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Medical Advances in the Evaluation and Treatment of Heart Failure

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All emergency physicians are comfortable caring for the patient in acute pulmonary edema. Initial treatment of this emergency condition has changed little in the past decade. However the treatment of less acute patients with congestive heart failure (CHF) has changed dramatically in the recent past. This article will emphasize the management of a patient with congestive heart failure. The ED physician increasingly is part of the health care team, working with the primary care physician. As America "grays," the ED physician will be increasingly involved in the care of these patients and their chronic care as well as emergency stabilization.

—Sandra M. Schneider, MD, FACEP, Editor

Introduction

Heart failure is a clinical syndrome that is characterized by the inability of the heart to meet the body's metabolic demands. This is attributed to either a functional or structural impairment of ventricular filling or ejection of blood. Patients manifest clinically with symptoms of low exercise tolerance and fluid retention.

Heart failure is a major public health problem in the western world. In the United States alone there are about 5 million Americans living with the diagnosis of heart failure. It is also estimated that another 550,000 Americans are diagnosed with this condition every year.[1] Heart failure accounts for about 15 million physician office visits every year.[2] It is the most common Medicare discharge diagnosis, accounting for more healthcare costs than any other condition in the United States.[3] The overall prevalence of heart failure is increasing. This increase has been attributed to the aging population and the advances in the treatment of coronary artery disease (CAD) that have led to more people surviving initial events of acute myocardial infarction.[4] Heart failure is primarily a disease of the elderly, as evidenced by the fact that 80% of the patients admitted with heart failure are older than 65 years.[5] The incidence of heart failure among people aged 65 years and older is about 10 per 1000 population.[1] The estimated direct and indirect cost of heart failure in the United States is $37.2 billion.[6]

Risk Factors

Hypertension is the most common risk factor for heart failure, followed by antecedent acute myocardial infarction (AMI).[7] Other risk factors include diabetes, metabolic syndrome, and dilated cardiomyopathy. (*See Table 1.*) Diabetes mellitus substantially increases the risk of developing heart failure, especially among post-menopausal women with established CAD. Heart failure incidence increases with each additional risk factor. For example, the annual incidence of heart failure among nondiabetic women with no risk factors is 0.4%. This

Executive Summary

- Common risk factors for congestive heart failure include hypertension, coronary artery disease, diabetes, obesity, and cardiomyopathy.
- Although brain natriuretic peptide correlates highly with symptoms of heart failure, other conditions such as advanced age, obesity, cirrhosis, and hypoxia also can increase values.
- ACE inhibitors are recommended in most patients with CHF but must be used in high doses to show reductions in mortality.
- Aldosterone antagonists have been shown to reduce mortality and hospital admissions in patients with NYHA class III or IV heart failure.
- Patients with advanced heart disease can have up to 50% one-year mortality, with half of these deaths being sudden.

risk increases to 3.4% in nondiabetic women with at least three risk factors. Among diabetic patients with no additional risk factors, the annual incidence of heart failure is 3.0%, compared with 8.2% among diabetics with at least three additional risk factors.[8] Dilated cardiomyopathy accounts for a substantial number of cases of heart failure. About one-third of patients with cardiomyopathy may have a genetic predisposition.[9]

Classification of Heart Failure

There are numerous ways of classifying heart failure and, in many cases, these categories overlap and may be ambiguous. The basis of these definitions has varied widely and has ranged from anatomical (right- vs left-sided heart failure), physiological (diastolic vs systolic), and, in some instances, clinical considerations (acute vs chronic). The World Health Organization (WHO) classification is based on myocardial disorder, i.e., dilated, hypertrophic, restrictive, arrhythmogenic right ventricular, and unspecified cardiomyopathies.[10] In recognition of the fact that heart failure is a progressive disorder, the American Heart Association (AHA) and American College of Cardiology (ACC) societies in collaboration have developed the staging criteria that take into account the development and progression of the disease. (*See Table 2.*) This classification has four stages: A through D. Stage A identifies patients who are at high risk for developing heart failure but have no structural abnormalities of the heart and have no symptoms of heart failure. These are patients who might have risk factors for developing heart failure, such as hypertension, diabetes, CAD, history of alcohol abuse, or exposure to cardiotoxins.

Stage B includes patients with structural heart abnormalities but with no overt symptoms of heart failure. Patients in this stage might have left ventricular hypertrophy (LVH), reduced left ventricular ejection fraction (LVEF), valvular disease, and prior AMI but are asymptomatic.

Stage C includes patients who have all the factors in stages A and B, and in addition have symptoms of clinical heart failure. They not only have structural abnormality of the heart but also manifest clinical symptoms and signs of heart failure.

Stage D includes patients who are refractory to standard treatment of heart failure. These patients may require intravenous pressor agents, mechanical assist devices, heart transplant, or hospice care. In the ACC/AHA classification, patients can progress from one lower stage to a higher stage but never the reverse (i.e., from stage A to B, but never from B to A).

Another more clinically based classification is the New York Heart Association (NYHA) classification. This classification uses the presence or absence of symptoms of heart failure on functional capacity to classify severity of heart failure.[11] There are four classes in this classification: I through IV. (*See Table 3.*)

Causes of Heart Failure

The most common cause of heart failure in the Western world is attributed CAD and often may be secondary to an AMI. CAD accounts for about 70% of the cases heart failure.[12] Heart failure also can result from nonischemic causes such as hypertension, cardiomyopathies, valvular disorders, pericardial disorders, or arrhythmias. See Table 4 for comprehensive list of etiologies of heart failure.

Pathophysiology

Numerous conceptual models have been proposed to explain heart failure; however, no single model effectively explains the complexity of the heart failure syndrome. Earlier models conceived heart failure as a result of excessive salt and water retention leading to poor renal flow (cardiorenal model).[13] Following this was the hemodynamic model that conceived heart failure as a problem of "pump failure" with resultant excessive peripheral vasoconstriction.[14] These models could not, however, explain the progression of heart failure once the original insult causing heart failure was removed.

More recently, the neuro-hormonal model has attempted to explain the progression of heart failure as resulting from the effects of biochemical molecules released in response to injury of the heart.[15] These biomedical chemicals include catecholamines, through the activation of the adrenergic nervous system (ANS); and angiotensin II and aldosterone through the activation of

Table 1. Risk Factors for Heart Failure

- Hypertension
- Coronary artery disease
- Diabetes
- Obesity
- Metabolic syndrome
- Use of cardiotoxins (illicit drug use)
- Family or personal history of cardiomyopathy
- Structural heart abnormalities (such as valvular abnormalities)

the rennin-angiotensin-aldosterone system (RAAS). With a reduction in cardiac output resulting from an insult on the heart (index event), compensatory mechanisms are set in motion with the goal of maintaining adequate cardiac output for tissue perfusion. An activated ANS and RAAS results in the over-expression of catecholamines, angiotensin II, and aldosterone, respectively. These biochemical molecules have profound maladaptive effects on myocardial cells. Some of these effects include loss of myocytes, hypertrophy, and fibrosis of the myocardium (also known as remodeling). The overall result is that the heart configuration changes from the elliptical shape to a more spherical shape, which is hemodynamically less efficient for pumping out blood. Patients may be asymptomatic after the index event, but with persistence of these maladaptive changes, patients develop overt symptoms of heart failure.

Therapeutic agents such as beta-blockers and angiotensin-converting enzyme inhibitors (ACEI) have been shown to improve survival in heart failure by ameliorating the effects of these maladaptive changes through directly acting on the ANS and RAAS, respectively.[16-18] This conceptual framework helps to explain the changes in the development of systolic dysfunction (heart failure with depressed ejection faction, less than 45%). However, the maladaptive changes in diastolic dysfunction (heart failure with preserved ejection fraction) are not very well understood.

Evaluation

Evaluation of patients with heart failure begins with a thorough history and physical examination. A detailed history should elicit patients' risk factors or behaviors associated with the development and progression of heart failure. Direct inquiry about past or current history of CAD, congenital heart disease, valvular diseases, and illicit drug use may help point to etiology of heart failure. It is also important to establish the baseline functional status using the NYHA classification for future monitoring of therapy and progression of the disease.

Many tests routinely are used to evaluate patients with heart failure. Diagnostic tests usually are better at detecting heart failure with systolic dysfunction than heart failure with preserved systolic function. Laboratory evaluation should include tests that reveal disorders that will lead to or cause progression of heart failure. Initial studies should include urinalysis, complete blood count, ECG, and basic metabolic profile. (*See Table 5.*)

Chest X-ray (CXR) allows the physician to assess pulmonary congestion as well as rule out other respiratory causes of shortness of breath. In chronic heart failure, about 20% of patients may not have pulmonary markings of congestion on CXR because of compensatory increase in lymphatic drainage of fluid.[19] One of the most useful diagnostic tests in evaluating heart failure is the 2-dimension echocardiogram (2-D echo). This test noninvasively assesses the functional capacity of the cardiac chambers and the presence of structural abnormalities. It also establishes a baseline reference point for future comparison of heart failure progression. In special situations in which myocardial disease is suspected, such as myocarditis, cardiac magnetic resonance imaging (MRI) can be done to evaluate for this disorder.[20] Myocardial biopsy is reserved for patients with acute fulminant heart failure of unknown etiology with ventricular arrhythmias and/or atrioventricular conduction delays, or in patients suspected of having infiltrative processes such as amyloid, hemochromatosis, and restrictive cardiomyopathy of unknown etiology.

Serum assays of brain natriuretic peptide (BNP) and N-terminal pro BNP (NT-proBNP) have been shown to correlate highly with symptoms of heart failure, particularly in systolic dysfunction.[21] BNP is released from distended heart muscle as occurs in volume overload states. A number of patient factors besides heart failure can cause elevated levels. Factors that can elevate these serum assays include advanced age, obesity, liver cirrhosis, tachycardia, hypoxemia, and renal function.[22] A normal level of BNP in a patient not receiving treatment has a high negative predictive value for heart failure.

Therapy

Treatment of HF is linked to the ACC/AHA staging guidelines.

Stage A. The goal of management of stage A heart failure is the prevention of progression of disease by treating risk factors. Adequately treated hypertension can lead to profound reduction in the incidence of heart failure. In some studies, optimally treating hypertension translated to as much as a 30-50% reduction in the development of left ventricular hypertrophy and heart failure.[23] Blood pressure readings may be falsely high in the ED and a single elevated reading should not be used to initiate treatment. However, individuals with elevated pressures should be referred to their primary care provider for further evaluation and possible treatment. Patients who smoke, drink alcohol, or use drugs are counseled to abstain from these risky behaviors.

Table 2. Classification of Heart Failure (ACC/AHA)

Stage A
At high risk for developing heart failure. No evidence of structural or functional abnormality. No signs or symptoms of heart failure.
Stage B
Presence of structural heart disease without evidence of signs or symptoms.
Stage C
Symptoms and signs of heart failure associated with underlying structural heart disease.
Stage D
Advanced structural heart disease with marked symptoms of heart failure at rest despite maximal medical therapy.

Source: Hunt SA, et al. *Circulation* 2005;112:1825-1852.

Stages B, C, D. The goal of therapy for patients with these stages of heart failure is to decrease the progression of disease, improve symptoms, and minimize the risk factors for the development and progression of the disease. Some of the interventions include salt restrictions and avoidance of nonsteroidal anti-inflammatory drugs (which can cause fluid retention and worsening of heart failure).[24] There are also specific therapeutic agents that have been shown to have improved outcomes in patients with heart failure that should be considered. (*See Table 6.*)

ACE Inhibitors

The use of ACEIs is recommended for most patients with stages B, C, and D. These classes of medications have been shown to confer survival benefit to patients with heart failure, after myocardial infarction, and improve heart failure symptoms and reverse remodeling by blunting the activity of the RAAS.[25-27] The effects of these medications are not dose-dependent. The effects of low-dose ACEI on mortality have not been any different than in those patients taking high-dose regimens in most randomized clinical trials.[28-31] A common adverse side effect of ACEIs is angioedema.

Beta-Blockers

Beta-blockers have been shown to confer clinical benefits in patients with all stages of heart failure. The benefits that beta-blockers confer include improved survival, reduced morbidity, improved quality of life, reduced rate of hospitalizations, improvement in remodeling, and reduced incidence of sudden cardiac death.[32-33] Studies have shown improvements in the systolic function and reversal of remodeling with just 3-4 months of treatment with beta-blockers.[34-36] In some cases the improvement in mortality and hospitalization was seen as early as 14-21 days after initiation of therapy.[37] Beta-blockers should be initiated when the patient is hemodynamically stable at low doses and titrated slowly over 2-4 weeks. Patients often have difficulty with hypotension and fatigue after starting beta-blockers and may present to the ED. Every effort should be made to continue this medication, as symptoms will decrease with time. Caution should be exercised in patients with hyperactive airways disease, bradyarrhythmias, and in patients known to be diabetics with frequent hypoglycemic episodes.

Angiotensin Receptor Blockers (ARBS)

ARBs block the effects of angiotensin II at the angiotensin II type 1 receptor site. ARBs are comparable but not superior to ACE inhibitors.[38-40] There are reports of increased adverse events in patients who were receiving a combination of ACEIs and ARBs.[41-42] ARBs, therefore, generally are recommended in patients who are not able to tolerate ACEIs due to cough and angioedema.

Aldosterone Antagonists

Aldosterone antagonists are another class of medications that have a beneficial role in heart failure.[43] Patients with heart failure have elevated levels of aldosterone, leading to salt and water retention. Aldosterone also works locally on the heart muscle to induce myocardial fibrosis and hypertrophy. Aldosterone antagonists counteract these effects, thereby inhibiting the remodeling process.[44] The addition of these agents in patients with advanced heart failure (NYHA class III or IV) has been shown in randomized controlled studies to improve mortality outcomes and hospital admissions for heart failure.[45] Patients on aldosterone antagonists must be monitored closely for serum potassium and creatinine. These medications generally are not started if the serum potassium is more than 5.0 mmol/L or the serum creatinine is more than 2.5 mg/dL.

Diuretics

Diuretics are usually the initial medications administered to patients who are symptomatic (stage C and D heart failure). These medications are effective at reducing the pulmonary congestion and cardiac afterload.[46] The combination of loop diuretics with thiazide can be effective in optimizing diuresis in advanced cases.[47-48]

Digoxin

Digoxin can be used in symptomatic patients with low ejection fraction. It has been shown in randomized controlled studies to reduce hospitalization and improve heart failure symptoms. It does not affect survival outcomes, however.[49] It is important to monitor electrolytes and renal function in patients receiving digoxin to prevent toxicity. Low

Table 3. NYHA Functional Classification of Heart Failure

Class I
No limitation of physical activity. Ordinary physical activity does not cause undue fatigue or dyspnea.
Class II
Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue or dyspnea.
Class III
Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity results in fatigue, palpitation, or dyspnea.
Class IV
Unable to carry on any physical activity without discomfort. Symptoms at rest.

Source: New York Heart Association

Table 4. Causes of Heart Failure

Cause	Comments
Ischemic heart disease	CAD is the most common cause
Valvular heart disease	Primary disorder is valve abnormality
Hypertension	Commonly due to LVH leading to diastolic dysfunction and not uncommonly systolic dysfunction.
Diabetes	Associated with diastolic/systolic failure
Infections	Viral myocarditis, bacterial endocarditis, and rheumatic fever are examples
Arrhythmias	Tachycardias such as atrial fibrillation
Metabolic causes	Hypo/hyperthyroidism, Paget's disease, hypophosphatemia
Systemic diseases	Connective tissue diseases such as systemic lupus erythematosus (SLE)
Toxin	Cocaine, alcohol, certain chemotherapeutic agents
Genetic disorder	Cardiomyopathies
Pregnancy	Post-partum cardiomyopathy
CAD = Coronary artery disease; LVH = Left ventricular hypertrophy	

serum digoxin concentration (lower than 0.09 ng per milliliter) can be as effective as higher therapeutic ranges previously recommended in maintaining therapeutic response.[50] Digoxin also is beneficial in patients with concomitant atrial fibrillation and systolic dysfunction, as it can be used to control ventricular rate in patients with atrial fibrillation. Contraindications for digoxin include second- or third-degree heart block (without a permanent pacemaker), pre-excitation syndromes, and previous evidence of digoxin intolerance.

Hydralazine and Isosorbide Dinitrate

In patients with heart failure and systolic dysfunction who are unable to tolerate ACEIs or ARBs, a combination of hydralazine and isosorbide dinitrate could be considered. This combination therapy has been shown to reduce mortality and hospitalization and to improve quality of life.[51-53] Evidence of benefit is strongest in African-Americans. The medications must be titrated over a period of 2-4 weeks. This combination is contraindicated in patients with symptomatic hypotension, lupus syndrome, and severe renal failure.

Diastolic Heart Failure

Diastolic heart failure deserves special mention because of the high prevalence of this disorder; yet, there is still a paucity of outcome data from long-term randomized placebo-controlled trials. Most of the available data on the therapeutic interventions have been in heart failure with systolic dysfunction. Diastolic heart failure is defined by the presence of heart failure symptoms in a patient with normal left ventricular ejection fraction and no valvular abnormalities on echocardiography.[54] Approximately 50% of patients with a diagnosis of heart failure have normal or preserved left ventricular function.[55] The prevalence of diastolic heart failure is highest in patients older than 75 years, and most often in women.[56]

The preeminent problem in diastolic heart failure is the impaired ability of the ventricles to relax, leading to increased end-diastolic ventricular pressures in relation to a given ventricular blood volume.

Table 5. Common Laboratory Tests in the Initial Evaluation of Heart Failure

Lab Test	Clinical Comments
Urinalysis	Rule out infections, proteinuria, glycosuria for diabetes
Complete blood count	Anemia can precipitate heart failure or can be a complication of infective endocarditis and other systemic diseases. High white blood cell count may indicate presence of infection.
Basic metabolic panel	Evaluate for hyperglycemia. Creatinine may indicate presence of renal failure and may necessitate heart failure therapy adjustment, i.e., ACEI/ARB or aldosterone blocker. Hypokalemia may be complication of diuretics.
Thyroid function test	Hyperthyroidism and hypothyroidism can both precipitate heart failure. Amiodarone therapy can complicate thyroid function.
Iron (Fe) studies	Anemia from iron deficiency can precipitate heart failure. Hemochromatosis is one cause of heart failure.
B-type natriuretic peptide	Elevated levels may make diagnosis of heart failure likely.
Elevated cardiac markers	May suggest the AMI in the setting of CAD as etiology of heart failure
Lipid profile	To guide the treatment of cardiovascular risk reduction
Liver function test	Elevated levels maybe seen hepatic congestion as a complication of heart failure.

Thus with just a slight increase in blood volume, blood pressure, or even the presence of tachycardia in a patient with impaired ventricular relaxation could lead to substantial elevation of left atrial pressures and pulmonary edema. Treatment in the acute setting involves diuresis as well as aggressively treating hypertension and controlling heart rate. Current long-term treatment recommendations have been generally extrapolated from the interventions used in the treatment of heart failure with systolic dysfunction. The use of beta-blockers, ACEIs, and calcium channel blockers generally is recommended in diastolic heart failure.[57-60] In the CHARM-preserved study, an angiotensin receptor blocker was associated with lower hospitalization and a non-statistically significant trend toward lower mortality.[61]

Nonpharmacological Therapies

There are nonpharmacological approaches to treatment of some patients with heart failure. All patients with heart failure must be evaluated and treated for CAD. Angioplasty and surgical revascularization can lead to improvement of ischemic symptoms and ejection fraction, and can reduce the incidence of sudden death.[62] Some patients with stage C and D heart failure may benefit from bypass surgery. Some studies are looking at the role of surgical procedures designed for the treatment of heart failure. Some of these procedures include mitral valve repair, mechanical devices to reduce wall stress, and surgical excision of infarcted tissue.[63-65]

Mechanical devices for stage D patients may provide a bridging gap modality in patients awaiting heart transplantation or even those patients who are not candidates for transplant surgery.[65] Cardiac resynchronization therapy with biventricular pacers has been shown to be effective in the treatment of patients with heart failure and left bundle-branch blocks on a 12-lead electrocardiogram. These devices have been shown to improve exercise tolerance and quality of life and to reduce the rate of hospitalizations. Resynchronization therapy also been to shown to reverse the remodeling effects and improve the ejection fraction of the heart.[66-69]

Sudden Cardiac Death

Patients with heart failure are at an increased risk of sudden cardiac death.[70] Sudden death usually results from ventricular tachyarrhythmias. Sudden death resulting from cardiac causes accounts for about 50% of all cardiovascular deaths.[71] Patients with advanced heart disease such as those with systolic dysfunction and NYHA class III or IV may have up to 50% one-year mortality, with half of these deaths being sudden.[72] Patients with severe systolic dysfunction presenting with syncope, for instance, may need to be referred for electrophysiological testing for inducible arrhythmias

Table 6. Commonly Used Drugs in Heart Failure

Drug	Dosage Range	Drug Class Comments
ACEI		
Captopril	6.25 mg tid to 50 mg tid	Needs close monitoring of electrolytes and renal function.
Enalapril	2.5 mg bid to 10-20 mg bid	
Lisinopril	2.5-5 mg bid to 20-40 mg bid	Cough as side effect common
Ramipril	1.25-2.5 mg qd to 10 mg qd	
Beta-Blockers		
Carvedilol	3.125 mg bid to 25-50 mg bid	Can cause worsening of HF, bradycardia, hypotension, and bronchial spasms.
Bisoprolol	1.25 mg qd to 10 mg qd	
Metoprolol succinate	12.5-25 mg qd to 200 mg qd	
Digitalis Glycosides		
Digoxin	0.125 mg qd to 0.25 mg qd	Narrow therapeutic window, monitor levels in patients with renal disease, elderly and with hypokalemia. No benefit on survival but shown to have effect on morbidity.
Aldosterone Inhibitors		
Spironolactone	25 mg qd to 50 mg qd	Electrolytes and renal function should be monitored carefully, especially in patients taking concomitant ACEI.
Eplerenone	25 mg qd to 50 mg qd	
Angiotensin Receptor Blockers		
Candesartan	8 mg qd to 32 mg qd	As effective as ACEI. Use these agents if patient is not able to tolerate ACEI due to cough or angioedema.
Irbesartan	75 mg qd to 300 mg qd	
Losartan	25 mg qd to 100 mg qd	
Valsartan	80 mg qd to 320 mg qd	

and possible insertion of mechanical devices.

Summary

Heart failure is a debilitating progressive disease that has severe morbidity and mortality. The prevalence of heart failure is increasing because of the aging population. All patients who present with CHF should be referred to their primary care physician for chronic therapy. Many patients recently started on treatment may experience side effects of fatigue, hypotension, and even worsening dyspnea. When possible, treatment should be continued or slowly tapered as these treatments lead to remodeling of the heart and better long-term outcomes. A promising report from the Framingham study showed that there was increased survival with the diagnosis of heart failure.[73] Despite this,

evidence shows that treatment of heart failure is less than optimal.[74]

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Physician CME Questions

51. A patient presents with no symptoms of heart failure but has a long-standing history of hypertension and is noted to have left ventricular hypertrophy on echocardiography. Which ACC/AHA classification stage of heart failure does he have?
    A. stage A
    B. stage B
    C. stage C
    D. stage D

52. All of the following combinations of medication have been shown to reduce mortality in patients with heart failure *except*:
    A. ACEIs and beta-blockers
    B. hydralazine-isorsobide dinitrate
    C. digoxin and a diuretic
    D. ARBs and beta-blockers

53. A 70-year-old man with a prior history of hypertension was recently diagnosed with stage B heart failure. His echocardiogram showed an ejection fraction of 40%. He was started on an ACEI in addition to a beta-blocker for optimal management of his heart failure. Two weeks after discharge from the hospital, he developed a dry irritating cough. He has no signs of fluid overload. What should be the next step?
    A. Start diuretic therapy.
    B. Stop ACEI and start an ARB.
    C. Add an aldosterone antagonist.
    D. Start digoxin.

54. A patient with a history of hypertension presents with increasing dyspnea of 14 days duration. She responds to treatment in the ED and feels well enough to be discharged. What is the best course of action now?
    A. The patient should be started on ACEIs, beta-blockers, aspirin, and digoxin in the ED.
    B. She should see her primary care physician in 1-2 days.
    C. She can wait to see her primary care physician in 1 month.
    D. She should be admitted to rule out a myocardial infarction.

55. All of the following conditions may cause an elevated brain natriuretic peptide level *except*:
    A. cirrhosis of the liver
    B. renal failure
    C. stroke
    D. obesity

56. A patient presents 2 days after starting beta-blockers for CHF. He was just started on a beta-blocker and an ACEI. He is feeling fatigued. His blood pressure is 100/70 and pulse is 60. His ECG is unchanged. All other lab work is normal. The best course of action is:
    A. Reassure the patient that his symptoms will gradually improve.
    B. Stop the beta-blocker.
    C. Stop the ACEI.
    D. Stop all of his medications.

57. Digoxin is used in CHF to:
    A. remodel the ventricle
    B. treat diastolic hypertension
    C. control the rate in patients with atrial fibrillation
    D. decrease peripheral vascular resistance

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion*. When your evaluation is received, a certificate will be mailed to you.

Emergency Medicine Reports

CME Objectives

Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

58. A patient with congestive heart failure presents with angioedema. Which of the following medications is likely the cause?
    A. ACEI
    B. digoxin
    C. beta-blocker
    D. loop diuretic

59. Patients with severe heart failure have a high mortality rate. Which of the following is the most common cause of death?
    A. stroke
    B. renal failure
    C. sudden death
    D. pulmonary embolism

60. Patients with severe heart failure and a left bundle-branch block may benefit from:
    A. a biventricular pacemaker
    B. aggressive diuretic
    C. digoxin
    D. physical training

CME Answer Key

51. B; 52. C; 53. B; 54. B; 55. C; 56. A; 57. C; 58. A; 59. C; 60. A

In Future Issues ENT Emergencies

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Medical Advances in Heart Failure

Risk Factors for Heart Failure

- Hypertension
- Coronary artery disease
- Diabetes
- Obesity
- Metabolic syndrome
- Use of cardiotoxins (illicit drug use)
- Family or personal history of cardiomyopathy
- Structural heart abnormalities (such as valvular abnormalities)

Classification of Heart Failure (ACC/AHA)

Stage A
At high risk for developing heart failure. No evidence of structural or functional abnormality. No signs or symptoms of heart failure.
Stage B
Presence of structural heart disease without evidence of signs or symptoms.
Stage C
Symptoms and signs of heart failure associated with underlying structural heart disease.
Stage D
Advanced structural heart disease with marked symptoms of heart failure at rest despite maximal medical therapy.

Source: Hunt SA, et al. *Circulation* 2005;112:1825-1852.

NYHA Functional Classification of Heart Failure

Class I
No limitation of physical activity. Ordinary physical activity does not cause undue fatigue or dyspnea.
Class II
Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue or dyspnea.
Class III
Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity results in fatigue, palpitation, or dyspnea.
Class IV
Unable to carry on any physical activity without discomfort. Symptoms at rest.

Source: New York Heart Association

Causes of Heart Failure

Cause	Comments
Ischemic heart disease	CAD is the most common cause
Valvular heart disease	Primary disorder is valve abnormality
Hypertension	Commonly due to LVH leading to diastolic dysfunction and not uncommonly systolic dysfunction.
Diabetes	Associated with diastolic/systolic failure
Infections	Viral myocarditis, bacterial endocarditis, and rheumatic fever are examples
Arrhythmias	Tachycardias such as atrial fibrillation
Metabolic causes	Hypo/hyperthyroidism, Paget's disease, hypophosphatemia
Systemic diseases	Connective tissue diseases such as systemic lupus erythematosus (SLE)
Toxin	Cocaine, alcohol, certain chemotherapeutic agents
Genetic disorder	Cardiomyopathies
Pregnancy	Post-partum cardiomyopathy
CAD = Coronary artery disease; LVH = Left ventricular hypertrophy	

Common Laboratory Tests in the Initial Evaluation of Heart Failure

Lab Test	Clinical Comments
Urinalysis	Rule out infections, proteinuria, glycosuria for diabetes
Complete blood count	Anemia can precipitate heart failure or can be a complication of infective endocarditis and other systemic diseases. High white blood cell count may indicate presence of infection.
Basic metabolic panel	Evaluate for hyperglycemia. Creatinine may indicate presence of renal failure and may necessitate heart failure therapy adjustment, i.e., ACEI/ARB or aldosterone blocker. Hypokalemia may be complication of diuretics.
Thyroid function test	Hyperthyroidism and hypothyroidism can both precipitate heart failure. Amiodarone therapy can complicate thyroid function.
Iron (Fe) studies	Anemia from iron deficiency can precipitate heart failure. Hemochromatosis is one cause of heart failure.
B-type natriuretic peptide	Elevated levels may make diagnosis of heart failure likely.
Elevated cardiac markers	May suggest the AMI in the setting of CAD as etiology of heart failure
Lipid profile	To guide the treatment of cardiovascular risk reduction
Liver function test	Elevated levels maybe seen hepatic congestion as a complication of heart failure.

Commonly Used Drugs in Heart Failure

Drug	Dosage Range	Drug Class Comments
ACEI		
Captopril	6.25 mg tid to 50 mg tid	Needs close monitoring of electrolytes and renal function.
Enalapril	2.5 mg bid to 10-20 mg bid	
Lisinopril	2.5-5 mg bid to 20-40 mg bid	Cough as side effect common
Ramipril	1.25-2.5 mg qd to 10 mg qd	
Beta-Blockers		
Carvedilol	3.125 mg bid to 25-50 mg bid	Can cause worsening of HF, bradycardia, hypotension, and bronchial spasms.
Bisoprolol	1.25 mg qd to 10 mg qd	
Metoprolol succinate	12.5-25 mg qd to 200 mg qd	
Digitalis Glycosides		
Digoxin	0.125 mg qd to 0.25 mg qd	Narrow therapeutic window, monitor levels in patients with renal disease, elderly and with hypokalemia. No benefit on survival but shown to have effect on morbidity.
Aldosterone Inhibitors		
Spironolactone	25 mg qd to 50 mg qd	Electrolytes and renal function should be monitored carefully, especially in patients taking concomitant ACEI.
Eplerenone	25 mg qd to 50 mg qd	
Angiotensin Receptor Blockers		
Candesartan	8 mg qd to 32 mg qd	As effective as ACEI. Use these agents if patient is not able to tolerate ACEI due to cough or angioedema.
Irbesartan	75 mg qd to 300 mg qd	
Losartan	25 mg qd to 100 mg qd	
Valsartan	80 mg qd to 320 mg qd	

Supplement to *Emergency Medicine Reports*, August 30, 2010: "Medical Advances in the Evaluation and Treatment of Heart Failure." *Authors:* **Sula Mazimba, MD, MPH,** Cardiology Fellow, Kettering Medical Center, Kettering, OH; **Trupti Patel, MD**, Jefferson School of Population Health, Philadelphia, PA; and **Nakash Grant, MD**, Wright State University Boonshoft School of Medicine, Dayton, OH.