Welcome

...to Volume 2 of Pensacola Heart Health. With this issue we have published three issues of our magazine, and we are proud of how far it has come and of the response we have received from our readers.

Last year was a monumental one for Cardiology Consultants, PA. We opened our newest facility in the Pace Medical Park on Highway 90. At approximately 5,200 square feet, this clinic has six exam rooms and offers numerous diagnostic imaging modalities, including nuclear medicine, stress testing, echocardiography, and vascular ultrasound.

Since our last publication, we also have recruited two physicians. Both are invasive, nuclear-licensed cardiologists who perform diagnostic caths, nuclear stress testing, and echocardiography, along with general clinical cardiology. Benjamin Lloyd, MD, completed his fellowship at the University of Missouri-Columbia and joins us after one year of private practice in St. Louis. Dr. Lloyd practices at the Gulf Breeze and Baptist Campuses. Steven Eilen, MD, was practicing in Atlanta for more than 20 years, and he now practices at our Sacred Heart Campus and Pace Campus.

We also had our first retirement from the practice when Steven Schang Jr., MD, stepped down in May after six years with Cardiology Consultants. Dr. Schang joined our practice after 25 years of solo practice in Pensacola. All of his patients continue to be seen by our cardiologists.

Cardiology Consultants continues to respond to our community’s changing needs and to deliver the highest quality of care to our patients and referring physicians. This is a dynamic practice in equally dynamic times.

Thank you for your support of our magazine and of our growing practice.

Andrew Radoszewski, MBA, MPH, CMPE
Administrator
Some 325,000 Americans fall victim to sudden cardiac death (SCD) each year. Many of these people die without any warning at all. An out-of-hospital cardiac arrest carries a 95 percent chance of dying. Despite the grim statistics, there are effective ways to help prevent SCD through careful screening and the use of implantable cardiac defibrillators (ICDs).

Of the screening modalities, the most important is the measurement of the ejection fraction. This tells physicians how effectively the heart pumps blood throughout the body. The ejection fraction is measured with several different methods, including the introduction of dye into the heart that is visible by X-rays, a nuclear study of the heart’s pumping action, or an echocardiogram (echo), which uses sound waves to assess the pumping function. The echo method is the least expensive and most time efficient way to diagnose a weak or failing heart.

Atherosclerosis, leading to heart attacks, is the most common cause of a declining or falling ejection fraction. Other causes include damage to the heart from viral infection, chronic hypertension, and genetic predispositions.

The normal ejection fraction is between 55 to 65 percent. Cardiologists target anyone who has an ejection fraction of less than 35 percent as a possible candidate for placement of an ICD.

The ICD combines the functions of a pacemaker with those of a defibrillator and monitors the patient's heart for any signs of arrhythmia (irregular heartbeat). The pacemaker function prevents the heart from beating too slowly, and if there is an episode of sudden death, the device shocks the heart in its defibrillator mode to restore normal rhythm.

Timing is crucial in preventing SCD. Without an ICD, people who experience SCD could suffer brain damage and death within minutes of the onset of the event. SCD is usually caused by very rapid heartbeats, known as ventricular tachycardia and ventricular fibrillation. Extreme slowing of the heart (bradycardia) causes a few cardiac deaths, but the defibrillator also can control this arrhythmia.

Defibrillators have evolved from large devices that had to be placed inside a patient’s body by open chest surgery to smaller devices that can be placed in the upper chest. I was the first heart rhythm specialist (electrophysiologist) to implant a defibrillator in Florida in 1993. My partners and I at Cardiology Consultants, PA, now implant hundreds of these devices in a simple procedure that is done under light sedation in less than two hours in most cases.

If you suffer from heart disease, contact your physician to see if you need your ejection fraction measured. If your ejection fraction is less than or equal to 35 percent, you may be a candidate for a defibrillator.

By S. Marcus Borganelli, MD, FACC

S. Marcus Borganelli, MD, FACC, attended Rice University and completed his medical degree at the University of Texas Southwestern Medical Center. His postgraduate training included an internship and a residency in internal medicine at the University of California, San Francisco. Dr. Borganelli also served a fellowship in cardiology at Vanderbilt University Medical Center and another in clinical electrophysiology at the University of Michigan. He is board certified in internal medicine, cardiovascular disease, and clinical cardiac electrophysiology.

References:

1 Sudden Cardiac Death, American Heart Association, www.americanheart.org
2 ibid
Acute Coronary Syndrome

Advances in therapy improve patient outcomes

By G. Ramon Aycock, MD, FACC

Acute coronary syndrome (ACS) accounts for more than 1.5 million hospital admissions each year. Of these, only 0.33 million cases represent ST elevation myocardial infarction (MI), or STEMI. Unstable Angina (UA) and Non-STEMI (NSTE-MI) account for the large majority remaining. All of these are characterized by cholesterol deposition in the coronary arteries (CAD). CAD remains the No. 1 cause of death in both male and female patients.

CAD begins at a young age, certainly by the 20s in the majority of patients. The progression of the disease is variable; but symptomatic disease is appearing at a relatively younger age. ACS takes approximately 10 years longer to present in women than it does in men. CAD has no cure, but recent advances in therapy have allowed significant improvements in outcome.

Evaluation and treatment
Evaluation and treatment of ACS should begin promptly in the emergency room. Initial history, physical exam, electrocardiogram (ECG), and specific laboratory tests are necessary for stratification into four subgroups: definite ACS, possible ACS, noncardiac chest pain, and stable angina. The first subgroup requires admission to the hospital and aggressive treatment protocols. The second subgroup should be stratified according to risk and may require inpatient evaluation. Any outpatient stress testing should happen within 72 hours. The last two groups likely require further outpatient evaluation.

Definite ACS should be stratified further into higher-risk or lower-risk groups. The higher-risk group is delineated by ECG changes, especially labile ST changes or significant ST depression, abnormal cardiac markers (Creatine Kinase — MB (CPK-MB), or troponin), clinical status (presence of continued chest pain or heart failure), reduced left ventricular (LV) function (ejection fraction (EF) < 40 percent), and older age. A history of prior MI or recent
cardiac intervention also is associated with higher risk. In general, this patient subgroup should be evaluated with an invasive strategy, to include cardiac catheterization. Catheterization results allow physicians to choose the best therapeutic approach, coronary artery bypass graft (CABG, or heart surgery), percutaneous coronary intervention (PCI, or angioplasty or stent), or medical therapy.

The lower-risk group should be stabilized on aggressive medical therapy and then possibly safely stratified to a more conservative strategy (with graded exercise testing), instead of cardiac catheterization, if desired by physician or patient. Conservative therapy should be abandoned for recurrent chest pain or abnormal stress test results. Patients with a negative stress test should reduce their risk factors and receive appropriate medical therapy. Additionally, causes of non-cardiac chest pain should be considered and evaluated as indicated.

Causes and prevention
ACS generally occurs as a result of plaque rupture, which accounts for at least 75 percent of events. The cholesterol plaque is made up of a fibrous cap that covers a core filled with cholesterol. The cap keeps the highly thrombogenic (clot producing) core away from circulating blood. A stable plaque is characterized by a thick cap and a small core. These plaques are associated with stable cardiac symptoms and have a low risk for plaque rupture. A vulnerable plaque, in contrast, has a thin cap and large core. These plaques are at much higher risk for rupture.

Plaque rupture occurs at the margins of the plaque, where there is a concentration of macrophages (scavenger cells), inflammatory chemicals, and enzymes. Under the proper conditions, a rupture at this margin can occur, leading to attraction and stimulation of blood platelets. The platelets then begin a healing process to close the rupture. This leads to the formation of a white thrombus (platelet-rich clot) with subsequent formation of a red thrombus (blood clot), which leads to reduced blood flow in the involved artery. Angina results, and if progressive blockage occurs, a heart attack may occur. Inciting factors include shear forces (blood flow issues), cigarette smoking, heavy physical or emotional stress, and certain drugs such as stimulants or cocaine.

To prevent progression of clot formation, treatment should be early and aggressive. Treatment with antiplatelet agents is a must. This includes aspirin, which should initially be given in a dose of 162 to 325 mg. Plavix is another agent shown to be effective orally, and for the highest risk groups (with abnormal troponins), an IV iiib/iiia inhibitor such as Integrelin should be used. Treatment also should include a medicine such as heparin or Lovenox to prevent the red thrombus. Early treatment with a beta blocker (Toprol XL or Coreg) reduces adverse events. It also is imperative to begin cholesterol-lowering treatment with a statin (Zocor, Lipitor, Crestor). This should be done no matter how low the cholesterol level is at presentation, as arterial stabilization may occur within hours of the first dose, well before cholesterol levels fall.

It also is important to know what not to do. Nonsteroidal anti-inflammatory drugs (NSAIDS), such as Motrin, Celebrex, etc., should be discontinued. They also should be avoided at discharge if possible. If they must be used, Naprosyn (Aleve) is probably safest from a cardiac standpoint. Other NSAIDS antagonize the antiplatelet effect of aspirin; Naprosyn does not. Hormone replacement therapy also should be stopped and not resumed if possible.

Cardiology Consultants, PA, prefers to perform cardiac catheterization on most if not all patients with ACS. This allows early evaluation with a gold standard procedure, as well as allows the best choice for definitive procedures such as CABG or PCI. As important as early and aggressive medical therapy is on admission, it is even more so following angioplasty and stenting. The long-term success of these procedures depends on excellent patient compliance with prescribed medications, smoking cessation, exercise, and diet changes.

At discharge, continuation of beta blockers offers cardioprotection and reduces cardiac symptoms and events. Aspirin should be continued indefinitely. A dose of 162 to 325 mg should be continued for the first month and for three months following placement of a medicated stent. Thereafter, a dose of 81 mg is safe and effective. Plavix should be continued in all ACS patients for at least one year, sometimes longer in patients with medicated stents. Renin angiotensin system blockers should be used in patients with reduced heart function. Statins remain the most important agents in the reduction of risk from elevated cholesterol levels, and they offer equal benefit in patients with low cholesterol levels at presentation.

Though cardiac interventions (stents, etc.) are excellent treatments for an acute event, long-term treatment is destined to fail without attention to aggressive risk factor modification and careful compliance with proper medical management.
The Aspirin Controversy

The truth behind the drug’s effectiveness

By Brent D. Videau, MD, FACC

Approximately 36 percent of adults in the United States, or more than 50 million people, take aspirin regularly to prevent cardiovascular disease. More people use this drug than any other drug in the world. Though physicians vary the doses they give to patients, data suggest that lower doses are safer and more effective for long-term use.

Aspirin, or acetylsalicylic acid, was first synthesized in 1897 by Frederick Bayer and Company. This pain reliever initially came as a powder, but because of frequent imitations, it was made into tablet form in the United States and the dose was chosen as 5 grains or 325 mg. A regular aspirin has 325 mg. The 81 mg children’s dosage, which is one-fourth of the adult dosage, was arbitrarily determined in 1922. In 1953, physicians noted that patients having tonsillectomies who had taken aspirin bled more. Physicians then realized that aspirin could thin the blood and unclog blocked arteries. It took another 30 years before clinical trials proved that aspirin could do just that — prevent cardiovascular disease.

Today, a wide range of aspirin doses, preparations, and methods of ingestion are available. Physicians prescribe doses ranging from 81 mg to 325 mg. Pharmacological studies and clinical trials show conflicting data on clinical benefit and effectiveness of the use of different dosages of aspirin. In addition, with a drug like aspirin, which is taken by such a huge population, even a low incidence of adverse effects can have a substantial impact.

Many studies have analyzed doses ranging from 30 mg/day to 1,300 mg/day. The one nearly constant finding in all these trials has been the lack of a relationship between increasing aspirin dosage and improved efficacy. In fact, the trend in benefit has almost uniformly favored lower dosages.

Pharmaceutical research suggests that 60 percent of physicians choose an 81 mg dose, and approximately 35 percent prescribe the regular adult-size aspirin pill of 325 mg/day.
Studies done in thousands of patients reveal the following: First, aspirins’ absorption rate and the onset of effect are significantly shortened by chewing the pill or drinking soluble aspirin (e.g., Alka Seltzer). Maximal inhibition of clotting is achieved within 20 to 23 minutes, as compared with the 60 minutes it would take to achieve an affect if the pill is swallowed whole\(^2\). Second, the maximal effect is only achieved with the larger 162 mg and 325 mg doses. For an acute event, like a heart attack, you must chew or dissolve and swallow at least 162 mg of aspirin for it to have a positive effect on unclogging the artery.

**Long-term and side effects**
Once aspirin has completely inhibited the clotting mechanism, only minimal doses are required to inhibit new platelets (the part of blood responsible for clotting) entering the circulation\(^3\). Low doses of 81 mg/day of aspirin are effective for long-term prevention in the majority of patients.

Questions have arisen regarding the effects of enteric coating on the biologic activity of aspirin. Some trials suggest that the coated aspirin is less effective. However, this evidence is conflicting and has been debated\(^4\).

Aspirin is so common that we give little thought to the adverse events associated with its long-term use. The major risk of taking aspirin long-term is bleeding from the gastrointestinal tract. A common belief is that ibuprofen-based drugs such as Motrin and Advil cause most bleeding. However, studies show that low-dose aspirin is the most common cause of GI bleeding\(^5\).

A common belief is that ibuprofen-based drugs such as Motrin and Advil cause most bleeding. However, studies show that low-dose aspirin is the most common cause of GI bleeding\(^5\).

**Dosages**
Although studies suggest that aspirin doses of 75 to 81 mg have the best clinical benefit, variability in response in individual patients has been recognized. Some trials suggest that up to 50 percent of patients treated with low doses of aspirin may not respond to it\(^6\). This data is difficult to reconcile with the clinical studies, which have all shown consistent benefit in patients with these lower doses.

**Conclusion**
No drug is used by a greater number of people worldwide than aspirin. Although safe, when used in such a large population, even a low rate of bleeding can become significant. When used in an acute event (e.g., heart attack) a person should take the larger dose of 162 to 325 mg of aspirin by chewing it or dissolving it in liquids. Once the patient has taken this higher dose for several days, the dosage can be reduced to 81 mg for long-term use in heart disease prevention. This type of multi-level dosage seems to give the best benefit with the lowest risk of adverse events.

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**References:**

2. Feldman M, Cryer B. Aspirin Absorption Rates and Platelet inhibition times with 325mg buffered aspirin tablets (chewed or swallowed intact) and with buffered aspirin solution. Am. J. Cardiol. 1999; 84: 404-409.
Congestive heart failure (CHF) is the leading cause of hospitalization in patients 65 years of age and older. It is one condition for which mortality continues to increase. According to the National Heart, Lung, and Blood Institute, the estimated direct cost associated with heart failure care in the United State is $33.2 billion annually.

Two-thirds of that is used for managing acute episodes of decompensated heart failure in the hospital.

CHF is a general term used to describe compromised or inadequate pumping of blood throughout the body by the heart. It is usually chronic, or long-term, and can have a number of causes, including high blood pressure, heart valve disease, atherosclerosis (coronary artery disease), heart muscle disease (cardiomyopathy), prior heart attack, and congenital heart defects.

Symptoms
Common symptoms include shortness of breath, edema (swelling around the legs, ankles, or other body parts), weight gain, nausea or vomiting, and general fatigue or weakness, especially after exertion. Most of these symptoms result from inadequate pumping by the heart.

The initial physical examination may reveal an arrhythmia (irregular heartbeat), distended neck veins, enlarged liver, or pleural effusion (fluid around the lungs). Several clinical tests, including echocardiogram, catheterization (placing a tiny tube inside the heart vessels), chest X-ray, chest computed tomography (CT) scan, or electrocardiogram (ECG) confirm diagnosis of CHF.

Depending on what the specific cause or causes of heart failure is in each individual patient, they can be effectively treated with a combination of lifestyle modifications (diet, exercise, and stress reduction) and drugs. Angiotensin converting enzyme (ACE) inhibitors, diuretics, and beta blockers for hypertension are the mainstays of therapy.

Biventricular pacemaker
A promising newer treatment for CHF is cardiac resynchronization therapy, involving the use of a biventricular pacemaker. A common result of CHF is that the left and right ventricles (lower chambers) do not pump in unison, as they do in a healthy heart. This only subtracts further from the heart’s ability to pump effectively and hastens the progression of the disease.

The biventricular pacemaker, using leads to both ventricles, synchronizes their contractions to increase the ejection fraction (amount of blood pumped). Sometimes a single lead implanted into the left ventricle is sufficient to correct the irregularity, which is added to the two standard leads of a regular pacemaker inserted into the right atrium and ventricle. This method of treatment has been shown to improve symptoms of heart failure for approximately half of patients who have been treated with medications but who still have moderate to severe symptoms.

While the overall death rate in the United States declined by 2 percent from 1994 to 2004, deaths from heart failure increased 28 percent.

Two-thirds of that is used for managing acute episodes of decompensated heart failure in the hospital.

Patients who are eligible for this treatment method include those who have moderate to severe symptoms of heart failure, are taking medications to treat their disease, and who have delayed activation of the heart. In some cases, this type of pacemaker is used to correct very slow heartbeats, and in others it is used in combination with an implantable cardiac defibrillator (ICD) to treat people at risk of cardiac sudden death.

While the overall death rate in the United States declined by 2 percent from 1994 to 2004, deaths from heart failure increased 28 percent.

References:
1 Congestive Heart Failure, American Heart Association, www.americanheart.org
3 Cardiac resynchronization therapy, WebMD, www.webmd.com/heart-disease
4 ibid
Cardiology Consultants, PA, has more than 20 years of experience conducting pharmaceutical and medical device research trials in inpatient and outpatient settings. The practice has more than a dozen clinical trials actively enrolling. Five of these trials are discussed below.

Inpatient trials
One trial is evaluating the safety and efficacy of an investigational drug-eluting stent compared with a Food and Drug Administration (FDA)-approved drug-eluting stent. In order to participate, subjects must have evidence of myocardial ischemia. This includes stable or unstable angina, a positive stress test, or reversible changes on an electrocardiogram (ECG).

An acute coronary syndrome (ACS) study is looking at the effectiveness of an investigational drug in reducing recurrent thrombotic events, such as death, myocardial infarction, and stroke. To meet inclusion criteria, subjects must have been admitted with ECG changes and/or elevated cardiac enzymes. Subjects are treated with either percutaneous coronary intervention or medical management and will be followed for 12 months.

Another trial evaluates the clinical benefit of two approved lipid-lowering medications in subjects with stabilized ACS. The trial evaluates which drug results in a greater percentage of subjects achieving a low-density lipoprotein (LDL) cholesterol of less than 70 mg/dL. Subjects must be admitted with either acute myocardial infarction or unstable angina and are stratified as statin-experienced or statin-naïve. Subjects will be followed every four months for a minimum of two and a half years.

Outpatient trials
An atrial fibrillation (AF) trial compares the efficacy and safety of an investigational drug with an FDA-approved drug for preventing stroke and systemic embolism. Subjects with nonvalvular AF and at least one risk factor are eligible for the trial. Risk factors include previous stroke, age 75 or older, left ventricular dysfunction, or age 65 or older with diabetes mellitus, history of coronary artery disease, or hypertension. Subjects will be followed for at least 12 months.
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