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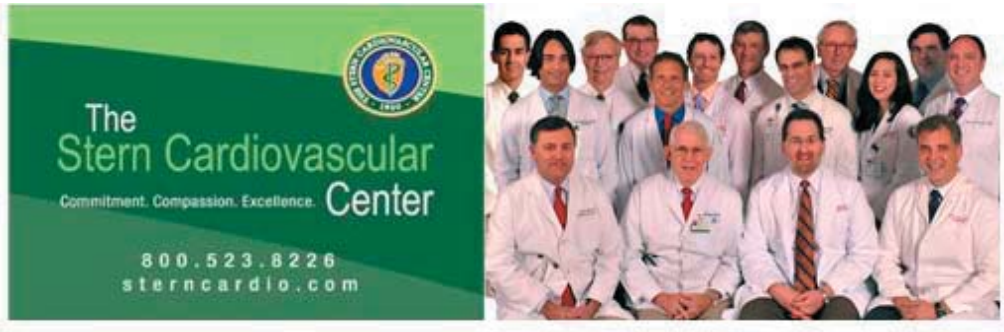

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Congestive Heart Failure: An Introduction

By Arie Szatkowski, M.D., FACC

Stern Cardiovascular Center

Heart failure is the end stage of all diseases of the heart. Congestive heart failure is a major causes of morbidity and mortality in the general population. The total cost of heart failure exceeds 38 billion dollars annually and is expected to rise. The financial costs pale in comparison to the toll this disease takes on affected individuals. Heart failure patients are greatly impacted by the disease, having to deal with limitations on their breathing, mobility and their general quality of life.

The increasing age of the population, success in the treatment of patients with heart attacks, the increasing prevalence of diabetes mellitus, and obesity are contributing to the increased prevalence and thus economic expenditures of chronic heart failure. The direct costs are mainly attributed to hospitalizations for acute heart failure.

Evidence based treatment of congestive heart failure is both effective in lowering morbidity and mortality as well as cost effective from an economic point of view. Both the underutilization of these known effective treatments and ineffective education of patients with congestive heart failure are obstacles to improving outcomes and costs.

Until a cure is found, the solution to heart failure lies in the center that specializes in heart failure. A center where a team collaborates to provide evidence based and up to date treatment and brings together the latest technology and the most promising clinical research projects is the one that will improve quality of life, reduce hospitalizations and prolong life for patients with heart failure. The Stern Cardiovascular Center is such a place.

In recognition of heart failure month, the doctors at Stern have volunteered to collaborate on a series of articles dealing with several important topics related to congestive heart failure. These articles will cover a spectrum of topics related to heart failure including definitions and terminology, optimal medical therapy, revascularization with stenting versus bypass, treatment of arrhythmias, device therapy, avoiding sudden death, conditions affecting women, mechanical circulatory devices and transplantation, and new frontiers and research. A few will be published in this journal and the rest in subsequent issues of The Wellness Guide.

We hope that the reader learns some valuable information that may impact his or her life or the life of a loved one. And we hope to see you at The Heart Failure Fair on February 11 at The Stern Cardiovascular Center. 901-271-1000.

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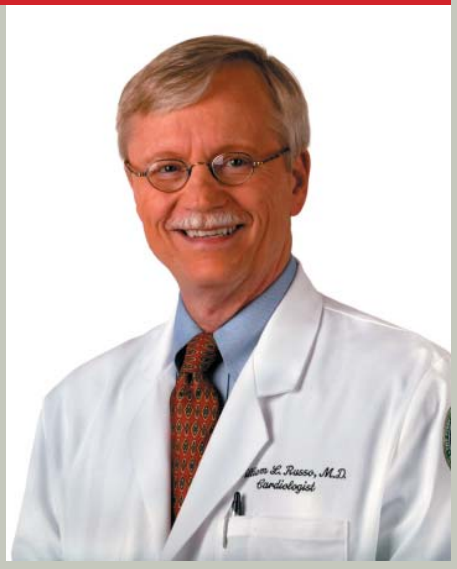
About The Author: Arie Szatkowski, M.D., FACC is Board Certified in Internal Medicine and Cardiology. He received his M.D. from Cornell University Medical College in New York. Dr. Szatkowski completed his Cardiology Fellowship, as well as, internship and residency in Internal Medicine at New York Presbyterian 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



What is Heart Failure?

By William Russo, M.D., FACC

Stern Cardiovascular Center

The following article will define heart failure, which is often called congestive heart failure (CHF). The mechanisms of how the heart can fail, the causes of CHF, and its symptoms will be outlined.

The term, heart failure, is a serious cardiac disorder. Some have the idea it is a sudden fatal catastrophic event, which comes on without warning or could be due to the effect of some horrible life experience. We have all heard people say something like, "I felt like I was going to die of heart failure right then and there."

However, most of us know it is nothing like that described above. What it amounts to is the inability of the heart to pump enough blood to supply the needs of the body's various organs.

To understand how the heart works and fails, let us think of our heart as a water pump that supplies water to irrigate a garden (our body). In order for the vegetables (our many organs) to survive, this pump has to provide, let us say, one gallon per minute. As long as the pump functions at the level to supply this amount of water, the vegetables (the body's organs) will thrive. The water that is going into the pump is coming from a reservoir that is fed by a waterfall (blood coming back from the body). As long as the pump functions, this reservoir will not overflow and soak the floor (with fluid getting into and flooding the airspaces in the lungs).

Now let us allow the pump to malfunction and puts out half the volume it was previously pumping. Two things happen: 1) The vegetables (organs) don't get enough water and begin to wither (malfunction). 2) The intake pool fed by that waterfall (blood coming in from the body) begins to overflow and the floor gets wet (fluid gets into the lung's air spaces causing shortness of breath and even back up enough to cause swelling or edema of the abdomen and lower extremities).

So with normal heart function with a normal cardiac output, the blood going into the heart does not back up and congest the lungs, abdomen, and legs, and the body's organs get sufficient blood to normally function.

There are several disorders that can reduce the cardiac output. One is systolic dysfunction or a weakness of the heart muscle the produces a drop in cardiac output (the amount of blood pumped from the main pumping chamber – left ventricle – per minute. This can result from damage to the muscles of the left ventricle. One or more segments of the wall of this chamber either become weaker or quits contracting altogether. You can think of this as an eight-cylinder gasoline engine that is operating on fewer that the full eight cylinders. In the case of the engine, one or more spark plugs are not firing.

The amount of blood ejected from the heart's main pumping chamber each beat is called the ejection fraction, which is normally 55% or more. When the left ventricle relaxes and fills with blood, it then contracts. When it ejects 55% of the blood, that is an ejection fraction of 55%. In those diseased hearts with weak walls of the left ventricle, the ejection fraction can be as low as 15-20%, but can be anywhere from this low range to just under normal. This form of congestive heart failure is termed systolic congestive heart failure. (Systole or the systolic part of the heart cycle is that involved in ejection of blood from the heart.)

The other form is diastolic congestive heart failure. (Diastole or the diastolic part of the heart cycle is that involved with relaxation and filling of the heart chambers with blood.) This is that heart failure that is due to abnormal relaxation of the main pumping chamber of the heart. Normally, the left ventricle relaxes allowing the chamber to fully fill with blood. In diastolic dysfunction, the muscle of the left ventricle is stiff, relaxes more slowly, and does not completely dilate.

Both systolic and diastolic heart failure create a backup of blood coming into the left ventricle, which, in turn, causes the pressure in the minor chamber feeding the left ventricle (left atrium) and vessels bringing blood from the lungs into the left atrium to elevate. This higher pressure causes fluid to leak into the tiny air spaces in the lungs (alveoli) thus taking up space where the oxygen comes into the lungs. This reduces the amount of oxygen getting into the blood. As the oxygen level drops, sensors in the brain detect this and cause the chest to breathe faster. As this problem progresses, fluid begins to accumulate in the abdomen and in the lower extremities.

There is a list of causes of CHF. Diseases that cause the left ventricle to be weak include the following:

1. Heart damage from heart attacks (myocardial infarctions due to blocked heart arteries).

2. Heart damage from viral infections.

3. Heart damage from long standing alcohol abuse.

4. Heart damage rarely due to pregnancy.

5. The prolonged effect of high blood pressure (hypertension) on the left ventricle.

6. Leaking or blocked heart valves.

7. Certain heart rhythm problems such as sustained atrial fibrillation (an irregular and usually fast heart rhythm) can cause the left ventricle to dilate and fail to pump properly.

8. There is a myriad of symptoms of congestive heart failure, which includes the following:

A. Shortness of breath, swelling of the feet, legs, and even the abdomen. These may be only minor and make one think that their weight or the lack of exercise is the cause. The trouble breathing can worse as more fluid collects in the lungs and tissues. The shortness of breath usually occurs with increased activity. The worse the condition, the greater the amount of trouble breathing with lower levels of activity, such as just walking across the floor. Shortness of breath upon lying flat, making the patient sit to breathe more easily and shortness of breath that wakes the patient from sleep causing the patient to sit for relief usually indicates more severe congestive heart failure. The swelling or edema of the feet, ankles, and legs can be mild to extreme. It can be so bad as to cause the legs to weep with a clear fluid that seeps through small breaks in the skin. In addition, this excess amount of fluid adds weight to the body thus making more difficult to get around. Each gallon of fluid weighs about seven pounds. This accumulation of fluid in the lungs often makes the patient sleep in the sitting position either on a chair or with many pillows supporting the body in the sitting position. The fluid accumulation in the abdomen causes nausea, abdominal pain, and a decrease or loss of appetite.

B. This increase in fluid in the lungs can give the patient the feeling of chest discomfort described as a fullness or tightness in the chest, which is similar to the pain of angina (chest pain due to blocked heart arteries.)

C. Weakness – this is due to a combination of the low cardiac output, the extra weight, and low blood oxygen.

D. Fatigue – this is an early symptom of congestive heart failure. Often, because many other conditions cause fatigue, it may not be obvious at first that this symptom is due to CHF.

E. Some patients notice an increase in urine output at night thus making it more difficult to rest.

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About The Author

William F. Russo, M.D., FACC is Certified in both Internal Medicine and Cardiovascular Disease. His special interests are Consultative Cardiology and Echocardiography. Dr. Russo is the Director of the Echo Lab. His particular interests are patients with mitral valve prolapse and those with chest pains unrelated to heart disease. He can be contacted at the Stern Cardiovascular Center, 901-271-1000.

Heart Transplant Update

By Todd D. Edwards, M.D., FACC, FACP
Stern Cardiovascular Center

This article is intended to address frequently asked questions concerning heart transplants. This Information is presented in a question and answer format.

Q. How many heart transplants are done per year?

A. Although an estimated 50,000 people are candidates for transplantation, there are approximately 5,000 cardiac transplants each year around the world. In the United States, approximately 2000 transplants are performed yearly from a potential recipient pool of 4000 patients. Therefore, the evaluation process is extensive to utilize the organs available efficiently.

Q. Who decides on who gets a donor heart?

A. To ensure the donor hearts are fairly distributed, an organization known as the United Network for Organ Sharing considers time on the wait list, severity of illness and geographic distance between the donor hospital and the transplant center. Increasing the donor pool for this widely accepted technique would be a significant move forward. Please remember to donate organs, don't bury them.

Q. Which patients meet the criteria for transplantation?

A. Patients that are in the hospital requiring I.V. medications to make the heart produce an adequate amount of blood flow to the rest of the organs. For unhospitalized patients, the usual requirements are repeated hospitalizations for heart failure, the need for a ventricular assist device or artificial heart support circulation, increasing dosages and complexity of medications to treat advanced heart failure and reproducible maximum oxygen consumption value of less than 14 ml/kg/min. This maximum oxygen consumption can be determined in an office setting with a special treadmill test.

Q. What is involved in the pre-transplant evaluation?

A. In addition to meeting the standard requirements above, the patient cannot have fixed pulmonary hypertension, active infection or cancer. Prior to transplant, patients undergo cardiac catheterization to make sure the right ventricle and the donor heart will not fail in immediate postop due to high pressures in the lungs. Patients are screened for chronic infections and cancer. Which are two contraindications to transplant due to immunosuppressant therapy.

Q. Are there any age restrictions to transplant?

A. At our local center here in Memphis, we transplant up to the age of 68. There are programs in the country that approve transplant for patients above that age. However, the complication rate tends to go up and longevity tends to go down with the older age group.

Q. What if I have other significant problems, such as diabetes mellitus or advanced lung disease?

A. These are relative considerations in transplant recipient selection, but diabetics can be transplanted. However, significant complications of retinopathy, neuropathy and kidney damage can occur

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with prolonged diabetes mellitus, making this patient less likely to benefit from transplant. In addition, patients with mild lung disease can be transplanted, but advanced lung disease with significant limitations is also a relative contraindication to transplant.

Q. Are there any other absolute contraindications to transplant?

A. Most transplant centers will not transplant anyone with renal failure unless it is combined with a kidney transplant. In addition, cirrhosis or significant psychiatric disease is also a contraindication.

Q. What nonmedical issues are integrated into the transplant work up?

A. The patient will need to be screened for use of alcohol, tobacco and recreational drugs. Most centers adhere to a strict no tobacco policy given the organ shortage. Psychologic and social assessments are critical in determining whether the patient is adequate for compliant follow-up with significant family support to help them through the postoperative period.

Q. What is the survival after first heart transplant?

A. There is approximately an 85% to 90% survival rate after one year with an annual death rate increase of approximately 4% thereafter. A three year survival rate approaches 75%.

Q. What are the risk factors for increased risk of death of the recipient?

A. The recipient risk factors for a less favorable outcome are the preoperative need for a ventilator, a second heart transplant, preoperative ventricular assist device, female sex and being less than 80% of ideal body weight or being more than 140% of ideal body weight.

Q. What can patients expect in terms of longevity post transplant?

A. In general, it is thought that most transplants are expected to live an average of 10 years. Certainly, some patients have not survived this long, but others have survived over 20 years. However, it is noted that being a candidate for heart transplant means your outcome without transplant would be much worse and therefore these additional 10 years are quite precious. Early mortality can be due to acute rejection which most likely can occur in the first three to six months. Incidences of rejection decline significantly after that time. Cardiac rejection is very common within the first year post-transplant and most recipients have one to three episodes of rejection. Later mortality is mainly due to infections, lymphoma and other malignancies, and arterial disease in the transplanted heart vessels. Some of the lymphomas that can develop may be responsive to decreasing immunosuppression. The patient is always at increased risk for cancers and infections due to a weakened immune system due to the transplant drugs. There is also the likelihood of developing coronary artery disease in the heart transplant. The incidence of developing coronary artery disease in the transplanted heart (allograft vasculopathy) is 7% at one year. Long-term incidence of allograft vasculopathy is 32% at five years and 53% at 10 years.

Q. What is rejection?

A. Normally, the body immune system protects the body from infection. This process occurs when the cells of the immune system move throughout the body checking for anything that looks foreign or different from the body's own cells. Rejection occurs when the body's immune cells recognize the transplanted heart as different from the rest of the body and attempts to destroy the foreign object. If left alone, the immune system would damage the cells of the donor heart and eventually destroy it. To prevent rejection, patients receive several drugs called immunosuppressants. These drugs suppress the immune system, so the new heart is not damaged by the body's own defense system. Because rejection can occur at any time, the immunosuppressant drugs are given to the patient the day before the transplant and thereafter for the rest of their lives. To avoid rejection, heart transplant recipients must adhere to the immunosuppressant drug regimen. We are still working on safer, more effective and well-tolerated immunosuppressant medications. However, too much immunosuppressant can lead to serious infections.

Q. What are the signs of rejection?

A. Signs of potential rejection are fever over 100 degrees F, flu like systems, shortness of breath, new chest pain or tenderness and fatigue.

Q. What is expected of a patient post-transplant?

A. Patients need to be seen in the transplant clinic once a month. Throughout early post-transplant there is a fairly vigorous biopsy schedule. During the biopsy, a small sample of the heart muscle tissue is removed to determine if there is any significant rejection present. After the initial postoperative period, biopsies are decreased to every six months with looking at the coronary arteries on a yearly basis in general.

Q. What type of drugs do I need to take post-transplant?

A. In general, immunosuppressant therapy is based on three types of drugs, including Prednisone, Prograf or Cyclosporine, and CellCept.

Q. Is heart transplant covered by insurance?

A. In most cases, the costs of a heart transplant are covered by health insurance, more than 80% of commercial insurers and over 90% of Blue Cross Blue Shield plans offer coverage for heart transplant. Medicaid programs in 33 states and the District of Columbia will pay for transplantation. Medicare will cover heart transplant in Medicare eligible patients if the operation is performed at an approved center.

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About The Author

Todd D. Edwards, M.D., FACC, FACP is a cardiologist (board certified in cardiovascular disease, echocardiography, and nuclear cardiology) at the Stern Cardiovascular Center with a great interest in heart and lung transplantation as well as mechanical devices for heart failure patients. Dr. Edwards sees patients at both locations of The Stern Cardiovascular Center, 8060 Wolf River Blvd., Germantown, Tennessee and 7362 Southcrest Pkwy., Southaven, Mississippi, 901-271-1000.

Medications for the Management of Chronic Congestive Heart Failure (CHF)

By Jason Infeld, MD, FACC

Stern Cardiovascular Center

Heart failure (HF) is a common clinical syndrome representing the end-stage of a number of different cardiac diseases. It can result from any disorder that impairs the ability of the heart to fill with or eject blood. In the past three decades, there have been tremendous advances in the understanding of CHF which have led to dramatic improvements in medical therapy. In this article, an overview of the medications used for the management of CHF will be presented.

Medications are always the initial line of management of the patient with newly diagnosed CHF. The goals of therapy are to improve symptoms, reverse the deterioration in heart function, and to prolong survival. The number of drugs now available to improve symptoms and prolong survival has grown dramatically and includes angiotensin converting enzyme (ACE) inhibitors, beta blockers, diuretics, digitalis, angiotensin-receptor blockers (ARBs), hydralazine/nitrate combinations, and aldosterone antagonists. The initial therapy almost always includes diuretics (to improve the symptoms of congestion and swelling), ACE-inhitors, and beta blockers.

ACE Inhibitors

ACE inhibitors are the first-line of therapy in patients with CHF and were the first drugs to show improved survival. ACE inhibitors have demonstrated improved patient outcomes and survival in multiple large, clinical trials over the past two decades. They provide rapid hemodynamic benefit and improve symptoms quickly. They have been shown to be beneficial in a wide range of patients including patients without symptoms to the most severe cases of CHF. Commonly used ACE-inhibitors can be seen in **Table 1.**

ACE INHIBITORS
Lisinopril
Ramipril
Benazepril
Trandolapril
Enalapril

ACE inhibitors are commonly started at low doses and then are gradually increased overseveral weeks to months. Attempts should be made to obtain maximum doses as they were proven more successful in clinical trials of CHF. These drugs are usually well-tolerated, but several important side-effects need to be monitored for. **(Table 2)**

Common Side Effects
Cough
Low blood pressure
High potassium
Worsening kidney function

Table 2

Beta Blockers

Beta blockers slow the heart rate and reduce both the contraction of the heart muscle and blood pressure, thus decreasing the force with which the heart needs to contract. Beta blockers improve heart function, reduce symptoms of CHF, and improve survival of heart failure patients. Carvedilol (Coreg), metoprolol (Toprol), and bisoprolol (Zebeta) are the three specific beta blockers which have been shown to improve survival in patients with CHF. **(Table 3)** Other beta blockers should be avoided as they have not been shown in clinical trials to improve survival in CHF and are not FDA approved for this indication. **(Table 4)**

Like ACE inhibitors, beta blockers should be started at a low dose and titrated upward to the maximum dose tolerated. The initiation of beta

Continued on Next Page

Beta Blockers for CHF
Carvedilol (Coreg)
Metoprolol tartrate
Metoprolol succinate (Toprol XL)
Bisoprolol

Table 3

blockers needs to be done cautiouslyas symptoms may temporarily worsen in the first one to two months. Over time the improvement in heart function and symptoms are often dramatic. Side-effects from beta blockers can be significant and often the initiation

Common Side Effects of Beta Blockers
Slow heart rate
Worsening CHF
Exacerbation of asthma/lung disease
Sexual dysfunction
Fatigue

Table 4

should be performed by a cardiologist who is experienced with CHF.

Diuretics ("Water Pills")

Sodium and water retention lead to the common congestive symptoms that are characteristic of heart failure. Excess fluid can typically be controlled and symptoms improved by diuretic therapy. Improvement in symptoms can occur within hours to days. In comparison, the clinical effects of digoxin, ACE inhibitors, and beta blockers may require weeks or months to become fully apparent.

Diuretics in CHF
Furosemide (Lasix)
Bumetanide (Bumex)
Torsemide (Demadex)
Metolazone (Zaroxylyn)
Hydrochlorothiazide

Table 5

The most commonly used diuretic in CHF is furosemide (Lasix), although bumetanide (Bumex) and torsemide (Demadex) are used in patients with refractory swelling **(Table 5).**

In patients who have severe swelling and shortness of breath despite adequate doses of furosemide multiple diuretics can be used simultaneously.

Common Side Effects of Diuretics
Low potassium
Dehydration
Worsening kidney function
Low blood pressure

Table 6

In most cases, spironolactone (to be discussed below) and metolazone are added to enhance fluid removal and improve symptoms. Please see **Table 6** for the common side effects of diuretics.

Digoxin

Digoxin is one of the first heart medications developed and has been used for nearly 200 years for various cardiac conditions. It has been shown in clinical trials to improve symptoms, prolong exercise tolerance, and decrease hospitalizations in patients with CHF. However, digoxin has not been shown to prolong survival. As a consequence, digoxin is recommended for patients who have severe enough CHF to be hospitalized and those with refractory symptoms despite ACE inhibitors, beta blockers, and diuretics. Unlike many other drugs, blood levels of digoxin can be measured. Studies have shown that relatively low levels provide maximum benefit and that higher levels, particularly in women, increase mortality.

Digoxin has several side effects and needs to be monitored carefully. In addition, digoxin is removed from the body by the kidneys and levels needs

Side effects of Digoxin
Slow and fast heart rhythms
Visual disturbances
Nausea
Dizziness

Table 7

to measured frequently in patients with kidney impairment. Common side effects are seen in **Table 7.**

Angiotensin Receptor Blockers (ARBs)

ARBs are a relatively new class of drugs that work in a similar fashion to ACE inhibitors. ARBs are typically used in patients who cannot tolerate ACE inhibitors due to a persistent cough or an allergic reaction. ARBs are more expensive than ACE inhibitors and have been less well studied in CHF and as a consequence, ACE inhibitors are considered the first choice. Valsartan (Diovan) and Candasartan (Atacand) are FDA approved for CHF. Their side effect profile is very similar except they cause less cough and less allergic reactions.

Aldosterone Antagonists

Spironolactone (Aldactone) and Eplerenone (Inspra) prolong survival in advanced CHF and in patient with CHF after large heart attacks. Aldosterone antagonists are mild diuretics that block sodium retention in the kidneys. Major side effects include dangerous elevations in potassium and hormonal abnormalities. Eplerenone causes significantly less hormonal side effects, but this is outweighed by a dramatic difference in cost. Potassium levels and kidney function should be measured within the first few weeks that these drugs have been started.

Hydralazine/Nitrate Combinations

Hydralazine and oral nitroglycerin combinations are blood pressure medications that dilate the body's blood vessels and have been shown to modestly improve outcomes in patients with CHF. In patients with persistent symptoms despite beta blockers and ACE inhibitors it is reasonable to add this combination. Hydralazine and nitroglycerin do not affect kidney function and therefore play an important role in patients who are unable to tolerate ACE inhibitors due to kidney dysfunction. For reasons that remain unclear,

Commonly Used Hydralazine and Nitrate Formulations
Hydralazine (Apresoline)
Bidil (Hydralazine/Isosorgide dinitrate)
Isosorbide dinitrate
Isosorbide mononitrate

Table 8

hydralazine and nitroglycerin combinations have been shown to be more effective in reducing mortality and hospitalizations in African-American patients. **Table 8**

Hydralazine/nitrate combinations are often difficult to take as three times per day dosing is often necessary. Side effects are frequent and include dizziness, rashes, headaches, and rarely drug-induced lupus. Bidil is a combination pill which is more convenient, but thiis often outweighed by a dramatic increase in cost.

Drugs to Avoid or Use with Caution	Possible Complications
Anti-inflammatory Drugs (alleve, ibuprofen, motrin, mobic)	Increase fluid retention, elevated blood pressure
Heart Rhythm Drugs (Propafenone, Flecanide, Procainamide, Sotalol, Ibutilide)	Exacerbate CHF, increase heart rhythm problems, sudden death
Calcium channel blockers (Verapamil, Diltiazem, Norvasc, Plendil)	Exacerbate CHF, worsen heart function. (Norvasc and Plendil less of a problem than Verapamil and Diltiazem)
Certain Diabetic drugs (Avandia, Actos)	Increased fluid retention, exacerbation of CHF
Drugs for peripheral vascular disease (Cilostazol, Pletal)	Exacerbate CHF, shorten survival

Table 9

Drugs to Avoid in CHF

Unfortunately, it is extremely important to mention the medications that can worsen CHF and shorten survival. There are multiple medications that are commonly used that can exacerbate CHF, lead to hospitalizations, and cause side effects. A brief list of these drugs can be seen in **Table 9.**

Summary

As one can see, the medical management of CHF is extremely complex. The monitoring of the multitude of medications required is difficult and can lead to significant side effects if not done properly. However, these medications dramatically improve the symptoms of this disease, improve quality of life, and prolong survival.

About The Author

Jason Infeld, M.D., FACC is board certified in internal medicine, cardiovascular disease, and echocardiography. He received his medical degree from Albert Einstein College of Medicine where he was elected to the Alpha Omega Alpha honor society. He completed his internal medicine residency and cardiovascular disease fellowship at Columbia University in New York, New York. He received specialized training in congestive heart failure, congenital heart disease, and echocardiography. He has been a member of the Stern Cardiovascular Center since 2004 where he is clinical cardiologist and director of the lipid clinic. Call 901-271-1000 to contact Dr. Infeld.

American Heart Association 2008 Top Ten Research Report

Top research advances include studies that influence medical care, apply science to 'real world' communities

Clinical studies that may influence medical care and research that demonstrates how science can be effectively applied in the real world top the list of heart disease and stroke advances from 2008, said Timothy Gardner, M.D., president of the American Heart Association. The American Heart Association has been compiling an annual list of the top 10 major advances in heart disease and stroke research since 1996.

"It's always difficult to choose from among such a broad array of new discoveries," Gardner said. "This year we included not only novel work in fundamental or basic science, but also important clinical studies that we believe will influence medical care in the future. In addition, we have chosen a number of studies that demonstrate the effectiveness of science applied in the real world, from hospitals to schools to whole communities. These implementation studies are of increasing importance as we try to determine how best to translate basic and clinical science for the benefit of the public."

Achievements in 2008 include:

1. Breathing cleaner air

Smoke-free legislation and hospitalizations for acute coronary syndrome

Data regarding the dangers of first-hand smoke exposure is generally accepted, but the concept that secondhand smoke exposure can cause serious harm still meets resistance. This has made passing effective clean air legislation more difficult, though there has been substantial progress, with many states in the United States and several European countries passing smoke-free legislation. Previous studies of the effects of such legislation in individual towns and cities, while positive, have been criticized by some for lack of controls and incomplete data collection. However, a study in 2008, addressing many of these concerns, makes a very strong case for this type of public health initiative. In Scotland, after smoke-free legislation covering all enclosed places was implemented, hospital admissions for acute coronary syndrome decreased by 17 percent. This compared with only a 4 percent decrease in England, where there was no such legislation. The decrease in Scotland was highest in never-smokers, but there was also a smaller decrease in former smokers. Smokers had the lowest decrease, but still saw a 14 percent decline. A total of 67 percent of the decrease in hospital admissions involved nonsmokers, supporting the argument that protection for these individuals is an important benefit of this legislation, and that it should be extended more broadly.
Source: *New England Journal of Medicine,* July 31, 2008; *N Engl J Med* 2008; 359; 482-49. www.nejm.org.
Funding: Supported by a National Health Service Health Scotland project grant and salary support from the British Heart Foundation.

2. The acute care of patients with heart attack or stroke: Can we do better?

Hospital treatment of patients with ischemic stroke or transient ischemic attack using the "Get With The Guidelines" program; An organized approach to improvement in guideline adherence for acute myocardial infarction

Because evidence-based therapy to improve heart attack and stroke outcomes was not being uniformly delivered in hospitals across the country, quality improvement programs were developed. Their goal was to facilitate the work of healthcare providers, and ensure that each patient receives the appropriate therapy. Now, more than one million patients have been treated with the assistance of one of these programs, the American Heart Association's Get With The Guidelines (GWTG), and the effect of these programs on delivered care is now being reported. Two studies, evaluating the GWTG - Stroke and the GWTG - Coronary Artery Disease (CAD) modules of GWTG, demonstrate that participation in hospital-based quality improvement programs such as GWTG is associated with substantially improved hospital performance.
Source: 1) *Archives of Internal Medicine,* Feb. 25, 2008; *Arch Intern Med* 2008; 168:411-17; 2) *Archives of Internal Medicine*, Sept. 8, 2008; *Arch Intern Med* 2008; 168:1813-19; www.jama.ama-assn.org. **Funding:** 1) Supported in part by an unrestricted educational grant to the American Heart Association by Pfizer Inc. 2) Funded in part by a grant from the National Center for Research Resources (NCRR), a component of the National Institutes of Health (NIH); GWTG-CAD is supported by an unrestricted educational grant from Merck–Schering-Plough Pharmaceutical; data analysis was funded by a grant from the American Heart Association.

3. Type 2 diabetes: What is the legacy of intensive treatment?

10-year follow-up of intensive glucose control in type 2 diabetes; Long-term follow-up after tight control of blood pressure in type 2 diabetes

The UKPDS (UK Prospective Diabetes Study) has now followed patients with type 2 diabetes for 10 years after the initial period in which the benefits of HbA1c (blood sugar) control by drug treatment were established. In 2008, follow-up data demonstrated that, while the improvement in HbA1c levels was lost after the first several years, the reduction in diabetes-related endpoints and microvascular disease continued, and over time, a significant reduction in heart attacks and deaths from any cause emerged. This reinforces the benefit of good diabetes control in type 2 diabetes, adding importantly to the benefits demonstrated by the earlier Diabetes Control and Complications Trial. In contrast, in another section of the study, when the group in whom blood pressure was tightly controlled during the study was followed over the same 10-year period, the early reductions in any diabetes-related endpoint, including death, as well as in microvascular complications and stroke, was lost. This suggests that continued blood pressure control is critical to the maintenance of this risk reduction.
Source: *New England Journal of Medicine,* Oct. 9, 2008*; N Engl J Med* 2008; 359: 1577-89. www.nejm.org.
Funding: Supported for the first 5 years of post-trial monitoring by the U.K. Medical Research Council, U.K. Department of Health, Diabetes UK, the British Heart Foundation, and the U.K. National Institute for Health and for the final 5 years by Bristol-Myers Squibb, GlaxoSmithKline, Merck Serono, Novartis, Novo Nordisk and Pfizer.

4. The epidemic of childhood obesity: Can anything be done?

A policy-based school intervention to prevent overweight and obesity

There is no lack of concern about the increasing numbers of children suffering from overweight and obesity, especially as the related development of other cardiovascular risk factors has become clear. Literally hundreds of programs have been developed to address this major public health problem, but very few have been adequately evaluated with hard clinical outcomes. This past year a school intervention based on changes in policy, carried out in grades 4-6 in 10 urban schools, was conducted. The intervention's design incorporated school self-assessment, nutrition education, nutrition policy, social marketing, and parent outreach. Over a two-year period, this multi-component program led to a 50 percent reduction in the incidence of overweight in the intervention schools compared with the control schools. While there was no reduction in the incidence or prevalence of obesity, these results suggest that carefully designed, multi-component programs can have

an important impact on this serious epidemic.
Source: *Pediatrics,* April 1, 2008*; Pediatrics* 2008;121:e794-802. http://pediatrics.aappublications.org.
Funding: This study was supported by grants from the Centers for Disease Control and Prevention and the US Department of Agriculture/Food and Nutrition Service through the Pennsylvania Nutrition Education Program as part of Food Stamp Nutrition Education.

5. As we age – treating valvular heart disease.

Transcatheter valve implantation for patients with aortic stenosis: a position statement from the European Association of Cardio-Thoracic Surgery (EACTS) and the European Society of Cardiology (ESC), in collaboration with the European Association of Percutaneous Cardiovascular Interventions (EAPCI)

The treatment of severe aortic stenosis has remained in the realm of surgery, with no significant medical options and with balloon valvuloplasty offering no sustained symptomatic benefit and no survival benefit. While traditional aortic valve replacement improves symptoms and survival, it is accompanied by morbidity and mortality, especially in the elderly and those with co-morbid conditions. Over the past six years, transcatheter valve implantation for patients with inoperable or very high-surgical-risk aortic stenosis has gradually improved, with increasing operator experience and a number of devices available and others in development. As experience has grown, this percutaneous, catheter-based technique is beginning to offer a reasonable alternative to conventional surgery for high-risk patients with aortic stenosis.
Sources: *European Heart Journal,* May 13, 2008*; Eur Heart J* 2008; 29:1463-70. www.eurheartj.oxfordjournals.org.
Funding: Edwards Lifesciences provided an unrestricted grant for the practical organization of the meeting.

6. Stable coronary artery disease: what treatment is optimal?

Effect of PCI on quality of life in patients with stable coronary disease

Advances in the technology underlying percutaneous coronary interventions (PCI) have allowed an approach to more technically difficult vessels, and this has encouraged the use of PCI for treating stable angina. Indeed, the presence of a lesion has almost seemed to mandate that it be corrected. However, head-to-head comparisons with optimal medical treatment have not been done. In the COURAGE trial, patient outcomes, including quality of life, after PCI with optimized medical therapy were compared with patient outcomes of those on optimized medical therapy alone who had stable coronary disease. Both patients treated with PCI and medical therapy and those treated with medical therapy alone had marked improvements in health status during follow-up, suggesting that many patients currently are not receiving optimized medical treatment. Although the PCI group had small, but significant, incremental benefits during the first two years, by 36 months there was no difference in health status. This trial should lead to more aggressive medical treatment of patients with stable coronary disease, and more thoughtful utilization of PCI.
Source: *New England Journal of Medicine*, Aug. 14, 2008; *N Engl J Med* 2008:359: 677-87. www.nejm.org.
Funding: No specific funding of this study was noted, although full author disclosures are available on the manuscript.

7. Selecting patients for prevention

Rosuvastatin to prevent vascular events in men and women with elevated C-reactive protein

Substantial evidence demonstrates the effectiveness of lowering low density lipoprotein (LDL or "bad") cholesterol to prevent repeat cardiovascular events, so-called secondary prevention. While lifestyle modification is important, and several types of medications can be used to lower the LDL, most patients are prescribed statins, (HMG CoA reductase inhibitors). For primary prevention to reduce the risk of an individual's first event, the decision about whether to add medications to a healthy lifestyle is based on the patient's overall risk of an event, assessed by a measure such as the Framingham risk score. In the JUPITER (Justification for the Use of Statins in Prevention: an Intervention Trial Evaluating Rosuvastatin) trial, individuals (median age 66) were recruited if they had no known coronary disease and had LDL levels below 130, which usually would not mandate treatment with a statin. Some had Framingham scores indicating moderate risk, but a substantial number did not. All, however, were included in the study because of elevated hs-CRP levels, a marker of inflammation, a process that has been speculated to be another cause of atherosclerosis. Treatment with rosuvastatin in both groups resulted in nearly halving LDL cholesterol and highly sensitive C-reactive protein (hs-CRP) levels and produced a significant relative reduction in nonfatal heart attack (55 percent), nonfatal stroke (48 percent) and the composite endpoint including cardiovascular death (47 percent). This large (17,802 patients) study and its many subgroups have generated extensive discussion about its implications for expanding indications for the use of statins, and the findings will be considered as primary prevention guidelines are revised in 2009.
Source: *New England Journal of Medicine,* November 20, 2008; *N Engl J Med* 2008;359:2195-207. www.nejm.org; also presented at the American Heart Association's Scientific Sessions 2008.
Funding: Supported by AstraZeneca

8. A platform for new hearts

Perfusion-decellularized matrix: using nature's platform to engineer a bioartificial heart

In patients with very severe heart failure, mechanical artificial hearts are of substantial benefit. However, their limitations provide the background for the much greater potential of new biologic, or "bioartificial" hearts, in which the pumping is done by actual heart muscle, rather than metal and plastic. There are multiple problems to be solved before new hearts can be grown for patients dying from heart failure, but one hurdle was overcome this year. In a study using a rat model, researchers demonstrated that it is possible to create a functioning bioartificial heart using a matrix from which cells had been removed as a platform in which new immuno-compatible cardiac cells could survive and function.
Source: *Nature Medicine*, Jan. 13, 2008; *Nature Med.* 2008; 14: 213-21. www.nature.com/nm
Funding: This study was supported by a Faculty Research Development Grant to H.C.O. and D.A.T. from the Academic Health Center, University of Minnesota, Minneapolis, and by funding from the Center for Cardiovascular Repair, University of Minnesota, and the Medtronic Foundation to D.A.T.

9. The building blocks of new hearts.

Human cardiovascular progenitor cells develop from a KDR1 embryonic-stem-cell-derived population

To grow a new heart, it is necessary to have not only heart muscle cells, but also endothelial cells to line the inside of the heart as well as cells that develop into blood vessels. This year it was demonstrated that all three kinds of cells can be derived from a common cardiovascular progenitor cell, and that these progenitor cells can be derived from human embryonic stem cells. This is a critical observation for the process of building new hearts for those suffering from scarring or damage to their heart muscle.
Source: *Nature,* April 23, 2008; *Nature.*2008; 453: 524-28. www.nature.com.
Funding: No specific funding was listed for this study although several of the researchers are supported by the National Institutes of Health/ National Heart, Lung and Blood Institute.

10. Hypertension: who is too old to treat?

Treatment of hypertension in patients 80 years of age or older

It is well accepted that the effective treatment of high blood pressure is a very important factor in preventing cardiovascular complications such as stroke and heart failure. And while isolated systolic hypertension was a "normal" aspect of aging, we have also learned from the Systolic Hypertension in the Elderly Program (SHEP) study that control of this form of high blood pressure is also important. But questions remained about the very elderly, as they have usually been excluded from previous trials. This year, a critical piece of evidence was added, when the HYVET (Hypertension in the Very Elderly Trial) was completed. The results of this important trial provide evidence that effective antihypertensive treatment, even in persons 80 years old or older, is beneficial in reducing the risk of cardiovascular events, and thus extends the group in whom prevention must be pursued.
Sources: *New England Journal of Medicine,* May 1, 2008*; N Engl J Med* 2008; 358:1887-1898. www.nejm.org.
Funding: Supported by grants from the British Heart Foundation and the Institut de Recherches Internationales Servier.