MIDSOUTH Wellness GUIDE

February 2007 **"America's Wellness Guide"** **Number 43**

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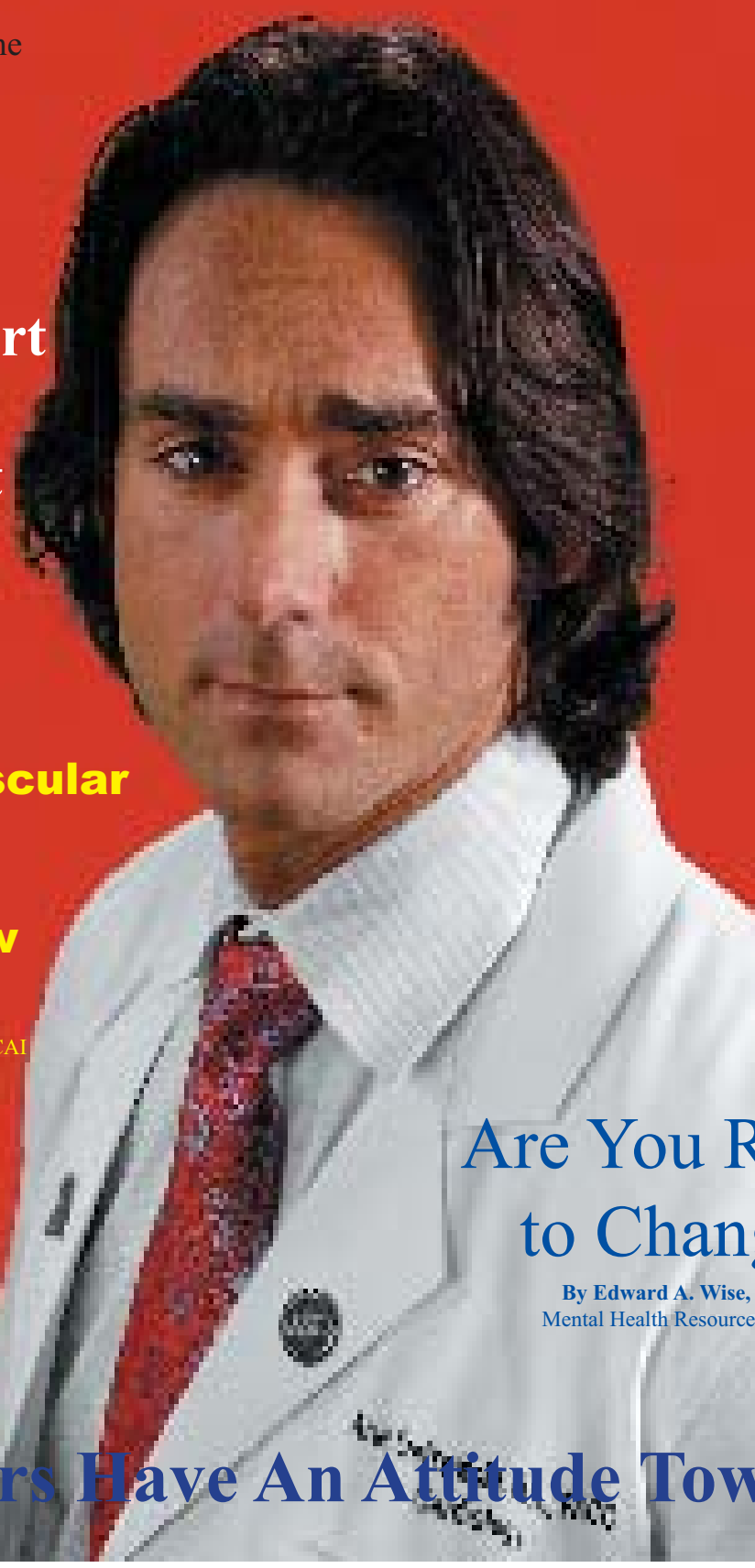

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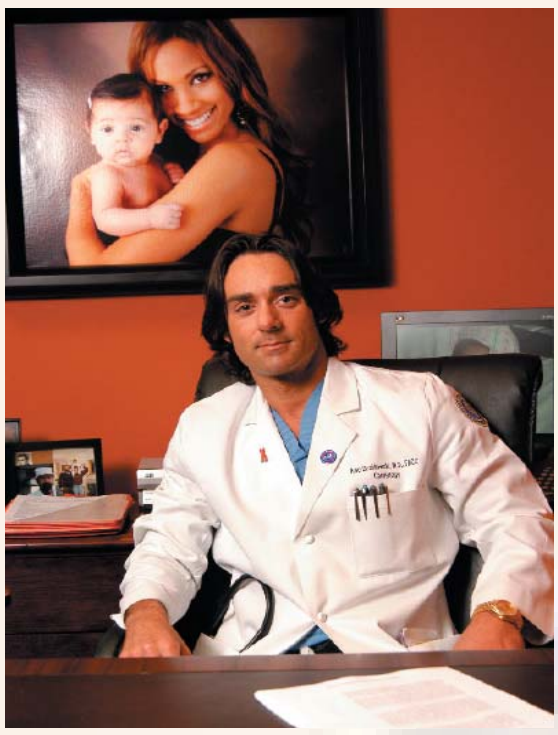

Standard of Care for Heart Failure Management

By Arie Szatkowski, M.D., FACC
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Heart failure is a clinical syndrome characterized by inadequate systemic perfusion to meet the body's metabolic demands as a result of impaired cardiac pump function. This may be further subdivided into either systolic or diastolic heart failure. In systolic heart failure, there is reduced cardiac contractility, whereas in diastolic heart failure there is impaired cardiac relaxation and abnormal ventricular filling. There are an equal number of systolic heart failure cases and diastolic cases. There can also be a combination of both. The body responds by activating multiple systemic neorohormonal pathways that compensate initially by redistributing blood flow to vital organs, but later exacerbate the patient's symptoms and lead to clinical deterioration.

Over the last decade and a half a wide array of evidence based therapies for left ventricular dysfunction has favorably impacted on the risk of death due to heart failure. Patients with heart failure on optimal medical management are able to anticipate a one year survival rate of greater than 90%, a rate that is substantially better than the expectations of survival just several years ago. However, for the impact on longevity to be truly meaningful, there must be associated improvements in morbidity, especially for patients with advanced heart failure. This article will focus on the most recent evidence based recommendations set forth by the American College of Cardiology (ACC) and American Heart Association (AHA) that if applied correctly can make a tremendous impact on mortality, morbidity and hospitalizations.

Prevalence

Heart failure is a common syndrome accounting for one million hospital admissions annually, and another 2 million hospitalizations occur with heart failure as a secondary diagnosis. One-third of these patients is re-admitted within 90 days for recurrent decompensation. There is a 20% overall annual mortality and those who are most likely to die exhibit ventricular arrhythmia's, higher New York Heart Association Heart (NYHA) Failure Class, lower left ventricular ejection fraction, high catecholamine and B-type natriuretic peptide levels, low serum sodium, hypocholesterolemia, and marked left ventricular dilatation. The cost of caring for patients with heart failure exceeded 30 billion dollars in 2005.

Etiology

It is always important to make attempts to determine the cause of heart failure. In left ventricular dysfunction (ejection fraction < 40%) the most common cause is coronary artery disease and so prevalent that perhaps every one with systolic heart failure should undergo coronary arteriography. Other common causes include idiopathic dilated cardiomyopathy, viral induced cardiomyopathy, valvular heart disease, hypertensive heart disease, toxin induced cardiomyopathies (adriamycin, alcohol, cocaine, etc.), and congenital heart disease.

Right ventricular dysfunction most commonly is a consequence of left ventricular systolic dysfunction. Other causes of right failure are right ventricular infarction, pulmonary hypertension, chronic lung disease, severe tricuspid regurgitation, and arrhythmogenic right ventricular dysplasia.

Diastolic left ventricular dysfunction usually is related to chronic hypertension or ischemic heart disease. If there is no evidence of ischemic coronary disease or hypertension present then an investigation for restrictive, infiltrative and hypertrophic cardiomyopathies must be entertained. Another type of diastolic dysfunction includes the hemodynamic impairment that occurs from either pericardial constriction or cardiac tamponade.

Thyrotoxicosis, arteriovenous fistulae, Paget's disease, severe chronic anemia and pregnancy can cause high output failure.

Pathophysiology (see Figure 4)

In left ventricular dysfunction, regardless of the cause, cardiac output is compromised and pulmonary pressures are high, resulting in pulmonary edema. In response to this physiologic state the body activates several neurohormonal pathways in order to increase circulating blood volume. The activation of these systems eventually has a deleterious effect. These circulating catecholamines cause arteriolar vasoconstriction, which results in activation of the renin-angiotensin aldosterone pathway from the kidneys. In addition endothelin an endogenous vasoconstrictor and growth factor is released into the blood as are pro-inflammatory cytokines and vasopressin. All of this leads to sodium and water retention, endothelial dysfunction, organ fibrosis, myocardial apoptosis, and cardiac remodeling. The antagonism of these pathways is the basis for the modern treatment for heart failure.

The primary abnormality in diastolic dysfunction is impaired relaxation of the left ventricular chamber resulting in high diastolic pressures and impaired filling of the ventricles. Neurohormonal activation also occurs in this form of heart failure. At this point in time our understanding of diastolic heart failure is limited but intensive investigation is underway

The new guidelines set forth by the ACC and AHA provide a new approach to classifying heart failure by dividing the syndrome into stages of disease progression. Stage A includes patients at high risk of developing heart failure such as those with hypertension, diabetes, coronary artery disease, familial history. Stage B represents patients who have cardiac abnormalities but are asymptomatic. Stage C patients with heart failure have structural heart disease with symptoms and stage D are those with refractory end stage heart failure.

Signs and Symptoms (See Table 2)

The clinical presentation of heart failure is vast. The most common symptoms are shortness of breath, inability to lay flat without getting short of breath (orthopnea), waking up with trouble breathing (paroxysmal nocturnal dyspnea), fatigue,

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effort intolerance, cachexia, edema (swelling), jugular venous distension, ascites, hepatomegaly and renal hypoperfusion resulting in renal dysfunction.

One of the schematics that is utilized by physicians who treat patients with heart failure is the NYHA classification system that categorizes heart failure patients by levels of impairment. This classification provides further guidance on treatment strategies. The AHA/ACC guidelines advocates that physicians recommend treatments for heart failure dependent on each particular patients level of impairment.

There are 4 classes of impairment. Class I: no symptoms with normal activity; Class II: ordinary physical activity somewhat limited by symptoms (i.e., long distance walking, climbing 2 flights of stairs); Class III: limited by dyspnea at mild work loads (short distance walking, one flight of stairs); Class IV: dyspnea at rest or with very little exertion.

Diagnosis

There is no single diagnostic test to diagnose heart failure because it is largely a clinical diagnosis that is based on careful history and physical examination. Any patient presenting with signs and symptoms of heart failure should have an electrocardiogram (ECG), chest radiograph, and B-type natriuretic peptide assay (BNP). The most useful diagnostic test is the echocardiogam. The ECG can show sinus tachycardia, atrial fibrillation, other tachyarrythmias, bundle branch block, evidence of myocardial infarction, hypertrophy. Chest x ray might show cardiomegaly, edema, effusions, or pulmonary vascular congestion.

The echocardiogram can distinguish between systolic and diastolic heart failure and can provide important evidence regarding valvular abnormalities, restrictive and infiltrative myopathies, ischemic versus nonischemic etiology, pericardial disease, and wall thickness.

Cardiac catheterization is important for ruling out an ischemic cause of heart failure. This is especially important because it is one of the few causes that is potentially reversible if treated before completed damage has occurred. Some patients may require multi gated acquisition scanning (MUGA) a nuclear based ventriculogram that helps establish the actual ejection fraction. This tool is helpful because the ejection fraction is important to know when deciding on certain therapies especially implantable defibrillators and biventricular leads. Metabolic cardio-pulmonary exercise testing helps distinguish someone's symptoms of dyspnea as either from a pulmonary or cardiac etiology and helps determine the severity of cardiac impairment which provides a guide for heart failure specialists to prepare their patients for cardiac transplantation. BNP assays are useful for determining prognosis because severity of disease correlates with level of BNP circulating in the blood. It is also very useful to help differentiate between patients who have symptoms of heart failure but have primary pulmonary disease.

Therapies **(See Figure 1)**

A discussion of recommended therapies will be provided here and presented in a fashion that will hopefully help guide appropriate clinical choices. Remember however that these are only guidelines and that each patient is unique and requires an approach unique to that patient. Also remember that if you are dealing with a patient at risk of cardiac disease or who has heart failure a referral should be made to a cardiologist specializing in heart failure. There is definitive data that shows better mortality and morbidity outcomes in patients when they are cared for by a heart failure specialist.

Non-Pharmacological

Dietary sodium and fluid restrictions should be implemented in all patients with congestive heart failure. Limit patients to 2 grams of sodium and 2 liters of fluid daily. You may have to alter the limits based on severity of congestion.

Cardiac rehabilitation and exercise programs are important for improving symptoms, increasing exercise tolerance, weight loss and preventing muscle atrophy. All patients must be counseled

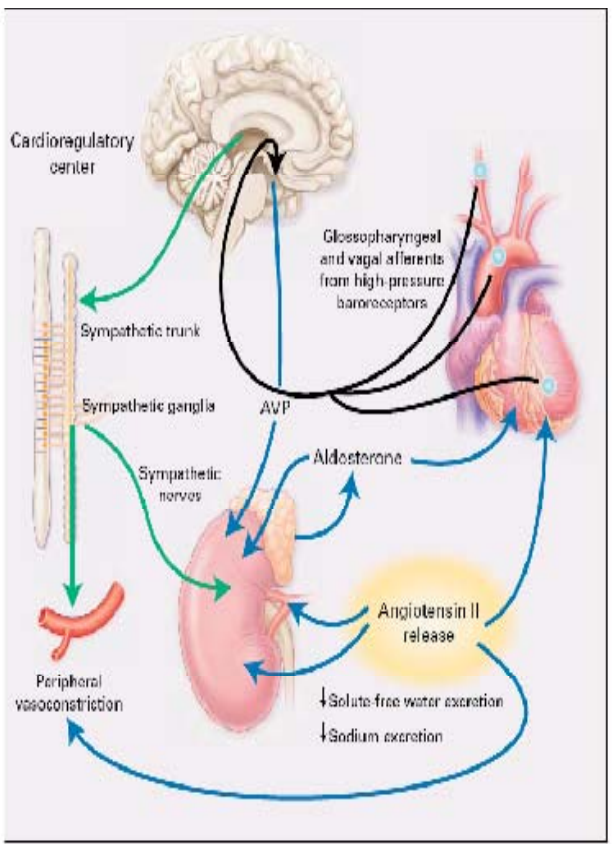

Figure 4. The Pathophysiology of Heart Failure.

Unloading of high-pressure baroceptors (blue circles) in the left ventricle, carotid sinus, and aortic arch generate afferent signals (black) that stimulate cardioregulatory centers in the brain, resulting in the activation of efferer pathways in the sympathetic nervous system (green). The sympathetic nervous system appears to be the primar integrator of the neurohumoral vasoconstrictor response to arterial underfilling. Activation of renal sympathet nerves stimulates the release of renin and angiotensin II, thus activating the renin–angiotensin–aldosterone systen Concomitantly, sympathetic stimulation of the supraoptic and paraventricular nuclei in the hypothalamus results i the nonosmotic release of arginine vasopressin (AVP). Sympathetic activation also causes peripheral and renal vas oconstriction, as does angiotensin II. Angiotensin II constricts blood vessels and stimulates the release of aldoste rone from the adrenal gland, and it also increases tubular sodium reabsorption and causes remodeling of cardia myocytes. Aldosterone may also have direct cardiac effects, in addition to increasing the reabsorption of sodium an the secretion of potassium and hydrogen ions in the collecting duct. The blue lines designate circulating hormone

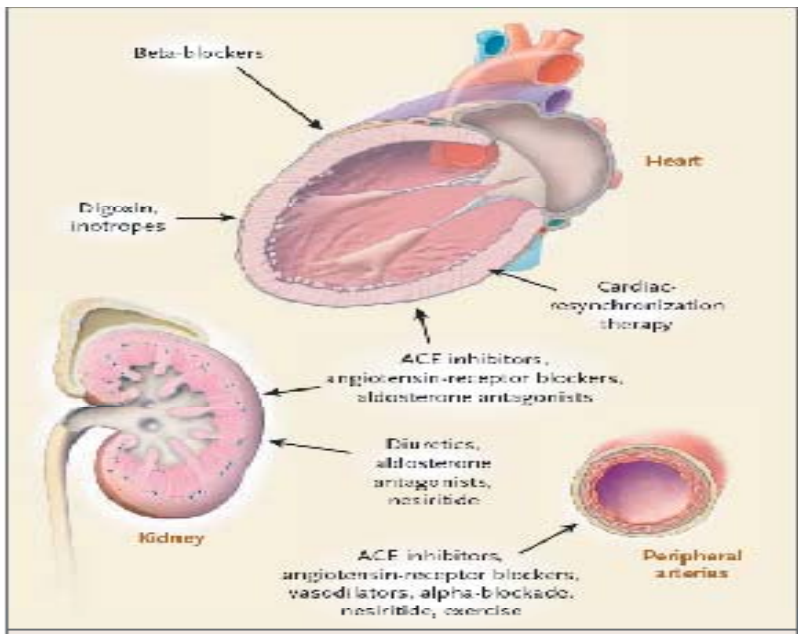

Figure 1. Primary Targets of Treatment in Heart Failure.

Treatment options for patients with heart failure affect the pathophysiological mechanisms that are stimulated in heart failure. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin-receptor blockers decrease afterload by interfering with the renin–angiotensin–aldosterone system, resulting in peripheral vasodilatation. They also affect left ventricular hypertrophy, remodeling, and renal blood flow. Aldosterone production by the adrenal glands is increased in heart failure. It stimulates renal sodium retention and potassium excretion and promotes ventricular and vascular hypertrophy. Aldosterone antagonists counteract the many effects of aldosterone. Diuretics decrease preload by stimulating natriuresis in the kidneys. Digoxin affects the Na^+/K^+–ATPase pump in the myocardial cell, increasing contractility. Inotropes such as dobutamine and milrinone increase myocardial contractility. Beta-blockers inhibit the sympathetic nervous system and adrenergic receptors. They slow the heart rate, decrease blood pressure, and have a direct beneficial effect on the myocardium, enhancing reverse remodeling. Selected agents that also block the alpha-adrenergic receptors can cause vasodilatation. Vasodilator therapy such as combination therapy with hydralazine and isosorbide dinitrate decreases afterload by counteracting peripheral vasoconstriction. Cardiac resynchronization therapy with biventricular pacing improves left ventricular function and favors reverse remodeling. Nesiritide (brain natriuretic peptide) decreases preload by stimulating diuresis and decreases afterload by vasodilatation. Exercise improves peripheral blood flow by eventually counteracting peripheral vasoconstriction. It also improves skeletal-muscle physiology.

for smoking cessation and provided with ways to help stop the addiction. Obese patients must be encouraged to lose weight.

Immunization with influenza and pneumococcal vaccines should be considered in all patients with heart failure and coronary atherosclerosis.

(See Figure 3)

1. **Stage A: patients at risk for developing heart failure:**
Systolic and diastolic parameters should be controlled in accordance with current Joint National Committee IV guidelines. Lipid disorders should be managed as recommended by current Adult Treatment Program III guidelines. Also, for patients with diabetes, blood sugar should be treated to optimal goals. Counseling to those patients who practice high-risk behaviors such as smoking, drinking alcohol, doing drugs should be provided. Ventricular rate should be controlled or sinus rhythm restored in patients with supraventricular tahycardias. Thyroid disorders should be controlled in accordance with accepted contemporary guidelines. Healthcare providers should routinely evaluate patients at risk of developing heart failure for signs and symptoms of heart failure. For those patients with documented coronary artery disease the healthcare practitioner should provide appropriate secondary preventive measures. And, for those with a family history of cardiomyopathy or a history of exposure to cardiotoxic drugs, routine noninvasive testing should be provided.

The only class of drugs that is somewhat recommended and considered reasonable first line therapy by the ACC/AHA for Stage A patients are Angiotensin Converting Enzyme Inhibitors (ACEI) and Angiotensin II Receptor Blockers (ARBS) in patients with a history of atherosclerotic heart disease, diabetes mellitus, and hypertension.

Nutritional supplements should not be recommended for patients to prevent structural heart disease

2. **Stage B: patients with cardiac structural abnormalities or remodeling who have not yet exhibited signs of heart failure:**
All recommendations for Stage A patients should be applied to patients in stage B. Beta blockers and ACEI should be used in all patients with a recent or remote history of myocardial infarction (MI) regardless of ejection fraction (EF) or presence of heart failure. Beta blockers and ACEI are indicated in all patients without a history of MI who have reduced ejection fraction. ARBS should be used in all post-MI patients without heart failure who are intolerant to ACEI and have reduced EF. Patients without heart failure who have suffered a MI should be treated according to contemporary guidelines. Coronary revascularization, and valve replacement or repair should be recommended in appropriate patients without heart failure according to contemporary guidelines.

It is reasonable to place patients on ACEI or ARBS who have hypertension and LVH. It is reasonable to use ARBS in patients with low EF and no symptoms of heart failure who can not tolerate ACEI. Placement of an ICD (Implantable Cardiac Defibrillator) is reasonable in patients with ischemic cardiomyopathy who are at least 40 days post-MI, have an EF of 30% or less, are NYHA functional class I on chronic optimal medical therapy, and have reasonable expectation of survival greater than 1 year.

For those patients with non-ischemic cardiomyopathy with EF less than or equal to 30% who are NYHA functional class I on chronic optimal medical therapy the placement of an ICD may be considered but the benefit is not as great as in those with ischemic cardiomyopathy.

Those therapies that are not recommended are use of digoxin in patients with low EF who do not have symptoms or are not in atrial fibrillation. There has been no benefit in patients with low EF, sinus rhythm and no heart failure. Use of nutritional supplements is not recommended. Also, do not use calcium channel blockers with negative inotropic effects (diltiazem, verapamil) in patients post MI without heart failure and low EF as these can have harmful side effects.

3. **Stage C: patients with structural heart disease and symptoms of heart failure:**
Patients with reduced EF Measures recommended in Stage A and B patients are appropriate to consider in patients in Stage C.

Diuretics and salt restriction are indicated in those who have signs of fluid retention. ACEI are recommended for all patients with current or prior symptoms of heart failure. Beta Blockers (using one of the three proven to reduce mortality, i.e. Bisoprolol, carvedilol and long acting metoprolol succinate should be used for all stable patients with current or past symptoms of heart failure. Note the importance of using the beta blockers

Table 3. Common Clinical Problems in Patients with Heart Failure and Recommended Solutions.*

Clinical Problem	Recommended Solutions
The patient has classic symptoms of heart failure with a normal left ventricular ejection fraction.	Consider diastolic heart failure, valvular heart disease, hypertensive heart disease, and ischemia.
The patient has hypotension: what is the systolic blood pressure too low?	Asymptomatic patients with dilated cardiomyopathy often tolerate a systolic blood pressure of 80 mm Hg. If the patient has no lightheadedness or undue fatigue, peripheral perfusion is adequate, and blood urea nitrogen and creatinine are unchanged, continue the same doses of medications. In symptomatic patients, decrease the dose of diuretic. If symptoms persist, adjustment of the timing of concomitant medications may be helpful. Decreasing the dose of the ACE inhibitor, beta blocker, ARB, or vasodilator is indicated.
The patient has hyperkalemia.	Ensure that the patient is taking no excessive potassium supplement or potassium-containing salt substitute. Avoid hypovolemia. Consider decreasing the dose of a potassium-sparing diuretic. Concomitant use of an ACE inhibitor or ARB and spironolactone may increase the risk of hyperkalemia. Avoid high doses of ACE inhibitors and ARBs in patients receiving spironolactone. Avoid use of spironolactone in patients with renal failure, and use low doses of ACE inhibitors and ARBs.
The patient has increasing azotemia while taking ACE inhibitors.	Decrease the dose of diuretic. Consider renal-artery stenosis if azotemia persists.
The patient has a cough while taking ACE inhibitors.	Rule out worsening congestive heart failure. Change to ARB if severe cough persists.
Should the dose of the ACE inhibitor be increased or should beta-blocker therapy be initiated in a symptomatic patient?	Start beta-blocker therapy if there are no contraindications.
Should an ARB be added to ACE-inhibitor therapy or should a beta-blocker be added in a symptomatic patient?	Start beta-blocker therapy if there are no contraindications.
The patient has worsening symptoms of congestive heart failure after starting beta-blocker therapy.	Increase the dose of diuretic and slow the titration of the beta-blocker.
The patient has worsening bronchospasm after starting beta-blocker therapy.	Decrease the dose of the beta-blocker. Consider a beta-selective agent. Discontinue treatment with the drug if the problem persists.
Persistent paroxysmal nocturnal dyspnea or orthopnea or daytime fatigue despite absence of fluid retention on physical examination.	Evaluate the patient for central or obstructive sleep apnea.
The patient requires repeated hospitalizations.	A multidisciplinary approach should be initiated, with a visiting nurse in the home. Referral for heart failure is indicated.

* ACE denotes angiotensin-converting enzyme, and ARB angiotensin-receptor blocker.

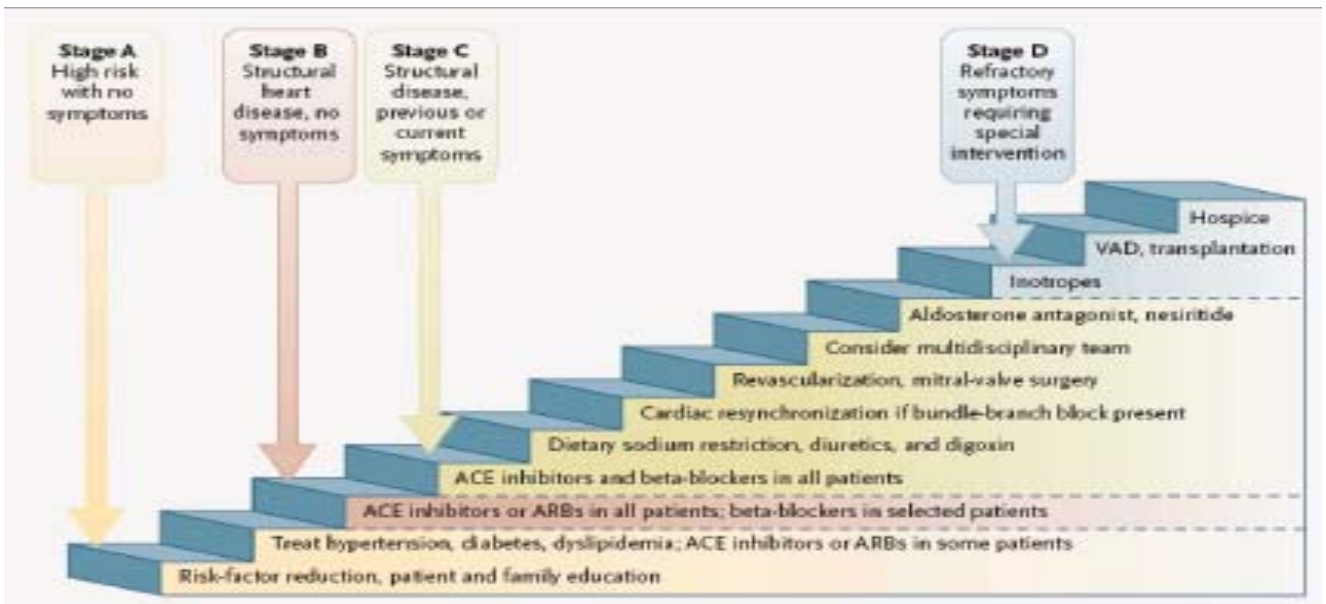

Figure 3. Stages of Heart Failure and Treatment Options for Systolic Heart Failure.

that have been proven to have benefit. ARBS approved for the use of heart failure are recommended in patients intolerant to ACEI (candesartan and valsartan only). Drugs known to adversely effect the clinical status of patients with reduced EF and symptoms should be avoided such as NSAIDS, certain antiarrhythmic drugs, and most calcium channel blocking drugs. Maximal exercise testing is recommended to help determine an appropriate exercise program. Exercise training is appropriate, beneficial and should be encouraged. ICDs are recommended to prolong survival in patients who have a history of cardiac arrest, ventricular fibrillation, or hemodynamically significant ventricular tachycardia. ICDs are recommended for patients for primary prevention of sudden death in those who have ischemic heart disease who are 40 days post-MI, have an EF less than or equal to 30%, with NYHA class II or III symptoms on optimal chronic medical therapy who have at least a one year life expectancy. Patients with nonischemic cardiomyopathy with EF less than or equal to 30% on maximum medical therapy with NYHA class II or III symptoms should get an ICD for sudden death prevention. Cardiac resynchronization therapy or Bi-Ventricular lead placement should be offered to patients with EF less than or equal to 35% who are class III or IV NYHA status despite optimal medical therapy and who have cardiac dysynchrony. Aldosterone blockers should be used in patients with moderate to severe symptoms who have acceptable renal function (creatinine < 2.5 in men, < 2.0 in women) and acceptable potassium levels (< 5.0) and in whom these levels can be monitored routinely.

It is reasonable to use ARBS as first line therapy for mild to moderate heart failure in patients already on ARBS. It is reasonable to use digoxin because it has been proven to

Table 2. Characteristics of Patients with Diastolic Heart Failure and Patients with Systolic Heart Failure.*

Characteristic	Diastolic Heart Failure	Systolic Heart Failure
Age	Frequently elderly	All ages, typically 50–70 yr
Sex	Frequently female	More often male
Left ventricular ejection fraction	Preserved or normal, approximately 40% or higher	Depressed, approximately 40% or lower
Left ventricular cavity size	Usually normal, often with concentric left ventricular hypertrophy	Usually dilated
Left ventricular hypertrophy on electrocardiography	Usually present	Sometimes present
Chest radiography	Congestion with or without cardiomegaly	Congestion and cardiomegaly
Gallop rhythm present	Fourth heart sound	Third heart sound
Coexisting conditions		
Hypertension	++–	–+
Diabetes mellitus	++	+/–
Previous myocardial infarction	+	–++
Obesity	++–	+
Chronic lung disease	+/–	0
Sleep apnea	+–	–+
Long-term dialysis	+–	0
Atrial fibrillation	+ (usually paroxysmal)	+ (usually persistent)

* A single plus sign denotes "occasionally associated with," two plus signs "often associated with," three plus signs "usually associated with," and a zero "not associated with."

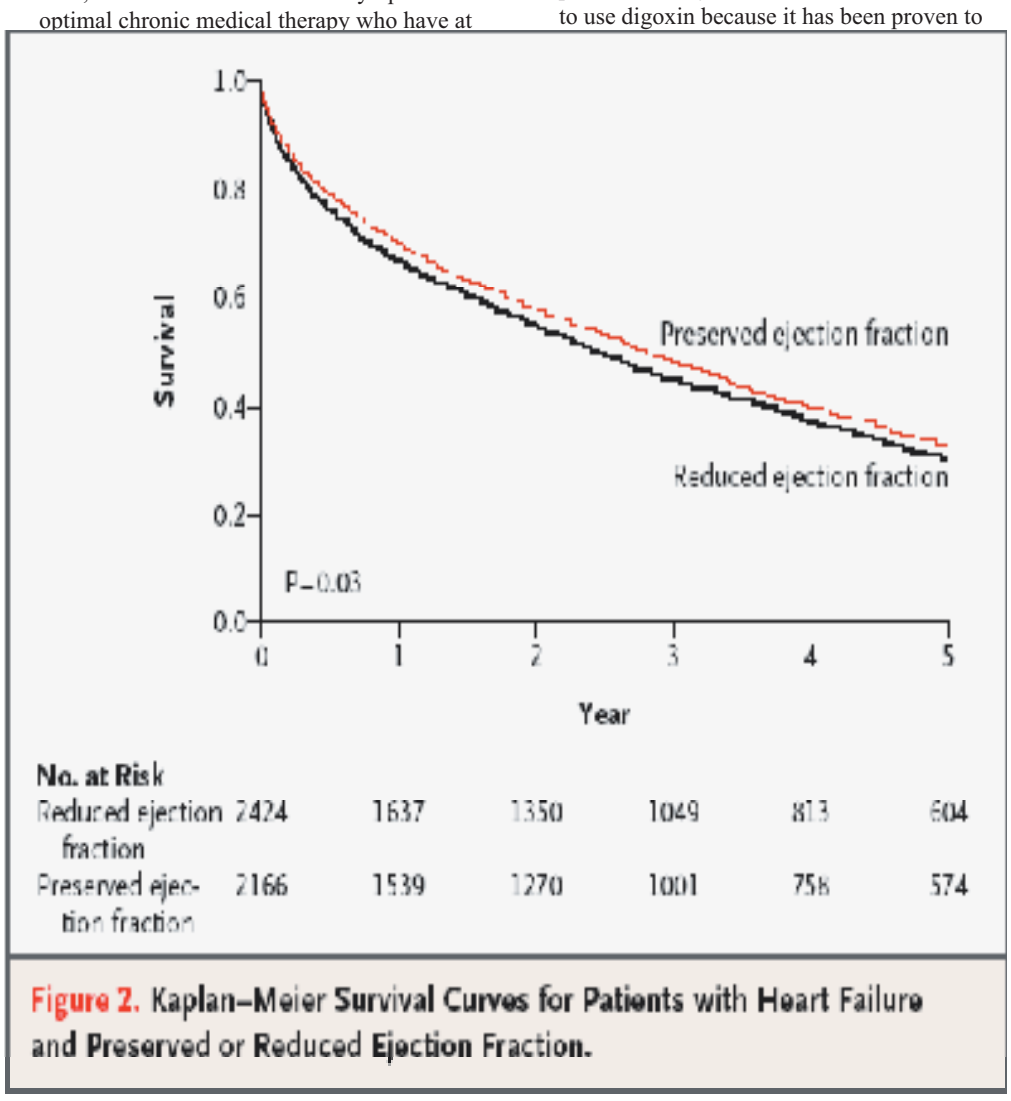

Figure 2. Kaplan–Meier Survival Curves for Patients with Heart Failure and Preserved or Reduced Ejection Fraction.

reduce hospitalizations. The addition of hydralazine and nitrate combination is reasonable in patients who are already on optimal ACEI and beta blocker therapy. Placement of an ICD in any patient with EF between 30% and 35% is reasonable who are class II or III and on maximal medical therapy.

Hydralazine/nitrate combination may be considered in patients who can not be given ACEI or ARBS. The addition of an ARB may be considered in persistently symptomatic patients on conventional therapy.

Routine use of ACEI, ARBS and aldosterone antagonist combination is not recommended for patients with symptomatic heart failure. Calcium channel blockers are not indicated as routine treatment for heart failure in Stage C patients. Long–term use of inotrope infusion is not recommended except as palliation in patients who can not be stabilized with standard medical treatment. Use of nutritional supplements and hormonal replacement is not recommended.

4. **Stage D : Refractory Heart Failure**

   In refractory heart failure meticulous identification and control of fluid retention is recommended, patients should be referred for cardiac transplantation, patients should be referred to heart failure specialty programs, and a discussion about end of life issues should take place. Those who have an ICD may want to talk about deactivating the device. Consideration should be made for potential Left Ventricular Assist Device placement. Although on may use inotropes for palliation of symptoms, routine intermittent infusions are not recommended.

5. **Normal Systolic Heart Failure (patients with symptoms who have a normal EF)**

   Because there is a lack of patients in this subgroup in clinical trials treatment is largely based on the control of physiologic factors that are known to exert negative effects on ventricular relaxation. Therefore, physicians should control systolic and diastolic hypertension, control ventricular rate in patients with atrial fibrillation, use diuretics to control pulmonary congestion and edema, offer revascularization in those who are symptomatic or have demonstrable ischemia. One may consider restoring sinus rhythm in patients with atrial fibrillation.

For Common Clinical Prblems and Recommended Solutions See Table 3.

Conclusion

There is still a lot to be discovered about the molecular pathways involved in heart failure; there are many areas of heart failure that remain poorly understood. A multitude of new trials and therapies are actively being investigated. However, a great number of people with heart failure remain inadequately treated. Although we already have highly beneficial and effective therapies in our armamentarium they are poorly utilized. We must first master what we know if we are to make an impact on an ever growing problem. The article presented here is a rough outline and may be a starting point for clinicians that are dedicated to helping their patients. Another important feature of effective treatment is educating your patients. Their involvement in the care of CHF is as important in reducing negative outcomes as your role is in recommending appropriate evidence based therapies. For a complete review of this topic please take the time to read the ACC/AHA Guidelines for Diagnosis and Management of Chronic Heart Failure in the Adult. *Note: All Tables and Figures are from The New England Journal of Medicine.*

About The Author

Arie Szatkowski, M.D., FACC is Board Certified in Internal Medicine and Cardiology. He received his M.D. from Cornell University Medical College in New York. Dr. Szatkowski completed his Cardiology Fellowship, as well as, internship and residency in Internal Medicine at New York Presbyterian Hospital, Columbia University in New York. In 2000, he was named “Physician of the Year”. Also, while in his residency, Dr. Szatkowski earned the prestigious Arnold P. Gold Award for Excellence in Humanism and Teaching and was appointed Chief Fellow in Cardiology. Dr. Szatkowski joined the Stern Cardiovascular Center in July, 2003. Dr. Szatkowski’s interests include: Clinical Cardiology, Congestive Heart Failure, Valvular Disorders, Adult Congenital Disease, Coronary Artery Disease and Preventive Cardiology. He also offers Nuclear Cardiology and Clinical Echocardiography including Transesophageal Echocardiography. Dr. Szatkowski is on the staff of Baptist Memorial, Saint Francis and Methodist hospitals. 901-271-1000