary heart diseases.

CAUSES OF CHF

1. Hypertension
2. Diabetes mellitus

3. Dyslipidemia
4. Coronary artery disease
5. Cardiomyopathy
6. Rheumatic fever
7. Rheumatic heart disease
8. Alcohol abuse
9. Smoking
10. Thyroid disorder
11. Pheochromocytoma
12. Sleep apnea disorders
13. Cardiotoxin agents
14. Thyroid diseases
15. Neuromuscular disease
16. Peripartum cardiomyopathy
17. Collagen vascular disease
    - Cardiac amyloidosis
    - Hemochromatosis.
18. High-output heart failure
    - Arteriovenous fistula
    - Severe anemia.
19. Idiopathic
    - Chronic viral myocardial infection?
    - Autoimmune mechanisms?
    - Genetic factors?

ACC/AHA classification of CHF:[9] This takes *into account the pathophysiology of CHF.* This new classification complements the New York Heart Classification (NYHC) and is not a replacement.[10]

The ACC / AHA classification focuses on pathophysiology of CHF and emphasis on the risk factors like hypertension and diabetes and their aggressive management which in turn would reduce the evolution to symptomatic failure.

Signs and Symptoms of CHF Include the following:

- Tachycardia
- Low cardiac output
  - Fatigue or low energy
  - Pallor
  - Sweating
  - Cool extremities
  - Poor growth
  - Dizziness
  - Altered consciousness
  - Syncope
- Venous congestion
  - Right-sided
    - Hepatomegaly
    - Ascites
    - Pleural effusion
    - Edema
    - Jugular venous distension

- Left-sided
  - Tachypnea
  - Retractions
  - Nasal flaring or grunting
  - Rales
  - Pulmonary edema

MANAGEMENT STRATEGIES

The main strategies are outlined below

1. Initial evaluation of patients
   - Assess clinical severity of HF by history and physical examination
   - Assess cardiac structure and function
   - Determine the etiology of HF
   - Evaluate for coronary disease and myocardial ischemia
   - Evaluate the risk of life-threatening arrhythmia
   - Identify any exacerbating factors for HF
   - Identify co-morbidities which influence therapy
   - Identify possible causes of non-compliance.
2. Prevent precipitating and aggravating factors
   - Infection
   - Brady or tachyarrhythmia
   - Myocardial ischemia or myocardial infarction
   - Physical or emotional stress
   - Pulmonary embolism
   - High-output states such as
     i. Anemia, Thyrotoxicosis, Paget's disease
     ii. Pregnancy, Beriberi and Arteriovenous fistula.
   - Cardiac infection and inflammation
     i. Myocarditis
     ii. Infective endocarditis.
   - Comorbidities
     i. Renal, liver, thyroid insufficiency
     ii. Respiratory insufficiency.
   - Cardiac toxin
     i. Chemotherapy
     ii. Cocaine
     iii. Alcohol, etc.
3. Essential pharmacologic management with BB, ACEI/ARB, Aldosterone antagonists.
4. Complimentary treatment with diuretics or vasodilators or digoxin.
5. Device therapy including implantable cardioverter defibrillators, bi-ventricular pacing, etc.
6. Additional therapy including lifestyle modifications, etc.

Pharmacological Treatment

Principles of Drug Therapy

The pharmacological drugs commonly employed in the management of Heart Failure patients are the ACE Inhibitors (ACEI), Angiotensin receptor blockers (ARB), Betablockers (BB), Diuretics, Aldosterone blockers and Digoxin. ACEI and BB are the main treatment and should be given to patients with asymptomatic LV dilation or hypertrophy, especially in patients with prior MI even with normal EF. ACEI and BB are to reduce mortality, prevent, delay or reverse LV remodeling.

The benefits of ACEIs were proved by the SOLVD and other trials. So the ACEI enalapril significantly reduced the incidence of HF and the rate of related hospitalizations, as compared with the rates in the group given placebo, among patients with symptomatic LV dysfunction.

Besides ACEI or along with ACE inhibition, BB are also recommended as the second most important drugs for HF. BB therapy is recommended even if there is concomitant diabetes, chronic obstructive lung disease, or peripheral vascular disease. Caution should be exercised in patients with asthma, peripheral vascular disease or diabetes with recurrent hypoglycemia, patients with marked bradycardia (</= 55 beats/min) or marked hypotension (systolic blood pressure < 80 mmHg). BBs are not recommended in patients with asthma with active bronchospasm.

Diuretics are not the first line drug treatment in stage B because the patient has no sign of fluid retention. If fluid overload is to be prevented, low sodium diet and fluid restriction is the first line of therapy. Low dose diuretics can be used intermittently or as needed.

Digoxin is not indicated either unless there are atrial arrhythmias. As the benefits of BB or ACEI are supported by reduction of mortality and morbidity from randomized clinical trials (RCT), the patients should be given the same dosage at which the benefits were proven.

Angiotensin-Converting Enzyme Inhibitors

ACEIs are important therapy for CHF, leading to a mortality reduction of 15 to 20% in patients with LV systolic dysfunction (EF < 40%). In addition, ACEIs reduce the combined end point of morbidity and mortality by 30 to 35%.

The CONSENSUS trial compared enalapril to placebo and enalapril significantly reduced mortality at one year as well as hospitalizations.[11] In the SOLVD trial also enalapril significantly reduced mortality.[12,13] Enalapril also significantly reduced development of heart failure and hospitalization from heart failure.[14] Also data showed that enalapril attenuates progressive increases of LV dilatation and hypertrophy in patients with reduced LV function.[15] Several trials have noted a mortality reduction with ACEI in patients with clinical evidence of CHF after sustaining an MI.

The AIRE study showed a significant reduction in the cumulative mortality with ramipril over placebo in post-MI CHF patients. In TRACE study trandolapril reduced significantly mortality in patients with reduced LV function after an MI and also it reduced overall mortality from cardiovascular causes, sudden death, and the development of severe heart failure.[16] In the SMILE,[17] zofenopril significantly reduced the mortality and morbidity after anterior MI at six weeks and risk of death or severe CHF at one year. Zofenopril significantly improved both short-term and long-term outcome when initiated within 24 hours of MI and continued for six weeks.

Angiotensin converting enzyme (ACE) inhibitors are recommended as first line therapy in all patients, with or without symptoms, who have reduced left ventricular systolic function expressed as a reduced left ventricular ejection fraction to improve survival, symptoms, functional capacity, and reduction of hospitalizations. ACE inhibition should also be initiated in patients with signs or symptoms of heart failure, even if transient, after the acute phase of myocardial infarction, even if the symptoms are transient to improve survival, reduce re-infarctions and hospitalizations for heart failure.

Angiotensin Receptor Blockers (ARBs)

Early studies comparing ARBs and ACEI in the management of CHF patients suggested that ARB was safe and effective in these patients. In the Randomized Evaluation of Strategies for Left Ventricular Dysfunction (RESOLVD) pilot study,[18] patients received candesartan, candesartan plus enalapril, or enalapril. LV cavity size increased less and BNP levels decreased more with combination therapy compared to ARB or ACEI alone.

In ELITE trial,[19] the primary end point of death and or hospital admission for heart fail-ure was significantly lower in losartan group compared to captopril group. In the ELITE II[20] trial in the patients randomly assigned to losartan or captopril, there were no differences in all-cause mortality or sudden death between the two groups. *ELITE II also confirmed that ARB could be a potential substitute to an ACEI because ARB did not lead to inferior outcome in this study.* The Val-HeFT trial[21] randomized patients valsartan or placebo and in it the mortality was similar in both groups but the combined end point of morbidity and mortality was significantly reduced in valsartan group. Furthermore, an echocardio-graphic substudy of the Val-HeFT study showed that valsartan taken with either ACEI or beta-blockers reversed LV remodeling.[22]

In CHARM trial with Candesartan in the "overall program" of this study, which included both preserved and reduced LV function, total mortality was not reduced compared to placebo. However, in a subset analysis, patients with symptomatic heart failure and reduced LV function, candesartan significantly reduced all-cause mortality, cardiovascular death and CHF hospitalizations when added to standard therapies including ACEI, beta-blockers, and aldosterone antagonists.[23] Candesartan also reduced progression to diabetes,[24] sudden cardiac death, and death from worsening heart failure in patients with symptomatic failure.[25]

The Valsartan in Acute MI Trial[26] randomized patients after an acute MI with HF to valsartan, valsartan plus captopril, or captopril alone in addition to standard therapy. Valsartan was equally effective to captopril in reducing all cause mortality.

In the OPTIMAAL[27] trial, patients after an acute MI were randomized to losartan versus captopril. A nonsignificant difference was seen in total mortality in favor of captopril. However, there were significantly more cardiovascular deaths with losartan than with captopril and losartan was better tolerated than captopril.[28] An echocardiographic substudy of the OPTIMAAL trial has shown that both losartan and captopril improve systolic function after an acute MI, but the benefit is greater for captopril.[29]

Currently the recommendation is to use an ACEI as a first-line therapy to treat CHF patients. However, evidence now also suggests that an ARB can be an alternative to an ACEI, if patients cannot tolerate the latter.[30]

Beta-Blockers in Heart Failure

The activation of the SNS in patients with reduced LV function leads to excess catecholamine secretion which affects the myocardium contributing to LV remodeling and progression to CHF. *Multiple beta-blockers have been shown to reduce mortality and morbidity in patients with heart failure and reduced LV systolic function.* Current data support the use of carvedilol, metoprolol succinate, and bisoprolol to treat patients with CHF. Beta-blockers significantly reduce mortality when added to standard therapy in mild-to-moderate[31-33] or advanced CHF.[34] They also reduce hospitalizations[31,32,35] and reduce cardiac remodeling, cavity size and improve EF.[36]

In the US-CHF study,[31] patients were randomized to placebo or carvedilol on top of conventional therapy. *The overall mortality at six-month follow-up was significantly reduced with carvedilol, which led to early termination of the study.* It also reduced hospitalization for cardiovascular and the combined risk for hospitalization and death.

In the MERIT-HF study of metoprolol CR/XL versus placebo,[32] the all cause mortality and sudden death were reduced significantly in the metoprolol group. In post-MI patients receiving contemporary management metoprolol, CR/XL significantly reduced total mortality and sudden death.[37]

The CIBIS-II study a double-blind, placebo -controlled trial randomized to bisoprolol or placebo, at 1.3 years, all-cause mortality and sudden death were significantly reduced in patients receiving bisoprolol.[38]

The COMET trial[39,40] is the only randomized trial that compared two beta-blockers in a randomized, double-blind study in the management of CHF patients. At 58 months, there was a

significant reduction in mortality with carvedilol compared to metoprolol tartrate. Recently, carvedilol was also shown in The Glycemic Effects in Diabetes Mellitus: Carvedilol-Metoprolol comparison in Hypertensives study not to alter glycemic control in diabetics when compared to metoprolol tartrate. Furthermore, it did improve some components of the metabolic syndrome such as improving insulin sensitivity.[41]

Currently recommended beta-blockers in the management of CHF are carvedilol, metoprolol succinate, and bisoprolol.[30] Beta-blockade is still underutilized in patients with CHF despite the overwhelming data to their effectiveness and safety.[42] Educational efforts need to continue to focus on promoting guidelines in heart failure management in order to improve the overall outcome of these patients. Aggressive titration of beta-blockers is needed in patients with CHF. Higher levels of beta-blockade and ACEI are associated with better improvement of EF and greater reductions in cardiovascular hospitalizations.[43-45] A stepwise approach in titration of beta-blockade is generally followed with an increase in the dose every two weeks as tolerated until achieving the maximum tolerable dose.

Aldosterone Blockers

Aldosterone leads to sodium and water absorp-tion and the excretion of potassium. Although AII is a dominant stimulus of aldosterone secretion,[46] ACEI or ARB is not sufficient to block aldosterone secretion.[18,47,48] Recent data confirms that aldosterone blockers are important to improve morbidity and mortality in patients with CHF and reduced LV systolic function. Recent data also suggest that aldosterone blockade reduces ventricular remodeling and myocardial fibrosis and has important effects on autonomic balance, fibrinolysis, oxidative stress, and activation of the nuclear factor kappa B and activating protein-1 signaling pathways.[49]

Two RALES[50] study randomized patients to spironolactone or placebo including ACEI, digoxin, and diuretics. After a mean follow-up of 24 months, the trial was stopped early. Spironolactone significant reduced the primary end point of mortality and progression of CHF and sudden cardiac death. However, the use of spironolactone increases the risk of hyperkalemia.[51] *Patients with elevated potassium levels (>5 mEq/L) and high baseline creatinine (>2.0 mg%) should not be initiated on spironolactone* to avoid serious hyperkalemia problem and close mon-itoring of potassium levels is needed when patients are started on an aldosterone antagonist.

Another recent trial, EPHESUS[52] study randomized post-MI patients to eplerenone or placebo. Eplerenone significantly reduced total mortality, cardiovascular mortality, hospitalizations and sudden cardiac death. *The EPHESUS study established the importance of aldosterone antagonism in post-MI patients with reduced LV function irrespective of the degree of failure.*

Based on these trials, aldosterone antagonists are now considered to be a primary therapy in patients with LV dysfunction and CHF.

Current Role of Digoxin CHF

Digoxin introduced a long time ago by William Wuthering,[53] although very effective in controlling heart rate in patients with atrial fibrillation, a controversy exists about its role in the treatment of patients with CHF.[54] The Digitalis Investigation Group (DIG)[55] is a randomized, double-blind clinical trial that studied the effects of digoxin on mortality and hospitalization in patients with CHF and followed for an average of 37 months. In this trial, digoxin had no effect on mortality but did reduce the rate of hospitalization both overall and for worsening heart failure. In a sub study of the DIG trial, there was no statistically significant difference in perceived health, quality of life measures, and the six-minute walk test between the digoxin and the placebo group in patients in normal sinus rhythm at 12 month follow-up.[56] Patients on digoxin and receiving standard treatment for CHF might experience a slight reduction in

EF,[57-60] worsening maximal exercise capacity, and increased incidences of treatment failure upon withdrawal of this drug.[58,59]

Currently, digoxin is indicated for the treatment of chronic heart failure in patients with LV dysfunction and NYHC class II to III despite optimal medical treatment with ACEI, beta-blockers, and diuretics (ACC/AHA Class IIa indication) especially in patients with arrythmias. Digoxin is not indicated for the acute treatment of CHF. When digoxin is administered with amiodarone, the dose should be reduced.[61]

MECHANICAL TREATMENT OF STAGE C HEART FAILURE

Cardiac Resynchronization Therapy

Cardiac resynchronization therapy (CRT) is indicated in those subset of patients who have advanced heart failure symptoms despite optimal medical management with an EF less than 35% and a wide QRS complex > or = 130 msec. This therapy was shown to be quite safe, well tolerated, and with a high success rate.[62] CRT has also been shown to induce opposite changes in QRS duration and LV function helping in the process of reverse remodeling.[63]

It is now clear that a CRT device improves symptoms, morbidity, and mortality in patients with clear evidence of ventricular dyssynchrony, and there is no evidence, as yet, that a combined CRT-ICD device is superior to a CRT alone in these patients. In the MIRACLE trial,[64] 369 patients with severe HF, QRS duration is equal to 130 msec or greater, and class III to IV NYHC despite optimal medical treatment were randomized to controls and the CRT group. CRT improved quality of life, functional status, and exercise capacity without adversely influencing ICD function.

In the InSync III study[62] sequential CRT therapy provided a modest increase in stroke volume, improved exercise capacity. In companion trial, there was significantly reduction in the risk death and hospitalization for heart failure in the pacemaker group compared to the pharmacologic therapy alone.[65] In this trial, the addition of a defibrillator reduced mortality beyond that achieved with CRT therapy alone.

Current guidelines recommend CRT therapy in patients with advanced heart failure symptoms and wide QRS complex, who are already optimized on medical treatment with the goal to improve exercise capacity, functional status, and quality of life and to help reversing the remodeling process.[30]

Implantable Cardioverter Defibrillators

Sudden death is a major cause of mortality in patients with LV dysfunction. In the SCD-HeFT patients were randomized to conventional therapy for CHF plus placebo, conventional therapy plus amiodarone, or conventional therapy plus ICD. Amiodarone had no favorable effect on survival whereas ICD significantly reduced overall mortality at nearly 4 year follow-up.[66] In addition, the COMPANION[65] trial showed that ICD therapy can significantly reduce death in patients with advanced heart failure due to ischemic or nonischemic CM and a QRS greater than or equal to 120 msec when compared to optimal medical therapy.

The MADIT-II randomized patients with ICD or conventional medical therapy. The mortality rates were 19.8% in the conventional therapy group and 14.2% in the defibrillator group.[67,68] A long-term follow-up study from MADIT-II showed that the probability of survival after successful therapy by an ICD for ventricular fibrillation or tachycardia was 80% at one year, and these patients will be subsequently at an increased risk of heart failure and non sudden cardiac death.[69] Although advanced heart failure patients have a poorer prognosis than those with less severe failure, analysis from the MADIT-II trial indicated that benefit from ICD therapy is similar among all the different heart failure subgroups.[70] Currently the MADIT-CRT trial is ongoing and is testing whether CRT-D will reduce the risk of mortality in patients with reduced EF, prolonged QRS equal to or greater than 130 msec and NYHC Class I-II.[71]

ICDs are currently indicated in patients with moderate CHF and reduced EF less than 30% on optimal medical therapy who (i) are at least 40 days post-MI, (ii) have nonischemic CM, or (iii) have had a serious arrhythmia such as ventricular fibrillation, ventricular tachycardia, or cardiac arrest.[66-68,72]

Ventricular Assist Devices

A ventricular assist device (VAD) is used to aid the pumping action of a patient with severe heart failure. It is sometimes referred to as "a bridge to transplant" until a heart transplant can be performed. A left ventricular assist device (LVAD) receives blood from the left ventricle and delivers it to the aorta whereas the right ventricular assist device (RVAD) receives blood from the right ventricle and delivers it to the pulmonary artery. A VAD partially relieves the symptoms of severe heart failure like breathlessness and fatigue. Since many VADs are portable, patients can live at home and resume some activities while waiting for a heart transplant. VAD implant surgery carries risks of complications like bleeding, intravascular clotting, respiratory failure, kidney failure, infection, stroke, and device failure. A VAD should be used only in patients who are eligible for heart transplants or who have severe end-stage congestive heart failure and are not candidates for heart transplants. Poor candidates for VADs include people with irreversible kidney failure, severe liver disease, clotting disorders and severe lung disease.

VADs were originally intended for short-term use to support failing hearts until donor hearts became available. Recent improvements in the HeartMate VE LVAD to the HeartMate XVE LVAD have recently led to significant improvements in the outcomes[73] indicating that as technology and experience with LVAD evolve, this therapy might become more accessible to the class IV heart failure patient who is ineligible for cardiac transplantation.

Management of the Stage D CHF

Acute severe uncontrolled CHF patients with severe LV dysfunction require intense pharmacologic and mechanical management. Positive inotropic agents such as dopamine and milrinone could be used for haemodynamic stability because they improve symptoms and increase functional capacity. But they -could worsen arrhythmias and possibly increase the risk of mortality.[74,75] In a randomized trial of milrinone versus placebo in 951 patients with decompensated CHF on standard therapy milrinone caused more sustained hypotension and atrial arrhythmias compared to placebo, with no positive impact on mortality, 60 day mortality, or composite incidence of death or admission.[76] An analysis from the Acute Decompensated Heart Failure National Registry, a large retrospective registry of patients with acute decompensated CHF, showed that patients who received milrinone and dobuta-mine had a higher in-hospital mortality than those who received nitroglycerin and nesiritide. Both nesiritide and nitroglycerin had similar in-hospital mortality.[77] IV nesiritide can be utilized for acute symptomatic relief of inpatients with acute decompensated CHF.

Current ACC/AHA guidelines consider the use of intermittent positive inotropic agents for the management of decompensated heart failure as a class III indication, indicating that their use should be discouraged except for hemodynamic stability. Mechanical devices including biventricular pacing, lCD, or LVAD can be considered for eligible patients.

ADDITIONAL THERAPY

In addition to pharmacologic therapy, CHF patients should be instructed on *dietary salt restriction, daily weight monitoring, fluid restriction, smoking cessation, regular exercise, avoidance of alcohol intake and aggressive treatment of hypertension and lipid disorders.* Supplemental oxygen may be needed in patients with O_2 saturation of less than 92% on room air. Sleep apnea may be associated with

CHF, and these patients need to be screened and aggressively treated for moderate-to-severe apnoea.[78] These patients also should *avoid nonsteroidal anti-inflammatory drugs, most calcium channel blockers, and antiarrhythmics*. *Long-term administration of positive inotropes is also discouraged except for palliative reasons*. Exercise testing should be done to determine the appropriate level of activity.

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MULTIPLE CHOICE QUESTIONS

1. Regarding left ventricular function, following statements are false:

A. Systolic function generally declines with age
B. Diastolic function generally declines with age
C. Diastolic dysfunction can occur as isolated entity
D. Diastolic dysfunction often accompanies systolic dysfunction

2. In the new ACC/ AHA classification for CHF, following are true:

A. The classification is targeted towards pathophysiology of CHF
B. Stage –A is equivalent to NYHA class-I
C. Stage –C is equivalent to NYHA class-II and III
D. Stage B is - Asymptomatic patients with LV dysfunction

3. Identify the false statements regarding treatment of CHF:

A. Diuretics are the first drugs of choice of in all patients of CHF
B. AR Blockers are preferred over ACE inhibitors in all patients
C. In all diabetics and COPD, beta-blockers are contraindicated
D. Digoxin is not indicated unless there are atrial arrhythmias

4. Identify the false statement regarding ACEI / ARB in CHF:

A. CONSENSUS, AIRE and SMILE studies evaluated role of ARBs in HF
B. ARB can be alternative to ACE-I in CHF, if patients cannot tolerate ACEI
C. ACE inhibitors should be started in all patients of acute MI
D. ACE inhibitors should be started in all patients of reduced LV systolic function even if asymptomatic

5. Regarding beta-blockers in CHF, following treatments are true:

A. Beta-blockers are indicated in most patients of HF
B. Carvedilol is the first drug to demonstrate the beneficial role of beta-blockers in CHF
C. Carvedilol significantly alters the glycemic control in diabetes
D. If tolerated, higher doses of beta-blockers and ACE-I are associated with better improvement in EF

6. Regarding management of CHF, following treatments are true:

A. Based on RALES and EPHESUS trials aldosterone antagonist are now considered to be a primary therapy in patients with LV dysfunction and CHF
B. Potassium levels > 5 mEq /L and creatinine >2 mg% are contraindications for initiation of spironolactone.
C. Digoxin is indicated in all patients for acute treatment of CHF
D. When digoxin is administrated with amiodarone, the dose should be reduced.

7. Regarding cardiac resynchronization therapy (CRT), following are true:

A. It is indicated in advanced uncontrolled HF despite full medical management and with EF < 35, QRS < / =136 msec
B. CRT helps to reverse LV remodeling
C. CRT improves exercise capacity and quality of life
D. MIRACLE and CAMPANION trial evaluated the roll of CRT in HF

8. Regarding implantable cardioverter defibrillators, following are true:

A. In SCD-HeFt trial amiodarone had no favorable effect on survival whereas ICD significantly reduced overall mortality at nearly 4 years follow-up
B. MEDIT-II trial indicated benefit of ICD therapy in all different heart failure sub-group uncontrolled with conventional therapy

C. 40 days period is required in post-MI who have HF and need ICDs
D. Ventricle assist device generally are used as permanent therapy for intractable HF

9. In management of stage – D CHF, following are false:

A. Use of dopamine and other positive ionotropic drugs increases survival rate
B. Milrinone can cause hypotension and atrial arrhythmia as complications
C. IV nesiritide can be utilized for acute symptomatic relief of inpatients with acute de-compensated CHF
D. Mechanical devices including biventricular pacing, ICD or LVAD can be considered for eligible patients

10. Following are important in management of CHF *except:*

A. Salt and fluid restriction
B. Regular tailored exercise
C. Supplemental oxygen if O_2 saturation is < 80% at room air
D. Patients should avoid NSAID, CCB, anti-arrhythmics

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8. A, B, C 9. A 10. C