

Cardiovascular Gram

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

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Update on Perioperative Cardiovascular Management of Surgical Patients

Author: Peter Schulman, M.D., Director of Cardiology Fellowship Program
Senior Cardiologist of the Calhoun Cardiology Center



Peter Schulman, M.D.

Each year, hundreds of thousands of surgical procedures are performed in the United States. As the US population grows and the median age increases, more procedures are being performed on older adults. Given the high prevalence of cardiovascular disease in the population, it's not surprising that an increasing number of non-cardiac surgical procedures are performed on patients with cardiac disease. All but the simplest of surgical procedures can impose significant stresses on major organ systems, most notably the pulmonary and cardiovascular systems. With respect to the cardiovascular system, the two most stressful periods for the patient undergoing major surgery occur during the induction of anesthesia and about three days postoperatively. During the stress of induction of anesthesia, blood pressure and heart rate can rise substantially, which can lead to myocardial ischemia (insufficient supply of blood and oxygen to the heart).

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

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During the second major stressful period, about three days after the surgery, the blood is hypercoagulable (especially prone to clotting), powerful intravenous pain medications are being weaned to less potent pills, patients are getting out of bed for prolonged periods, and fluids lost into the tissues during surgery are completing their reabsorption into the bloodstream. All of these factors can lead to clot formation, activation of the sympathetic nervous system, intravascular fluid overload, and substantial stress on the heart that elevate the risk of heart failure, unstable angina, or heart attack.

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Director's Corner

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

Dr. Bruce Liang

Hypertrophic heart disease is an extremely common manifestation of cardiovascular disease, and is in fact a result of virtually all forms of heart disease such as hypertension, diabetes, coronary artery disease, and certain genetic causes such as hypertrophic obstructive cardiomyopathy (HOCM). Cardiac hypertrophy from heart disease is considered pathological and is mostly likely maladaptive even from its onset. Although such hypertrophy may be compensatory at the beginning, nearly all agree that progressive hypertrophy contributes to not only adverse remodeling of heart failure but also arrhythmogenesis. Physiological hypertrophy as a result of exercise and exertion, in contrast, is benign, except perhaps in extreme form of ventricular enlargement that fails to regress after cessation of exercise.

Existing therapies to treat patients with cardiac hypertrophy include normalizing the underlying hypertension, preventing or blocking neurohormones with beta-adrenergic or angiotensin receptor antagonists or converting enzyme inhibitor, or enhancing the relaxation of hypertrophic heart muscle with non-dihydropyridine calcium channel blockers or even disopyrimide when HOCM is present. At the Calhoun Cardiovascular Center, we have a well-developed program to treat and target cardiac hypertrophy from all forms of heart disease. Further, research scientists and clinicians here are actively studying new therapies to reduce cardiac hypertrophy and failure. This has been motivated by the fact that effective method is lacking in reducing "pathological" hypertrophy.

In the current issue of the Newsletter, Dr. James Menzoian, who co-directs the Diabetic Heart and Vascular Section of the Calhoun Center with Dr. David Hager, writes on peripheral vascular disease as a complication of long-standing diabetes. Dr. Peter Schulman, director of the cardiology fellowship program and a senior non-invasive cardiologist at the Calhoun Center, writes on evaluation and treatment of medical patients undergoing non-cardiac surgery. We are also introducing a new feature of the Newsletter, presentation of an interesting cardiovascular case treated here. We are fortunate to have Dr. Arnie Katz, emeritus Chief of Cardiology at UConn School of Medicine, join us as co-editor of the Newsletter.

We welcome Dr. Michael Fucci as a new faculty member of the Calhoun Cardiovascular Center. Dr. Fucci was a chief medical resident and has completed his three years of cardiology fellowship at UConn School of Medicine. Dr. Fucci is a non-invasive cardiologist and an expert on echocardiograph imaging and nuclear cardiology. The overall program at the CCC continues to grow. The clinical activities are getting more robust both qualitatively and quantitatively. Our Echocardiography Laboratory was accredited by the InterSocietal Commission on the Accreditation of Laboratories (ISCAL). This is a worthy recognition and is a credit to the efforts of Dr. David Silverman, Director of Echocardiography Laboratory, Ms. Linda Manzelli, faculty practice administrator, Kim Sheriff, lead sonographer, and Bobby Bowman and JeanAnne Campbell of the Echo Laboratory.

The construction of a new state-of-the-art Electrophysiology (EP) Laboratory continues and is expected to be operational during the summer of 2006. This will enable Dr. Stoenescu, our new Director of the Clinical Electrophysiology Laboratory, to perform implantation of defibrillators and biventricular pacemakers, and to carry out diagnostic electrophysiological study and advanced ablations. The construction of a new second Cardiac Catheterization/Interventional Laboratory will begin after the completion of the EP Laboratory. This new second Cardiac Cath Lab will allow Dr. Azrin and staff to continue the tradition of excellence in delivering cardiac intervention to patients with both elective and urgent needs. At the CCC, we have already been performing emergency angioplasty and stenting to stop heart attack and minimize the damage to heart muscle for the last ten years. We continue to be ready 24-7 and 365 and do whatever it takes to help our patients. Carole and Ray Neag kindly donated a piece of life-saving equipment for the new second Cardiac Catheterization Laboratory. We are thankful for their generosity.

We are grateful to Dr. Deckers for his continued support of the cardio-vascular program here. We look forward to the Jim Calhoun Celebrity Classic Tournament and Events in August, 2006 and are grateful to Pat and Jim Calhoun for their generosity and continued support.

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

In order to assess the risk of surgery, it is recommended that patients who will undergo major surgery be evaluated by a qualified internist or family medicine specialist, and often by a cardiologist. The term "surgical clearance" is often used, but most cardiologists eschew that phrase because we believe that only a surgeon – after determining the risks and benefits and a frank discussion with the patient – can "clear" a patient for surgery.

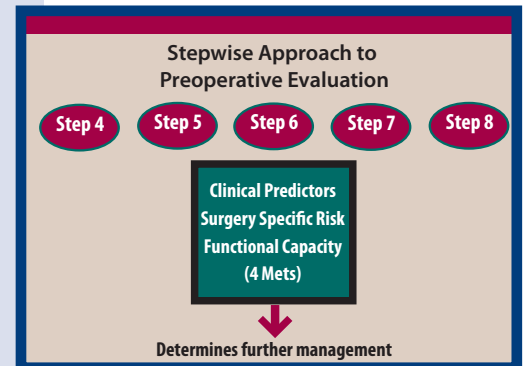
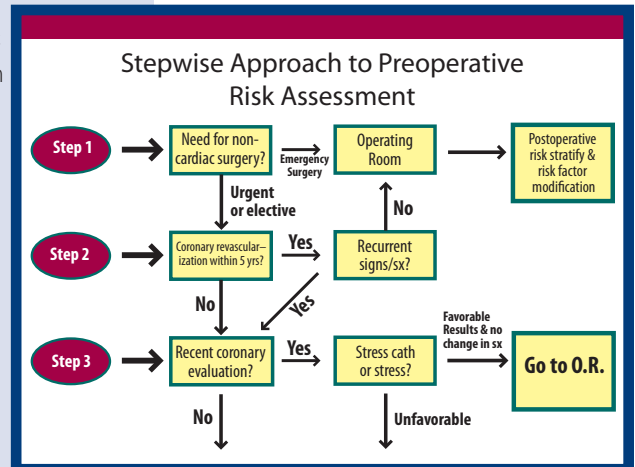
Surgical Risk

A major role of the cardiologist in the preoperative evaluation is to help determine the risk of a cardiac complication and to suggest ways to minimize this risk. It's then up to the surgeon whether to proceed or not. A number of studies have examined the risk to the cardiac patient who is about to undergo major non-cardiac surgery.

To determine the risk to the patient, we look at three factors: the patient's clinical status, his or her functional capacity, and the risk inherent in the type of surgery. Clinical factors involve chiefly the cardiac and other medical conditions; for example, a patient with an acute or recent myocardial infarction, worsening heart failure, or a dangerous arrhythmia has a substantially elevated risk of a cardiac complication. The functional capacity is the patient's ability to perform certain activities, such as climbing stairs. If the patient can jog up two flights of stairs with little difficulty, this level of activity reduces the impact of any cardiac diagnosis or other abnormality. The type of surgery is self-explanatory; clearly, the risk of having a small mole removed from the leg is far less than having a lung removed.

Additional assessment of clinical risks includes cardiac testing. For example, if the patient has had a stress test or a cardiac catheterization in the past two years and the result was good, and if no new cardiac problems have occurred, then additional testing is generally not required before the patient undergoes surgery. If the patient had an angioplasty or bypass operation in the past five years and no new problems have occurred, then the patient is considered to have an acceptable risk of surgery.

Exercise stress tests that quantify functional capacity can predict the risk of surgery. In some instances, particularly when a non-cardiac condition – e.g. a foot problem – prevents us from determining the patient's baseline functional capacity, non-exercise cardiac stress testing is indicated for the preoperative patient. The most commonly used tests used in these situations include a dipyridamole (Persantine®) nuclear stress test or a dobutamine-echo stress test. With the nuclear stress test, the patient is given a radioactive tracer that binds in the heart muscle in proportion to resting blood flow, which allows the perfusion of each region of the heart to be imaged by determining the distribution of the radioactive tracer. A second tracer dose is given after the patient receives an intravenous infusion of a potent coronary vasodilator (the "stress"). The resting and the post-stress nuclear images are compared to differentiate normal from ischemic or infarcted heart muscle.



A Report of the American College of Cardiology/American Heart Association Takes Force on Practice Guidelines.

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Significant perfusion abnormalities are associated with an elevated risk of surgery.

Another noninvasive test that can be done in patients who cannot walk on a treadmill is a dobutamine stress echo. This test involves the infusion of dobutamine, an adrenaline-like compound that elevates the heart rate and blood pressure. A baseline and post dobutamine echo can determine left ventricular function at rest and after stress, and can also help predict the risks associated with a surgical procedure.

Perioperative Beta Blockers

If a patient requires surgery to prolong life or substantially improve lifestyle, and if the patient is deemed to have a high risk of a cardiac complication, then a perioperative beta blocker therapy can substantially reduce the risk of a cardiac complication. Studies with several beta blockers,

including atenolol, bisoprolol, metoprolol, esmolol, have shown that the risk of coronary ischemia, myocardial infarction or death can be substantially reduced by aggressive treatment beginning before the operation.

In cardiac patients or high risk patients who are scheduled to undergo non-cardiac surgery, a beta blocker is generally recommended beginning one or two weeks prior to surgery. If the patient is already taking a beta blocker with an appropriate response, that drug should be continued and given on the morning of surgery with a sip of water. If the patient is currently not taking a beta blocker, such therapy is best started one or two weeks prior to surgery. Alternatively, a rapid-acting beta blocker can be given intravenously in the pre-operative anesthesia holding area. In any event, this therapy should be continued throughout the perioperative period because, as described above,

the second period of greatest risk to the patient occurs three days after the surgical procedure.

Summary

Hundreds of thousands of non-cardiac surgical procedures are performed yearly in the United States. Because of the high prevalence of heart disease, many of these surgical procedures are performed on cardiac patients. Cardiologists are frequently called upon to aid the surgeon in determining the patient's risk of surgery and to help minimize that risk. Cardiologists utilize a risk evaluation plan that encompasses the clinical characteristics of the patient, the level of the patient's functioning, and the specific risk of the surgery. If patients are at a high risk of a cardiac complication during the perioperative period, beta blockers can substantially reduce that risk.

Insights from Recent Medical Articles

Author: Arnold M. Katz, M.D.

Differences between the "physiological" hypertrophy seen in the hearts of athletes and the "pathological" hypertrophy that causes cardiac enlargement in patients with heart failure have been recognized for many decades. Most important is that pathological hypertrophy shortens life expectancy whereas exercise-induced physiological hypertrophy has no obvious adverse effects and may even prolong survival.

Studies carried out in animals have demonstrated several molecular differences between these two types of hypertrophy (1). Most important is that chronic overload, which occurs in most patients with heart failure, causes re-expression of genes that predominate in fetal life, whereas the exercise-induced hypertrophy is accompanied by increased expression of adult genes. Although reversion to the fetal gene program might not seem to be such a bad thing, these changes weaken the heart muscle and so worsen symptoms in patients with heart failure.

Molecular changes that accompany reversion to the fetal gene program have also been shown to be a major cause of sudden cardiac death in these patients. For this reason, modification of this growth response could have a beneficial effect in heart failure.

A recent study using transgenic mice provides clues regarding the way that these two types of hypertrophy are initiated (2). It turns out that the duration of the initiating stress – which is much shorter in exercise-induced physiological hypertrophy than in pathological hypertrophy – influences the extent of the heart's enlargement but not the molecular characteristics of these two types of cardiac enlargement. Instead, the key to understanding these different types of hypertrophy lies in the signaling systems that cause the heart to enlarge. By adding to a substantial body of evidence regarding hypertrophic signaling, this study points to new ways that could to improve prognosis in patients with heart disease.

1. Scheuer J, Buttrick P. The cardiac hypertrophic responses to pathologic and physiologic loads. *Circulation*. 1985;75(1 Pt 2):163-168.

2. Perrino C, Naga Prasad SV, Mao L, Noma T, Yan Z, Kim H-S, Smithies O, Rockman HA. Intermittent pressure overload triggers hypertrophy-independent cardiac dysfunction and vascular rarefaction. *J Clin Invest* 2006;116:1547-1560.

DIABETIC PERIPHERAL ARTERIAL DISEASE (PAD)

Author: James O. Menzoian, M.D.

Professor of Surgery

Co-Director, Collaborative Center for Clinical Care Improvement (C4I)

University of Connecticut Health Center



James O. Menzoian, M.D.

The development of atherosclerotic peripheral arterial disease (PAD) in our society is a very pressing problem because PAD is so prevalent. PAD currently affects 12 million U.S. adults, and that number can be expected to increase dramatically by the year 2020. There are well-defined high-risk groups in whom the disease is likely to develop and progress.

Risk Factors for PAD

The risk of developing atherosclerosis increases with:

- Advancing age
- Smoking
- African-American race
- Diabetes
- Poor renal function
- High blood pressure
- Dyslipidemia

Other potential risk factors for PAD include elevations in:

- C-reactive protein
- Fibrinogen
- Homocysteine
- Apolipoprotein B
- Lipoprotein (a)
- Plasma viscosity

There appears to be an inverse relationship between alcohol consumption and PAD.

Symptoms of PAD

The most common symptom of PAD is intermittent claudication, defined as exercise-induced pain, cramping or aching in the calves, thighs or buttocks, which is relieved with rest. A more extreme manifestation of PAD includes rest pain, tissue loss and gangrene. These manifestations of PAD, which can lead to loss of the limb, are termed "critical limb ischemia" (CLI).

Diabetes and PAD

The well known association between diabetes and atherosclerosis is a very important consideration in the treatment of patients with diabetes. Diabetes, which affects 7% of the U.S. population (approximately 20.8 million people), is accompanied by a significant increase in cardiovascular diseases; the risk of coronary artery disease is 2-4 fold greater in individuals with diabetes, and the likelihood of developing PAD is 4 fold greater in diabetics. As many as 80% of deaths in diabetics are due to cardiovascular disease, and 50% of the amputations done in the U.S., other than amputations done for trauma, are performed in patients with diabetes.

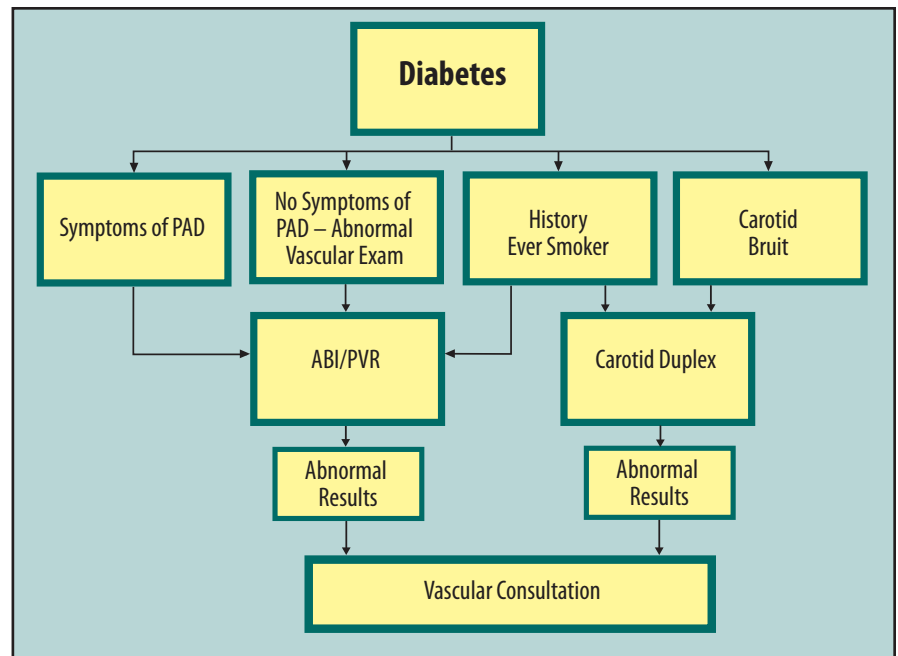
The correlation between diabetes and PAD is strong; 10% of diabetics have PAD. Ten years after the diagnosis of diabetes, 15% of these patients will have developed PAD. This number increases to 45%, 20 years after the diagnosis of diabetes is made. It must be pointed out that many people with PAD can be asymptomatic and some may never have any clinical problems. Data from the Framingham Heart Study show that 20% of patients with symptomatic PAD have diabetes, but this is probably an underestimate of the true prevalence because many people with PAD are asymptomatic.

Individuals without diabetes can also develop PAD. Although there is no difference in the type of atherosclerosis that diabetics develop when compared with non-diabetics, atherosclerosis progresses more rapidly in diabetics and has a unique pattern and location with a propensity to develop in the tibial arteries. The reason for this unique anatomical location is not known.

Significance of PAD

PAD is significant because 27% of these patients demonstrate progression of symptoms and possible limb loss. A striking number of cardiovascular events occur in patients with PAD, with 20% sustaining MI or stroke; 30% die over a 5-year period. For those patients with CLI, the outcomes are even worse. The effect of diabetes on the natural history of PAD has not been studied longitudinally, but prospective clinical trials have shown that the cardiovascular event rates in patients with PAD and diabetes are higher than in non-diabetic patients.

Evaluation of individuals to determine the presence of PAD is important because these patients are at high risk of subsequent MI and stroke. It is also essential to identify and treat symptoms of PAD that could result in functional disability and limb loss. This is especially important in diabetics because these patients often have associated peripheral diabetic neuropathy with impaired sensation, which can blunt the usual symptoms of claudication and rest pain. It is also important to note that patients with diabetes and PAD are more likely to develop sudden ischemia from arterial thrombosis. If patients with sub-clinical disease can be identified and preventive measures instituted, acute limb-threatening ischemia may be prevented.



PAD Diagnosis

The diagnosis of PAD in individuals with diabetes is important and should include a clinical evaluation with a history and physical exam. Other causes of exercise-induced extremity pain, such as spinal stenosis and a variety of popliteal artery entrapment syndromes, must be excluded. The feet should be evaluated for pulses, hair loss, dependent rubor (abnormal redness), cool skin, and skin ulcers at points of potential pressure and between the toes. The dorsalis pedis pulse is absent in 8% of normal individuals and the posterior tibial pulse is absent in 2%.

Non-invasive evaluation for PAD is very helpful and can be accomplished by measurement of the ankle/brachial index (ABI). Using a Doppler, the ankle blood pressure at either the dorsalis pedis artery or the posterior tibial artery can be compared to the brachial artery pressure. A normal ABI is in the range

of 0.91 to 1.0, while patients with intermittent claudication have ABI measurements around 0.5, and the ABI in patients with CLI is often 0.4 or less. After adjustment for the usual cardiovascular risk factors, an ABI of < 0.9 has been shown to be an independent predictor of cardiovascular risk that can help identify patients at risk for future cardiovascular events. A pulse volume recording (PVR) is also helpful, especially in diabetics in whom calcified tibial arteries causes a falsely elevated ABI. The PVR provides a record of blood flow at various levels in the leg that, along with the ABI, gives valuable information. Some patients with clinical symptoms, a normal pulse exam, and a normal ABI require an exercise treadmill test to unmask underlying PAD. In these patients, a decrease in the ABI after a period of exercise on the treadmill can reveal PAD.

Recommendations for Patients with Diabetes

Because of the increased likelihood of patients with diabetes developing or having occult PAD we recommend the following:

- A complete history and physical exam, specifically looking for signs and symptoms of PAD. This should include a pulse exam at the carotid, femoral, popliteal, dorsalis pedis and posterior tibial arteries, and listening for carotid or femoral bruits.
- In patients with diminished or absent pulses, an ABI and PVR to document the magnitude of the PAD.
- In patients with a carotid bruit, a carotid duplex exam to evaluate the possibility of significant carotid atherosclerotic occlusive disease.

An Interesting Case

History. Mr. M, an 86 year old retired teacher, presented with a 3 week history of dyspnea, ankle edema, and exertional chest tightness. IV furosemide given a few days earlier provided only transient relief. Past history included mild asthma, paroxysmal atrial fibrillation, hypercholesterolemia, and hypertension. There were no known allergies, history of tobacco or alcohol use, or family history of heart disease or sudden death. Medications included metoprolol succinate 25 mg/d, terazosin 10 mg/d, lovastatin 20 mg/d, furosemide 20 m/d, amiodarone 200 mg/d, aspirin 81 mg/d, warfarin, albuterol, and advair.

Physical exam. T: 99° F, HR: 90/min (irregular), BP 120/54 mmHg, R: 16/min, SaO₂ on ambient air: 97%.

Venous pressure: normal. Chest: clear. Cardiac Exam: Quiet apical impulse, S1 S2 distant, no rubs or murmurs. Abdomen: Soft, no tenderness or organomegaly, normal bowel sounds. Extremities: 1+ dependent edema, symmetrical 1+ pulses. Neurological: no focal abnormalities.

Laboratory Data. Hgb; 11.8, WBC: 4,000, Hct: 34.8, Plt: 96,000 ESR: 10, electrolytes normal, BUN: 24, Cr: 1.7. Total protein: 5.2 (albumin: 3.1, SPEP: normal). CK: 85 (MB: 4). Troponin: 0.07

EKG. Atrial fibrillation, low voltage. Chest X-ray. Enlarged heart, mild pulmonary vascular congestion. Persantine® stress test with myocardial perfusion imaging. No evidence of ischemia. Pulmonary function. Mild reversible obstructive disease, slightly reduced DLCO.

Echocardiogram. Large circumferential pericardial effusion without evidence of tamponade. Concentric LVH with hyperdynamic function and a “speckled” pattern.

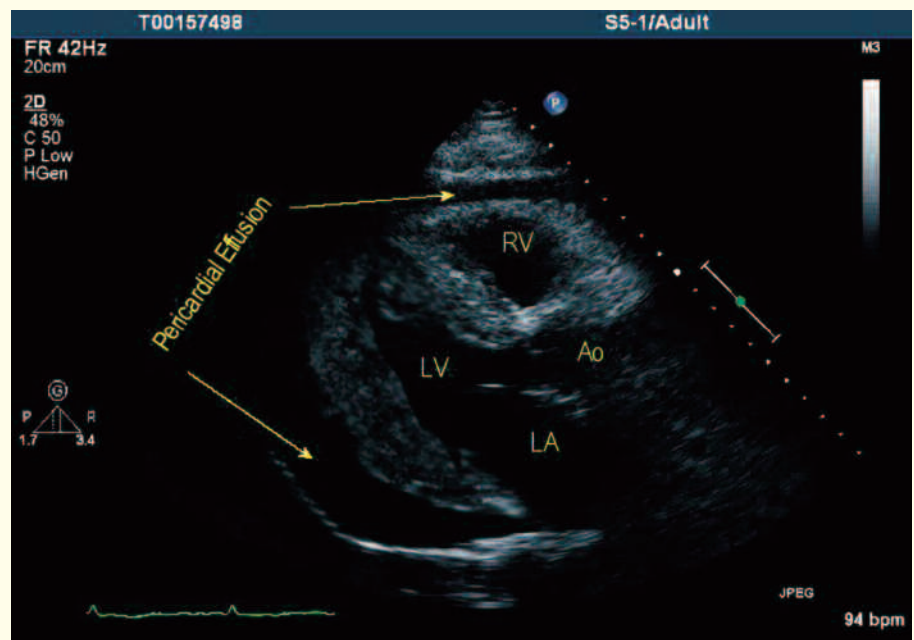
Cardiac Catheterization. LVEDP: 21 mmHg, PCW: 27 mmHg, RAP: 16 mmHg, CO: 3.9 l/min.

Hospital Course and Follow-up. After removal of 1100 cc cloudy yellow pericardial fluid, RAP fell to 6 mmHg and CO increased to 5.1 l/min but PCWP remained elevated at 20 mmHg. There was no step-up in oxygen saturation. Pericardial fluid: glucose: 100, protein: 4.1, LDH: 213, WBC: 1855 (100% lymphocytes), RBC: 410, no malignant cells, negative cultures. Abdominal fat pad biopsy: negative Congo red and thioflavine stains. The patient remained stable after the

pericardiocentesis and elective cardioversion. He remained in sinus rhythm, subsequent echocardiograms did not show re-accumulation of the pericardial fluid, and his functional status returned to baseline.

Diagnosis: Pericarditis, presumably viral in etiology, was associated with hemodynamically significant pericardial effusion. The elevated PCWP after pericardiocentesis suggests the presence of diastolic heart failure secondary to long-standing hypertension; an infiltrative myocardial process such as that caused by amyloidosis, however, cannot be ruled out.

George Kantis, M.D.
Cardiology Fellow, University of Connecticut School of Medicine



Studies and Awards

selected examples

- 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 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 aldosterone 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 natreacor 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. Liang was inducted into the Association of University Cardiologists.
- Dr. White was elected as a new member of the American Society of Hypertension Board of Directors.

Noteworthy Events

- Cardiovascular Grand Rounds (8-9 am in Link Room):
- Dr. Scott Manaker, University of Pennsylvania, Update on Cardiovascular Documentation and Coding, Sept 6th, 2006.
- Dr. Mark Taubman, Strong Memorial Hospital, title TBA, Oct. 4th, 2006.
- Dr. Robert Wilensky, University of Pennsylvania, title TBA, Oct. 18th, 2006
- 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 Liang
- Dr. Peter Schulman
- Dr. David Silverman
- Dr. Matt Stoenescu
- Ms. Gabriella Smith, PA
- Ms. Marybeth Barry, APRN
- Ms. Jill Desjardins, APRN

- Hypertension Specialists (679-3343)
- Dr. William White
- Dr. George Mansoor
- 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. Joan Long, APRN

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. 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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