Cardiovascular Gram

A NEWSLETTER TO PHYSICIANS FROM THE PAT AND JIM CALHOUN CARDIOLOGY CENTER AT UCONN HEALTH CENTER

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Heart Failure

W. David Hagar, M.D., Marybeth Barry, APRN, Laura Kearney, RN

An 83 year old woman with diabetes, hyperlipidemia, chronic renal disease, and anemia was admitted to the hospital in January 2007 because of shortness of breath. She had a history of hypertension and in 1998 had a right femoral artery bypass for an iliac artery occlusion. In February, 2006, she developed chest pain with ST segment elevations anteriorly and had emergent angiography. The left ventricular ejection fraction was 15%. The circumflex and right coronary arteries were chronically occluded, and the left anterior descending was acutely blocked. This artery was opened and stented with a drug eluting stent. She was followed in the Heart Failure Center and did well for several months. Between November 2006 and January 2007 she gained eleven pounds. She developed edema and marked orthopnea and was admitted for acutely decompensated heart failure.

This patient represents many ongoing issues in heart failure. A consequence of the successful treatment strategies for heart failure is further disease progression in an aging and therefore increasingly frail population requiring repeated hospitalizations and the management of multiple co-morbidities. This short article from the staff of the Heart Failure Center will focus on the newer ways to classify and define treatment options for patients with heart failure. While much of the discussion is focused on systolic failure, these principles also apply to the 45 to 50% of the heart failure population whose congestion is characterized by preserved systolic function and impaired diastolic function. Guidelines and recommendations for the management of heart failure and for discharge planning of inpatients have been established. The classic way of describing the severity of a patient's heart failure is to assign the patient to a New York Heart Association functional class.

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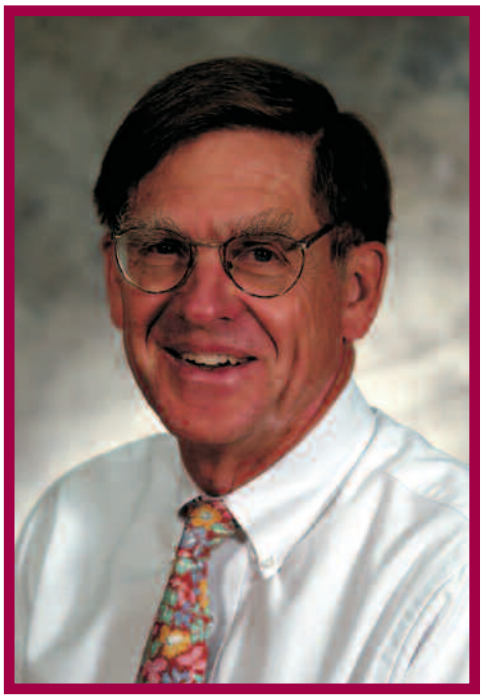

W. David Hager, M.D.,
Director of Heart Failure Program

In this issue:

Editor: Dr. Bruce T. Liang
Co-Editor: Dr. Arnold Katz

Director's Column

Director of the Pat and Jim Calhoun Cardiology Center, Chief of Cardiology

Dr. Bruce T. Liang

Diabetes mellitus and diabetic coronary disease represent significant cardiovascular health burden in the U.S. with the number of diabetic subjects projected to increase to at least 29 million people in 2050. The global atherosclerotic burden and prevalence of multivessel disease are similar in diabetic patients without a history of coronary heart disease (CHD) and in non-diabetic patients with a history of CHD. The prevalence of CHD is 2-4% in the general population while that of CHD in adult diabetic patients approaches 55%. Compared to those without diabetes, patients with diabetes have a mortality that is twice as great in men and 5 times higher in women. Heart disease is responsible for one-half of all deaths in patients with diabetes. Thus, diabetes is considered a CHD equivalent, that is, having diabetes is similar to having a history of CHD.

For these reasons, treatment of patients with diabetes requires a more proactive approach. Screening for CHD in diabetic subjects is indicated for those with typical or atypical CHD symptoms, those 55 years or older, those with peripheral or carotid vascular disease, and those with 2 or more of the following risk factors: dyslipidemia, hypertension, smoking, family history, microalbuminuria, and retinopathy. Because of the prevalence of diabetic neuropathy, diabetic patients with CHD may have no symptoms. Detection of CHD involves the use of stress testing such as stress echo-cardiography, nuclear imaging stress testing, and more recently, the use of multi-slice CT coronary angiography. At the Calhoun Cardiovascular Center, we have the expertise and experience on these procedures and offer them on a routine basis.

Lifestyle modification by dietary measures and physical exercise aiming at weight reduction as well as pharmacological interventions such as the use of aspirin and a statin are warranted even in those without evidence of CHD or elevated cholesterol. The blood pressure targets are <130/<80 mm Hg in diabetic patients. Diabetic patients with hypertension tend to require 3 or 4 antihypertensive drugs to lower the BP to the recommended level. ACE-I and ARB are first-line therapy. Beta1-selective adrenergic blocker and thiazide diuretic agents are viewed as second-line therapy in diabetic hypertensive patients. Recent studies demonstrated that ACE-I can prevent the onset of diabetes in those without history of diabetes (to cite a few references J. Hypertension 22:645-652, 2004; Circulation 107:1291-6). However, these studies must be interpreted with caution since diabetes prevention was not the primary endpoint in any of the studies. In terms of reduction of MI, stroke or cardiovascular event, the current guidelines recommend primary prevention with ACE-I therapy in diabetic patients with one other CHD risk factor and secondary prevention in diabetic patients with CHD.

Finally, diabetic patients develop diabetic cardiomyopathy which is characterized by abnormal LV relaxation while systolic function is still normal. When shortness of breath or other symptoms occurs, they then experience isolated diastolic heart failure. Myocardial cell death, likely mediated by apoptosis, followed by fibrosis, plays an important etiologic role. The use of echocardiography tissue Doppler and trans-mitral inflow will make the diagnosis of the diabetic diastolic cardiomyopathy. Both methods are developed and used in the Echo-cardiography Laboratory here at the Calhoun Cardiology Center.

Thanks to Dr. Deckers and the leadership at the UConn Health Center, the new cardiac catheterization laboratory is now completed and operational. The new laboratory offers state-of-the-art catheterization and interventional care, led by Dr. Michael Azrin and supported by interventionalists Drs. David Hager and Kanwar Singh (Dr. Singh, currently at Duke University, will join the group in July) as well as by a capable team of nursing and technical staff led by Maureen Worley. We look forward to continuing the tradition of excellent cardiovascular care here at the cardiology center. Meanwhile, we hope that all will have a productive as well as an enjoyable summer!

Bruce T. Liang, M.D.
Ray Neag Distinguished Professor

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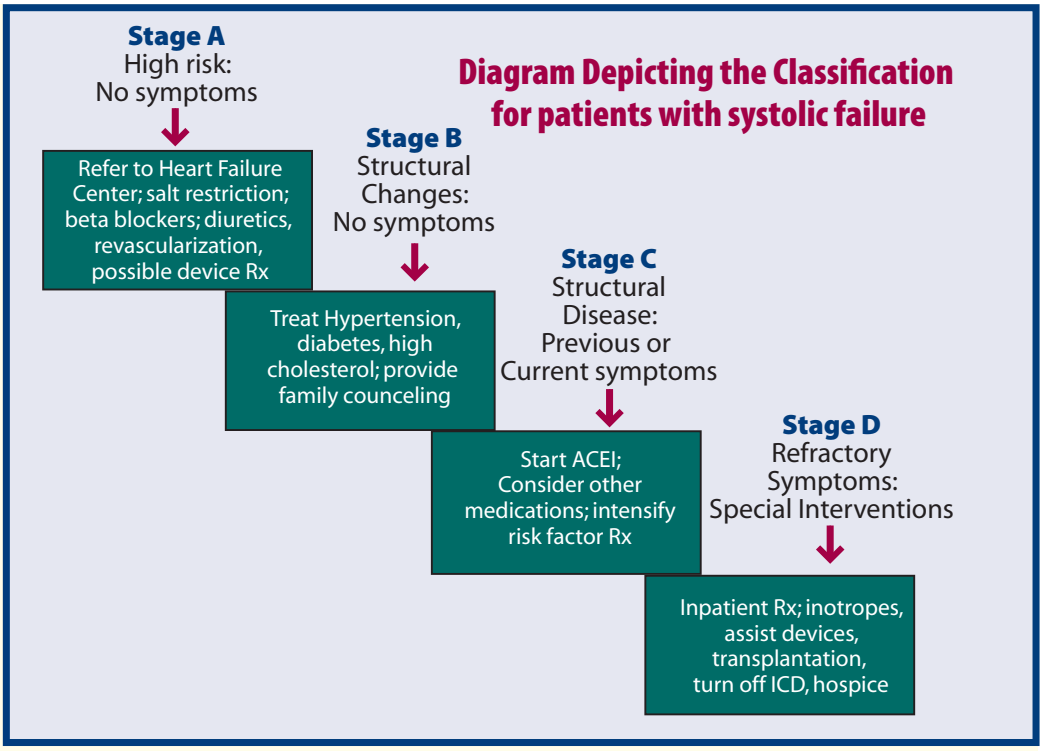
CLASS I: No symptoms at any level of exertion
CLASS II: Symptoms with heavy exertion
CLASS III: Symptoms with light exertion
CLASS IV: Symptoms with no exertion

This system defines the degree to which heart failure limits a patient's lifestyle at any one point in time. A patient can move from class to class depending on symptoms, and there is a certain amount of subjectivity in assigning the class. In 2001, the American College of Cardiology and the American Heart Association developed a new classification of heart failure which defines the evolution and progression of heart failure by defining four stages, A through D.

STAGE A: At risk for developing heart failure without evidence of cardiac dysfunction
STAGE B: Evidence of cardiac dysfunction without symptoms
STAGE C: Evidence of cardiac dysfunction with symptoms
STAGE D: Symptoms of heart failure despite maximal therapy

In this system, a patient can move in only one direction. Someone with Stage C heart failure may be in NYHA Class I, II, or III. Whereas someone like the patient described may have a NYHA Class IV status on admission, that can change to Class III with a little diuresis. However, once someone is defined as being in Stage C, there is no reversal. Only progression to Stage D would be possible.



The diagram above depicts this classification for patients with systolic failure:[1,4]

This staging permits an approach to heart failure that allows treating the risk factors before symptomatic and structural changes occur. For example, the genetic profiles of some cardiomyopathies are beginning to be defined and may permit treatment and allow family counseling. Hypertension, hypercholesterolemia, and diabetes are often apparent before vascular injury occurs, and the treatment options are multiple.

One of the continuing challenges is finding a cost effective way to screen for patients in Stage A. The use of biomarkers, such as brain natriuretic peptide, inflammatory markers such as C-reactive protein, echocardiographic imaging, and scanning for coronary calcifications have all been used.

For patients in Stage B with evidence for structural disease but no symptoms options for treatment are multiple. For example, the SOLVD Prevention Study (Studies of Left Ventricular Dysfunction) showed that angiotensin-converting enzyme inhibitors are effective in treating patients without symptoms but with ejection fractions < 35%.

Refining approaches to preventative treatment will benefit the greatest number of patients. Whereas the greatest expenditure of resources occurs in managing Stage C and D patients, most patients in this staged profile of the heart failure population are in Stages A and B.

When a patient reaches advanced Stage C or early Stage D hospitalization is often required. At this point defining a patient's hemodynamic profile is helpful in determining therapy. A practical way to design therapy for patients hospitalized with acutely decompensated heart failure is to assign patients to one of four hemodynamic classes as defined by Dr. Lynne Stevenson and her colleagues.[5,6]

Usually patients in class A do not need hospitalization. About two thirds of the patients admitted with acutely decompensated heart failure are in

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class B and are warm and wet. Another one third are in class C and are cold and wet. A low percent (5%) are in class L and are cold and dry. The prognosis in terms of death or needing transplantation is about 25% for class B and 40 % for class C at one year.

Patients in class B need enhanced diuresis as well as maintenance therapy with ACEI, ARB's, beta blockers, and aldosterone antagonists. Sometimes outpatient intravenous diuretic administration in the Heart Failure Center can avoid hospitalization. For patients in class C intravenous inotropic therapy may be required before diuresis is possible. Outpatient beta blockers and ACEI may need to be discontinued or reduced because of the reliance these patients may have on the vasoconstricting reflexes needed to support the circulation. Inotropic therapy is associated with increased risk and mortality, but stabilization often requires its use.

Often patients hospitalized have evidence of renal dysfunction, atrial fibrillation, and anemia. Consultation with the nephrologists, restoration of sinus rhythm, and transfusion to a hemoglobin level > 10 gm/dl may help to optimize responses to cardiac medications. Upon discharge administration of iron and epogen often adds further support.

Probably over 30% of patients hospitalized with decompensated heart failure are rehospitalized within three to six months. Often discharge is rushed and a favorable outpatient plan is not established. To optimize care the team at the Brigham and Women's Hospital in Boston has developed the following criteria for discharge:[4]

Assessment of Hemodynamic Profile

Evidence for Congestion
Orthopnea
High JVP & S
Ascites & Edema[3]

Evidence for Perfusion
Narrow pulse pressure
Pulses Altermans
Cool forearms and legs
Low sodium level
Poor renal function

Low Perfusion at Rest

	Congestion at Rest: No	Congestion at Rest: Yes
Low Perfusion at Rest: No	A: Warm & Dry	B: Warm & Wet
Low Perfusion at Rest: Yes	L: Cold & Dry	C: Cold & Wet

In addition to these approaches to treating heart failure which have been outlined, the availability of biventricular pacing units, internal defibrillators, ventricular support devices, and transplantation permits extension of life even at a time when the heart failure syndrome has progressed to end stage. The decisions to use these means of support are similar to the decisions made between physicians and patients with advanced malignancies. Individualization, maintenance of dignity and life quality, and consistency with a patient's wishes all go into the discussion. At some point support from hospice and alleviation of pain with analgesics may be the appropriate therapies.

The staff of the Heart Failure Center follows many patients like the individual presented at the beginning of this article. By participating in clinical research trials through the years it has been possible to be involved in the development of many of the therapies and approaches which have been mentioned for both inpatients and outpatients. Finding ways to continually educate ourselves and our patients is an ongoing process and hopefully leads to continued improvement in patient care.

1. How much sodium is in a teaspoon of salt? *(Answer: a)*

a. 2400 mg c. 750 mg
b. 1650 mg d. 900 mg

2. Can you name three foods that are high in sodium content? *(catsup, soy sauce, bouillon cubes, popcorn, can of tomato soup {1775mg}, pickles, sardines, etc)*

3. A single serving of which of the following foods contains the most sodium? *(Answer : b)*

a. Apple c. Doughnut
b. Canadian bacon d. Pretzels

4. Do you know the number to call if you have to reach the Heart Failure Center personnel? *(Answer: Laura Kearney, RN: 860-679-4134; Marybeth Barry, APRN: 860-679-4121)*

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CARDIAC VALVE SURGERY – 2007

Author: Paul Preissler, M.D.,
Clinical Chief of Cardiothoracic Surgery,
John Dempsey Hospital/University of Connecticut Health Center

Paul Preissler, M.D.

Surgery for heart valve abnormalities is safer, more accurate, and more predictable than ever before. Since the 1960's when valve replacement first became available, pre-operative assessment was crude, and operations were rudimentary and timed as desperate interventions, when all else had failed, modern techniques of diagnosis, surveillance, and reconstruction have evolved to levels where heart valve surgery is largely elective, completely restorative and at virtually no risk.

Two relatively modern developments have made this progress possible. First is modern echocardiography. By imaging the heart and its valves with ultrasound, abnormalities can now be defined early and accurately, and surgery can be properly timed. Historically, when only symptoms and the stethoscope were available, patients with heart valve pathology often developed myocardial dysfunction, left ventricular hypertrophy (both heart muscle abnormalities) and/or pulmonary hypertension (excess pressure in the blood flow into the lungs). These secondary pathologies increased risks and made surgical outcomes unpredictable.

Through the use of echo surveillance, we now realize that heart valve surgery should be done before or at the very first signs of the development of these secondary problems. If surgery for heart valve pathology is done while the heart muscle is still normal, good patient outcomes are expected. Normal valve function together with normal muscle function yield a safe and normal future.

In addition, the surgery for heart valve abnormalities is more sophisticated and accurate than ever before. New prosthetic (artificial) valves are durable and safe, routinely remaining problem free beyond 20 years. Techniques for the repair and reconstruction of leaking mitral valves allow the patient to maintain their own "living" valve reconstructed from their own heart valve tissue, which are essentially "cured for life."

Some isolated single valve operations are now done through "small incision" or "minimally invasive" approaches so that there is less external scarring. While some aortic valve abnormalities can also be repaired, the majority of aortic operations are replacement procedures. Isolated mitral valve repair operations for myxomatous mitral insufficiency (leaking or floppy mitral valve) now are done with outcomes approaching near zero percent mortality.

These modern, up-to-date, approaches are routinely available at UCHC. Here, heart valve abnormalities, often first detected by the recognition of a murmur, can be diagnosed quickly. Therefore, surveillance or intervention can be planned to preserve and protect heart function so that patients can return to full function with safe future.

STATE OF CARDIO-PULMONARY BYPASS AND PERFUSION

Author: David Rosinski
Head of Cardio-Pulmonary Perfusionist Service

David Rosinski

For over 50 years, open heart surgery has been performed by placing a patient on cardiopulmonary bypass and artificially supporting the circulation for a period of time allowing a surgeon the ability to operate upon the heart.

Despite the evolution of cardio-pulmonary bypass the current practice of open heart surgery via management of patients on a heart-lung machine remains unphysiologic body temperature is cooled, blood is 100% anticoagulated, hemodilution occurs, blood pressure is non-pulsatile, the heart is stopped from beating, the lungs are deflated, and a variety of end organ complications can occur. Today, evidence-based best practice models for management of patients on cardiopulmonary bypass are evolving and the consequences of the heart-lung machine are being ameliorated by improved technology and enhanced practice techniques. Here at the University of Connecticut Health Center, state-of-the-art management of cardiopulmonary bypass is in place and is continuing to evolve with enhanced clinical outcomes. The current heart-lung circuit for extra-corporeal circulation has been customized as a "mini" circuit reducing the effects of hemodilution and inflam-mation. Historically the hematocrit on bypass would routinely drop 10 points. Today, a drop in hematocrit will only be 2 to 3 points. Today's standard of red blood cell conservation allows the surgical team to maintain a hematocrit of >25% during Cardiopulmonary bypass so as to avoid transfusion.

Platelet count and function are preserved now 95% of open heart cases requiring cardiopulmonary bypass maintain a platelet count over 100,000 thereby avoiding the need for platelet transfusion. The inflammatory response to cardiopulmonary bypass is minimized using state of the art biocompatible surface polymer technology. All artificial blood contacting surfaces are coated with a hydrophilic/hydrophobic biopassive polymer that reduces the systemic inflammatory response to bypass. These surface coatings prevent protein denaturation and platelet consumption. A non-invasive brain specific cerebral blood flow monitor used on all cases allows real time evaluation of cerebral blood flow during surgery. All physiologic parameters such as electrolytes, glucose, blood gases and monitored real time and maintained within normal limits.

Best practice management of patients requiring cardiopulmonary bypass for open heart surgery is a multimodality team approach that improves outcomes. Case management is a coordinated effort between cardiac surgeon, cardiac anesthesiologist and perfusionist in the operating room that assures the highest standards of quality at the University of Connecticut Health Center.

ION CHANNEL MUTATIONS AND SUDDEN DEATH

Author: Arnold M. Katz M.D.

Last week, a high school student in a nearby town collapsed during football practice and was found to be in full cardiac arrest. Fortunately the coaches knew CPR and had access to a defibrillator, and trained paramedics were nearby, so that the student was quickly resuscitated. After being taken to hospital he woke up and appears to be on the way to full recovery. Unfortunately, not all stories like this have a happy ending. It is for this reason that prevention, rather than treatment, is the ideal way to prevent sudden death on the athletic field and elsewhere.

There are many reasons why the heart of an otherwise young person stops beating; a recent article lists 20 mechanisms that include abnormalities of the coronary arteries, molecular disorders of heart muscle that lead to cardiomyopathies, and inherited abnormalities involving genes that encode cardiac ion channels.[1] The latter are membrane proteins whose opening and closing are responsible for initiating and propagating the electrical signals that control the heart beat.[2] Research carried out over the past decade has identified literally dozens of mutations involving six key proteins that participate in generating these electrical signals.

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These molecular changes, along with abnormalities in several other regulatory proteins, are now known to cause sudden cardiac death in patients like the student mentioned above.[2-4]

Two important features of these molecular syndromes are described in a recent article by Peolzing et al.[5] The first confirms earlier reports that the major effect of some ion channel mutations is reduced "trafficking" of the abnormal protein, which refers to transport from the site inside the cell where the protein is synthesized to the surface membrane where the channel carries out its functions. In some cases, although the channel is not normal, it can still function, which allows interventions that increase trafficking of the abnormal protein to reduce the risk of sudden death. (It is not surprising that these findings have stimulated a search for drugs that can increase trafficking.) The other new finding in this paper, which I find to be even more remarkable, is that patients with a sodium channel abnormality caused when replacement of a single amino acid increases the risk of sudden cardiac death, can be protected by a second molecular abnormality in a different region of the same channel. The latter, which by itself is clinically unimportant, causes a commonly observed change in molecular composition called a polymorphism. Quite unexpectedly, the polymorphism was found to prevent arrhythmias in patients who carry the dangerous mutation. The underlying mechanism for this "rescue" was shown to be the ability of the polymorphism to increase trafficking of the abnormal protein encoded by the first mutation.

These fascinating findings, in addition to explaining why some carriers of potentially dangerous gene mutations do not suffer adverse effects (a phenomenon sometimes called "incomplete penetrance"), suggest novel targets for therapy that could prevent arrhythmias in individuals who carry potentially lethal ion channel abnormalities. Hopefully, future research will provide new ways to prevent potential tragedies such as that which occurred last week.

References

[1] *Maron BJ, Pelliccia A. The heart of trained athletes. Cardiac remodeling and the risks of sports, including sudden death. Circulation 2006;114:1633-1644.*

[2] *Katz AM. "Arrhythmias", in Physiology of the Heart. 4th Ed. Philadelphia, Lippincott/Williams and Wilkins, 2006. pp. 462 – 521.*

[3] *Priori SG. Inherited Arrhythmogenic diseases. The complexity beyond monogenic disorders, Circ Res. 2004;94:140-145.*

[4] *Antzelevitch C, Brugada P, Borggrefe M, Brugada J, Brugada R, Corrado D, Gussakl, LeMarec H, Nademanee K, Riera ARP, MD; Shimizu W, Schulze-Bahr E, Tan H, Wilde A. Brugada Syndrome. Report of the second consensus conference. Circulation 2005;111:659-670.*

[5] *Poelzing S, Forleo C, Samodell M, Dudash L, Sorrentino S, Anaclerio M, Troccoli R, Iacoviello M, Romito R, Guida P, Chahine M, Pitzalis M, Deschênes I. SCN5A polymorphism restores trafficking of a Brugada Syndrome mutation on a separate gene. Circulation 2006;114:368-376.*

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Discharge Criteria After Hospitalization for Heart Failure

Clinical Status Goals

Achievement of Dry/Weight
Definition of Optimal BP
Walking without dizziness or dyspnea

Stability Goals

24 hrs without changes in oral heart failure regimen
48 hours off intravenous inotropic agents
Fluid balance even on oral diuretics
Renal function stable or improving

Home Maintenance Plan

Patient & family education about:
Sodium restriction (2 gm.)
Fluid limitation
Daily weights & call for gain of ≥ 3 pounds
Medication schedule
Medication effects
Exercise prescription
Flexible diuretic plan
Scheduled call to patient within 3 days
Indications for when to call nurse, MD, or 911
Clinic appointment in 5-10 days

[1] *ACC / AHA Guidelines for the Evaluation and Managemnet of Chronic Heart Failure in the Adult: Executive Summary, JACC 2001;382102-2113.*

[2] *Executive Summary: HFSA 2006 Comoprehensive Heart Failure Practice Guideline, J Card Failure 2006;12:10-38.*

[3] *Tang WHW, Francis GS, The Year in Heart Failure, JACC, 2006;48:2575-2583.*

[4] *Jessup M, Brozena S, Heart Failure, N Engl J Med 2003;348:2007-2018.*

[5] *Nohria A, Lewis E, Stevenson LW, Medical Management of Advanced Heart Failure, JAMA 2002;287:628-640*

[6] *Nohria A, Tsang SW, Fang JC, et al, Clinical Assessment Identifies Hemodynamic Profiles That Predict Outcomes in Patients Admitted With Heart Failure, JACC 2003; 41:1797-1804.*

Studies Honors, and Awards

selected examples

- The Cardiovascular Program at the Calhoun Center received the 2006 Care Science Award as a National Quality Leader in the treatment of acute myocardial infarction.
- Young Investigator Award from the International College of Angiology to Dr. Manika, Das for her lecture titled 'Ceramide signalling in the ischemic and preconditioned heart', 48th Annual World Congress. ICA2006, Charlotte, North Carolina.
- Sc.D. and the highest honor [HONORARY SCOUZA] from the University of Debrecen was presented to Dr. Dipak Das during November, 2007.
- mAKAP-orchestrated phosphorylation events: regulation of PDE4D3, RO1 HL082705, Dr. Kimberly L Dodge-Kafka.
- Mechanism of Myocardial Angiogenesis in Transgenic/Knockout Animals, 1R01HL085804-01, Dr. Nilanjana Maulik.
- IMPROVE-IT A Multicenter, Double-Blind, Randomized Study to Establish the Clinical Benefit and Safety of Vytorin (Ezetimibe/Simvastatin Tablet) vs. Simvastatin Monotherapy in High-Risk Subjects Presenting with Acute Coronary Syndrome, Schering-Plough Research Institute, Dr. Michael Azrin.
- The EXACT Study, a post FDA device approval trial for the EXACT carotid stent, Dr. Michael Dahn.
- Persantine: Variation in Response, Department of Defense, Drs. Michael Azrin and Bruce T. Liang.
- "Effects of potassium alginate on ambulatory blood pressure in subjects with prehypertension" – Ocean Nutrition Canada, Dr. William White.
- TOPCAT,"Treatment of Preserved Cardiac Function with an Aldosterone AnTagonist," an NIH sponsored trial on aldactone and cardiac mortality in patients with EF > 45%, Dr. Dave Hager.
- "PROVE," study on the effect of anti-tachycardia programming (ATP) on reducing need for shock, Dr. Matt Stoenescu.
- Exercise testing for detection of stress induced pulmonary hypertension in patients with scleroderma, Drs. Naomi Rothfield, Raymond J. Foley, and David I. Silverman.
- New Inflammatory Markers in Human Atherosclerotic Plaques, Drs. Liang, Menzoian, Dahn, Bhimidi, Samee.
- FUSION II, follow-up serial infusions of natrecor for the management of patients with heart failure, Dr. David Hager (P.I.), Ms. Marybeth Barry, coordinator.
- Stradivarius: Rimonabant IVUS Study, Sanofi Synthelab. Dr. Michael Azrin.
- Dr. Schulman was named a "Top Doctor" in Cardiology in July issue of Hartford Magazine
- Marybeth Barry, Laura Kearney, Dave Hager et al presented at the Heart Failure Society of America meeting in Seattle Washington on "Heart Failure Caregivers: An Assessment of Burden and Concern."
- Dr. Michael Dahn received training and is initiating a new approach to treatment of descending thoracic aneurysm with Gore Thoracic Aortic Graft via an endovascular percutaneous approach
- Dr. Matt Stoenescu has successfully employed a new device by Cardio-Optics to see inside the heart via an endovascular approach while performing bi-ventricular pacer implantation.

Noteworthy Events

- Cardiovascular Grand Rounds (8-9 am in Link Room):
- Dr. Muredach Reilly, University of Pennsylvania, title TBA, Sept 12th, 2007.
- Dr. Frederick Schoen, Harvard Medical School, title TBA, September 19th, 2007.
- Dr. Dipak Das, University of Connecticut Health Center, Oct. 17th, 2007.
- Dr. Stephen Kimmel, University of Pennsylvania, title TBA, December 19th, 2007.
- Citywide Conferences: Every 2nd Friday at 7:30 am, C2136, contact Ms. Barta 860-679-2771.

Physicians and Surgeons

- Cardiologists, Electrophysiologist (679-3343 or 2626), and Interventionalists (679-2828)
  - Dr. Michael Azrin
  - Dr. Michael Fucci
  - Dr. David Hager
  - Dr. Moz Karimeddini
  - Dr. Bruce T. Liang
  - Dr. Peter Schulman
  - Dr. David Silverman
  - Dr. Matt Stoenescu
  - Mr. Scott Harrison, PA
  - Mr. Brad Biskup, PA
  - Ms. Marybeth Barry, APRN
  - Ms. Jill Panetta, APRN
- Hypertension Specialists (679-3343)
  - Dr. William White
  - Dr. Beatriz Tendler
- CT Surgeons (679-3343 or 2626)
  - Dr. Daniel Fusco
  - Dr. Jonathan Hammond
  - Dr. Paul Preissler
  - Dr. David Underhill
  - Ms. Dorota Pawlak, APRN
  - Ms. Thersa Barnett
- Vascular Surgeons
  - Dr. Michael Dahn (679-3540 or 4801)
  - Dr. James Menzoian (679-3540 or 4801)
  - Dr. David Underhill (679-3343)
- Grove Hill Cardiologists (223-0220)
  - Dr. Sanjayant Chamakura
  - Dr. Alan Kudler
  - Dr. Jared Insel
  - Dr. Jan Paris
  - Dr. Morgan Werner

Scientists

Dr. Dipak Das
Dr. Kimberley Dodge
Dr. Bruce T. Liang
Dr. Nilanjana Maulik
Dr. Achilles Pappano
Dr. Jian-bing Shen
Dr. Lixia Yue

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