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Edition 5

To Our Readers

THESE CONTINUE TO BE exciting times in the field of medicine. This is particularly true in the specialties of cardiology and endocrinology, where research findings and newer therapies frequently affect our evaluation and treatment algorithms. In addition, accepted standard therapies are being re-evaluated in regards to their optimal indications and use.

In this year's fall issue of the Oklahoma Heart magazine, Dr. Ivanoff highlights the tremendous benefits being derived from External Counterpulsation Therapy (ECP) in patients with end stage coronary artery disease. Dr. Martin presents a pictorial review of the tremendous value of cardiac MRI and cardiovascular MRA imaging modalities, which provide impressive images and information without the risks of radiation or contrast agents.

The utility of pacemakers and their re-evaluation as to what is optimal pacemaker therapy is presented by Dr. Sandler. Dr. Johnsen reminds us that cardiac rehabilitation, although not new, remains a very effective, but unfortunately, underutilized therapy.

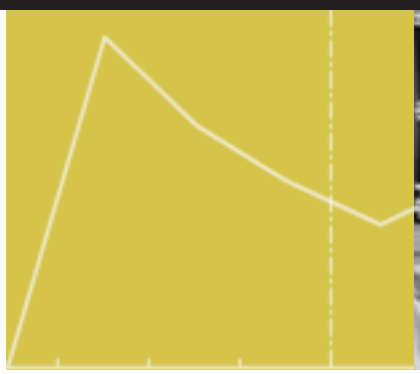
Dr. Aspenson, from our Division of Endocrinology, provides a particularly interesting review of the story of calcium and vitamin D, which may affect not only how you treat your patients, but also how you treat yourself.

As always, the Research Corner provides information on the newest therapies being investigated at Oklahoma Heart Institute.

We hope you enjoy these articles and welcome any comments or suggestions in regard to the magazine content.

Sincerely,
Wayne N. Leimbach, Jr., MD

by John G. Ivanoff, MD, FACC, FSCAI



External Counterpulsation (ECP) In The Treatment of Angina

Why Is It Needed?

Angina pectoris continues to affect patients' lives and the economies of modern societies, despite major advances in medical therapy, interventional cardiology and surgical revascularization.

Many patients are still limited in their daily activities despite maximal medical therapy and may have already undergone, or may not be a candidate for, interventional or surgical revascularization. A good example would be a 65-year-old diabetic, limited by angina, on therapy that includes a long acting nitrate, beta-blocker and calcium channel blocker with previous coronary bypass surgery. Repeat coronary angiography reveals diffuse disease and occluded grafts with small vessel disease that is too severe for redo-bypass surgery.

Another good candidate for ECP is a 56-year-old with an occluded left circumflex that cannot be recanalized by intervention, but who continues to have angina, despite maximal medical management. The risk of bypass surgery for single vessel left circumflex disease outweighs the benefit, but the patient is still limited by his disease. Both of the above patients are excellent candidates for ECP.

What Is ECP?

ECP, in a simple view, could be thought of as an external intra-aortic balloon pump (IABP) that looks like MAST (Military Anti-Shock Trousers) trousers.

A series of pneumatic cuffs cover the lower abdomen down to the lower leg. The sequential inflation and deflation of the cuffs are timed to the cardiac cycle with an ECG monitor. The timing and pressures can be manipulated by the technologist operating the ECP console (Figure 1).

At the start of diastole, the cuffs inflate sequentially, starting at the lower leg and progressing to the lower abdomen. This increases coronary perfusion pressure without increasing cardiac work, much like an IABP. Venous return to the right heart is also augmented in this phase. The onset of systole triggers a rapid deflation of the cuffs lowering diastolic blood pressure and decreasing afterload on the heart, again much like an IABP. The increased venous return, along with a fall in afterload, results in improved cardiac output with a reduction in myocardial oxygen demand. The treatment program con-

Patients experience fewer episodes of angina with less medication and an improved exercise tolerance following ECP treatment.

Figure 1. A series of pneumatic cuffs cover the lower abdomen down to the lower leg. The sequential inflation and deflation of the cuffs are timed to the cardiac cycle with an ECG monitor.

sists of 35 daily one-hour sessions, usually given five days per week for seven weeks.

How Does It Work?

It is obvious from the preceding description of ECP why a patient would feel better during the treatment session, but it is not so clear how that benefit is maintained months to years after the treatment. There is no conclusive answer as to why it works, but there are a few theories.

The most widely believed hypothesis is that ECP increases the development of collaterals to the ischemic areas. Investigators have measured an increase in the levels of the angiogenic factor, vascular endothelial growth factor (VEGF) after a single ECP session, which supports this theory (Fig. 2)(1).

Another hypothesis is that ECP improves endothelial function. Beneficial changes in the levels of the neurohumoral agents endothelin and nitric oxide have been measured with ECP treatments. ECP decreases plasma levels of the vasoconstrictor endothelin and increases the vasodilator nitric oxide (Fig. 3)(2). Beneficial changes in other neurohumoral agents, such as atrial natriuretic factor (ANP) and brain natriuretic factor (BNP), have also been demonstrated with ECP treatment.

Expected Results After ECP

Patients experience fewer episodes of angina with less medication and an improved exercise tolerance following ECP treatment. The randomized multicenter study of enhanced external counterpulsation (MUST-EECP) Trial published in 1999 demonstrated a statistically significant increase in exercise time until ST-segment depression, following ECP treatment over placebo. An improvement in total exercise duration in the ECP group was observed, but this was not statistically significant (Fig. 4)(3). More recent studies have shown improved exercise duration as well as improvement in myocardial perfusion scans following ECP (Fig. 5)(4).

Figure 2

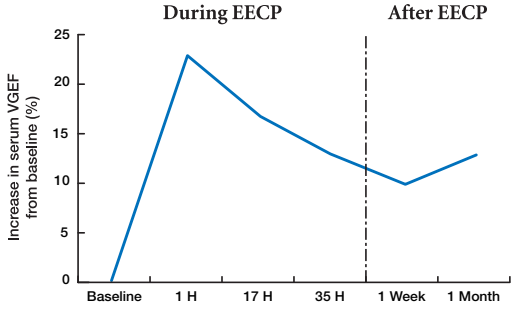


Figure 3

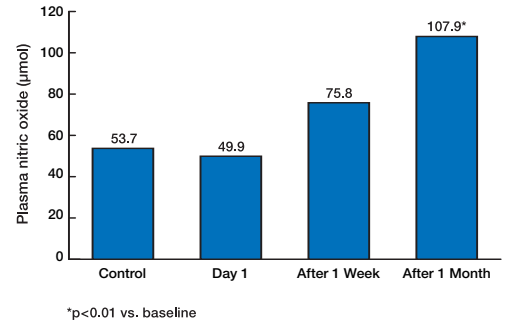
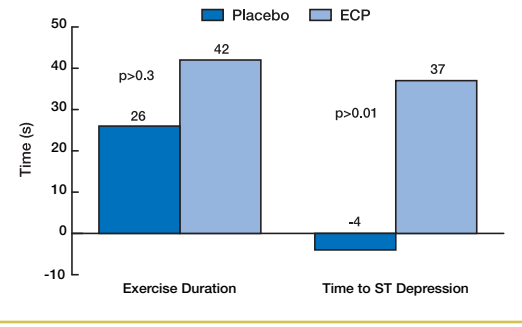


Figure 4



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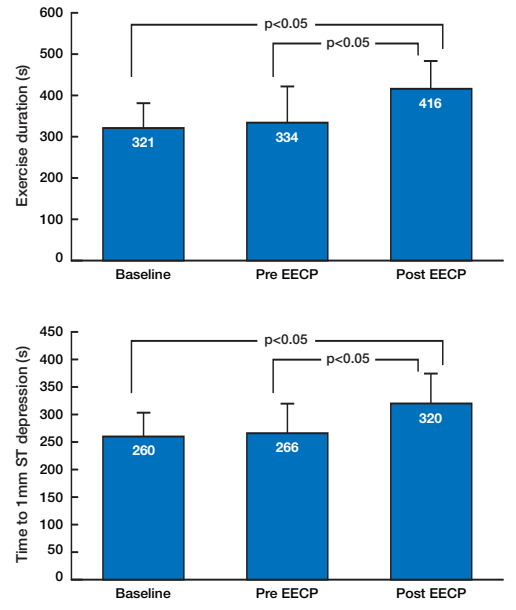

Results of the prospective evaluation of EECP in congestive heart failure (PEECH) trial were presented earlier this year at the American College of Cardiology Scientific Sessions. ECP treatment of heart failure patients improved quality of life, increased exercise duration at six months post treatment and improved the patient's New York Association Heart Failure Classification.

Conclusion

ECP is a safe and effective treatment for angina patients who may not be candidates for interventional or surgical revascularization. The results of the PEECH trial are expected to expand the indication for ECP to congestive heart failure patients with left ventricular dysfunction.

(Dr. Ivanoff specializes in interventional cardiology, including cardiac catheterization, coronary angioplasty and related interventional procedures such as stents, atherectomy and direct PTCA for acute myocardial infarction.)

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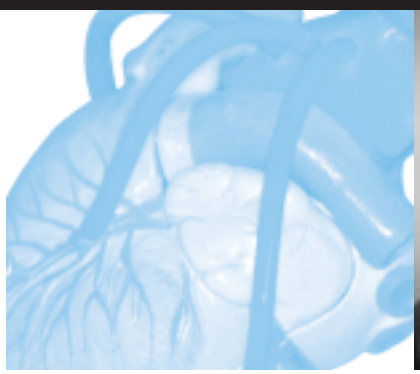
by David A. Sandler, MD



When Less is More:
The Detrimental Effects of Right Ventricular Pacing

In 1952, Dr. Paul M. Zoll delivered energy through wires attached externally to the chest of a patient with complete heart block. The patient's heart responded and was electrocardiographically recorded. Since this early experience, the field of cardiac pacing has expanded dramatically. Current pacemakers are inserted transvenously and are completely self-contained. Batteries can frequently last over a decade.

With the expanding technology, we have also begun to understand the limitations of pacing. Over the past several years, there have been numerous reports describing the potentially hazardous effects of right ventricular pacing. Since the vast majority of patients undergoing pacemaker implantation today have at least intermittent intact atrio-ventricular (AV) conduction, it is imperative that we understand how to avoid these deleterious effects.

The MOST study investigated the proposed benefits of dual chamber (DDD) pacing as compared to simpler single-chamber ventricular (VVI) pacing for patients with sinus bradycardia. Although atrial fibrillation was reduced with DDD pacing, no mortality benefit was seen. The results of this (and other similar studies) were disappointing, given the long-held belief that maintaining AV synchrony is vitally important. To understand this paradox, a sub-study investigated the effects of ventricular pacing. The sub-study demonstrated a clear association between the amount of ventricular pacing and the relative risks of both atrial fibrillation and heart failure hospitalization (figures 1 and 2). Thus, the potential benefits of maintaining AV synchrony with DDD pacing are counteracted by the presence of ventricular pacing.

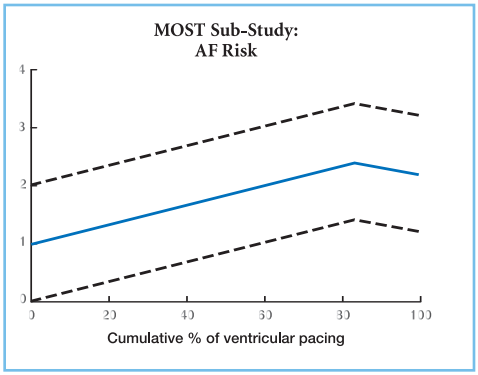


Figure 1. Relative risk of atrial fibrillation by increasing cumulative ventricular pacing in DDDR mode. (Adapted from Sweeney MO et al Circulation. 2003;107:2932-2937)

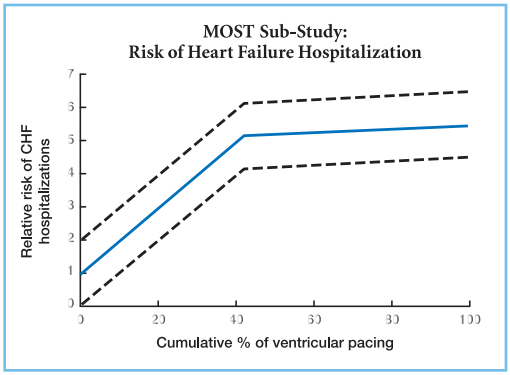


Figure 2. Relative risk of heart failure hospitalization by increasing cumulative ventricular pacing in DDDR mode. (Adapted from Sweeney MO et al Circulation. 2003;107:2932-2937)

The DAVID Study evaluated patients with reduced left ventricular function undergoing insertion of an implantable cardioverter/defibrillation (ICD). In these patients, right ventricular pacing led to increased heart failure hospitalization and death as early as six months. Clearly, patients with reduced heart function are extremely vulnerable to the ill effects of right ventricular pacing.

The similarity between the MOST and DAVID studies is that they included patients without an indication for ventricular pacing. In light of the unfavorable results, we can now view *ventricular* pacing in these patients as an unnecessary side effect of our therapy.

There are many explanations for these outcomes. Normally, activation of the ventricles occurs through specialized conduction fibers allowing for near-simultaneous activation of the septum, right ventricle and left ventricle. The fundamental problem with right ventricular pacing is the abandonment of this efficient network leading to inefficient activation across ventricular myocardium. A left-bundle branch block pattern then ensues.

The pattern of ventricular activation

initiated from the right ventricular apex has numerous effects. Most notably, right ventricular pacing causes dyssyncronous left-ventricular activation, including dyssyncronous activation of the mitral valve apparatus. This leads to an immediate reduction in left ventricular function and increased mitral regurgitation. With sustained pacing, the left ventricular function continues to decline and the left atrium dilates.

What is even more concerning is that left ventricular dysfunction persists, even when intrinsic conduction has been restored. This implies that, in addition to the anatomic and physiologic changes we are able to measure, a darker, more sinister effect of pacing may be occurring on a cellular level. Animal studies have shown that pacing leads to cellular disarray, changes in ion-channel expression and down-regulation of gap junction proteins. These effects would further undermine normal activation of the heart and continue the downward spiral of left ventricular function.

There are many strategies to avoid the deleterious effects of right ventricular apical pacing. In most cases, adjusting the delay between atrial and ventricular activation of the pacemaker will allow for normal, intrinsic activation. Furthermore, pacemaker manufacturers have developed pacing modes, which minimize ventricular pacing in sinus rhythm, while protecting the patient from bradycardia during AV block or atrial fibrillation.

In patients with unavoidable ventricular pacing due to AV block, studies are underway examining the long-term effects of alternative sites for pacing. These include sites in the right ventricle (e.g. outflow-tract and septum) as well as the left ventricle (for prophylactic cardiac resynchronization therapy).

Although cardiac pacing has progressed dramatically since the early days of Dr. Zoll and has improved the lives of millions, there is no doubt that this therapy still has limitations. As we learn to avoid the detrimental effects of forced right ventricular pacing, we will be able to provide the beneficial effects of this therapy without the morbidity.

(Dr. Sandler is a cardiology subspecialist in electrophysiology.)

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by D. Erik Aspenson, MD, FACE, FACP

Vitamin D: *An Epidemic Hormone Deficiency*

Most physicians have cared for this patient: middle aged, sedentary, possibly a smoker, office worker focused on staying healthy, but resistant to taking a lot of medicines. Instead, they take flaxseed, Echinacea, Coenzyme Q10, vitamin B complex, chromium picolinate, glucosamine chondroitin sulfate, vitamin C and E, and garlic. What they don't take – despite proven benefits greater than all of the aforementioned products, despite intriguing potential benefits in areas unrelated to calcium metabolism (such as solid tumors), and despite their mom's hip fracture – is vitamin D (with or without calcium).

Vitamin D is appropriately considered a hormone. It is stored in the liver, released into the circulation, converted to a more active form, and subsequently exerts its effects at distant sites all over the body via the ubiquitous vitamin D receptor (VDR). Other than fatty fish, vitamin D is found naturally in few foods. The source of 90% of our vitamin D is the conversion of 7-dehydrocholesterol by UV light in the skin to cholecalciferol (D3). A few minutes of casual exposure each day to the arms and face will produce the equivalent of 200 IU of cholecalciferol. In the liver, circulating D3 is converted to the storage form, 25-cholecalciferol (calcidiol), by the 25-hydroxylase enzyme. Circulating calcidiol is converted in the kidney to 1,25-dihydroxy vitamin D (calcitriol). Calcitriol then functions, primarily, to promote calcium absorption in the gut and reclamation in the kidney. In this role, it is 100 times more potent than calcidiol. Other organs also have 1-alpha-hydroxylase and convert calcidiol to calcitriol for local, paracrine like, use.

It does not seem intuitive that a vitamin so readily produced in the skin and supplied in fortified foods like bread, milk and cereal would become deficient. However, since 90% of our supply is from one source (UV light), increased time indoors, darker skin pigmentation, sunscreen use, and winter months produce a high risk for deficiency. Other less common causes of insufficiency include problems with conversion to inactive metabolites (phenytoin therapy, alcohol) or absorption (cystic fibrosis, inflammatory bowel disease, celiac sprue and gastrectomy). Sprue and gastrectomy, while uncommon causes of vitamin D deficiency in general, are the most common causes of clinically apparent osteomalacia in the U.S. today.

A consensus for what defines vitamin D deficiency has been elusive. One definition is the level of vitamin D at which supplementation produces a fall in PTH. Other researchers have defined various cut-points such as 25-OH vitamin D (calcidiol) severely deficient < 20 nmol/L (8ng/mL), insufficient 20-50 nmol/L (8-20 ng/mL), and sufficient > 50 nmol/L (>20ng/mL). The definition I prefer is severe deficiency <20ng/mL and insufficiency <30 ng/mL. The prevalence rates of vitamin D deficiency (by various yardsticks) have been measured in young and old, men and women, sunny climes and northern latitudes, among pain clinic patients, medical inpatients and free-range Floridians.

The numbers are not encouraging. A 2003 study in Minnesota patients complaining of ill-defined pain and weakness found a 100% rate of vitamin D deficiency in non-Caucasians. Rates of vitamin D deficiency noted in other populations include 42% of African-Americans age 15-49, 32% of 18-29 year old white Bostonians in winter months, 48% of 9-11 year old girls in Maine, up to 75% of mother-baby breastfeeding pairs (especially in dark skinned mothers and exclusively breastfed infants), and 42-57% of people over 50 years old at any latitude.

A very concerning finding was found on examination of 1500 community dwelling women being treated for osteoporosis that dramatically illustrated the need for improved physician vigilance. Fifty-two percent of these women were found to be vitamin D insufficient with levels below 30 ng/ml, despite their status as osteoporosis patients. Because almost 1 in 5 women were severely deficient (<20 ng/ml) the mean vitamin D level for the group barely reached "sufficiency" at 30.4 ng/ml. The rate of vitamin D deficiency is increased in the elderly due to diminished production in the skin for a given level of sun exposure and decreased consumption. One study estimated approximately 50% of elderly women consume less than 137 IU/day. Even with good dietary intake, older people confined indoors may have low calcidiol levels.

The consequences of vitamin D deficiency are, perhaps, more widespread than once thought. Certainly it is a painful disorder. The secondary hyperparathyroidism in response to low vitamin D may produce low bone density, especially at cortical bone predominate sites (radius, femoral neck) and osteomalacia. With osteomalacia, low vitamin D, combined with high PTH values, produces a low to low-normal calcium and low serum phosphorous. This diminished calci-

um phosphorous product impairs the mineralization of new collagen matrix, which continues to be deposited. The unmineralized matrix expands with hydration and stretches, or puts pressure upon, the periostium leading to dull aggravating pain and tenderness diffusely in the bones. This bone tenderness (elicited with gentle pressure to the sternum and anterior tibia) should not be confused with the tender points of fibromyalgia syndrome. Hypovitaminosis D also produces muscle weakness, typically proximal, that may be associated with muscle wasting and discomfort with exertion.

The Effects of Repletion/ Supplementation

Osteoporosis: Vitamin D plays a major role in preserving or restoring bone health in osteoporotic or at risk patients. A study of nursing home patients demonstrated that, while 400 IU/day of vitamin D alone improved bone mineral density (BMD) at the femoral neck and decreased PTH 15%, it did not prevent fracture. The addition of calcium to the vitamin D reduced PTH 50% and increased hip BMD 6%, producing a significant decrease in hip and other non-vertebral fractures.

Another trial (n=2578) confirmed the necessity of providing calcium and vitamin D together. The patients received 400 IU/day of vitamin D and consumed on average 870 mg/day of calcium from the diet. There was an improvement in their vitamin D levels, but no fracture reduction. It must be pointed out that the researchers simply may not have used enough vitamin D. For instance, another study of elderly patients, this time given high dose vitamin D at 100,000 IU every four months for 5 years, demonstrated a 30% decrease in any osteoporotic fracture. In a similar vein, a small study of 18 patients with osteopenia or osteoporosis and vitamin D deficiency (<14 ng/ml) found that replacement with 50,000 IU twice weekly for 5 weeks produced an annualized 4-5% increase in BMD at the spine and hips. Finally, a randomized controlled trial of 389 elderly men and women over three years found that 700 IU/day of vitamin D3 and modest (500 mg) calcium supplementation reduced non-vertebral fractures significantly.

One of the main risk factors for osteoporotic fracture is the fall risk. Vitamin D replacement has been shown to improve muscle strength and decrease the risk of falls in the elderly. Similar to its relationship with other diseases, the effect of vitamin D on the disease process is probably a combination of the absolute level of vitamin D and any vitamin D receptor (VDR) polymorphisms.

Calcitriol and calcidiol have activity throughout the body unrelated to calcium/phosphorous homeostasis. Recall that the VDR is found throughout the body. Some non-calcium regulating actions include: inhibition of cellular growth, immune function modulation, inhibition of renin production, and stimulatation of insulin secretion. There is epidemiologic evidence demonstrating that people at higher latitudes, in general, are more prone to vitamin D deficiency and have an increased risk of: prostate, breast, colon, and other solid tumors; multiple sclerosis, hypertension, and cardiovascular disease. While these are mere associations, there is considerable research to suggest a significant relationship between vitamin D and these diseases.

Blood pressure: Vitamin D3 800 IU/day plus 1200mg/day of calcium was compared to calcium supplementation alone for its effect on PTH, vitamin D stores, and blood pressure. After 8 weeks, the calcium plus vitamin D3 group increased serum 25 OHD 72%, decreased serum PTH 17% and impressively, decreased systolic blood pressure (SBP) 9.3%. Eighty-one percent of the calcium plus vitamin D3 subjects lowered SBP more than 5 mmHg.

Cancer: There is substantial evidence that vitamin D has an effect on the differentiation, multiplication, and invasiveness of prostatic cancer cells. Studies on various VDR polymorphisms have been mixed. In a large case controlled study, sun exposure, determined by history and reflectometry, and multiple VDR polymorphisms were compared between 450 cases and 455 controls. Significant reductions in prostate cancer were seen with higher sun exposure and certain high activity alleles of the VDR, but not others. There is some evidence that vitamin D may have its impact on prostate cancer via enhanced transcription of the tumor necrosis factor alpha gene. Also, it appears that the less active, for calcium metabolism, calcidiol form of vitamin D may be more important than calcitriol in the prostate.

Similar to prostate cancer, low vitamin D levels and certain VDR polymorphisms seem to confer an increase risk of breast cancer. In the United Kingdom, a case controlled study found that subjects with a low vitamin D level with or without the bb BsmI VDR genotype had a significantly increased risk of breast cancer.

MS: A prior history of high sun exposure and vitamin D supplementation have been associated with a reduced risk of multiple sclerosis. In addition, lower vitamin D levels have been observed in MS patients during their relapses, suggesting further study needs to be done to determine if vitamin D levels play a role in disease activity.

Vitamin D may also play a role in determining risk for pre-menstrual syndrome. Women in the highest quintile of vitamin D intake had a RR of 0.59 for pre-menstrual syndrome compared with the lowest quintile in a nested case control study within the Nurses' Health Study II.

Replacement: In patients deficient in vitamin D, high dose replacement for 2-3 months is in order to restore liver stockpiles. Simply starting replacement with 1000 IU/day will not be adequate, unless sun exposure also significantly changes, because the body uses about 3000-5000 IU/day. I replete patients with 50,000 units of vitamin D2 three times a week for one month, then weekly for two months. This can be done even in patients with mild hyperparathyroidism as long as calcium is <12mg/dl and hypercalciuria is not already a significant problem. Unfortunately, vitamin D2 (ergocalciferol; available in the U.S.) may be less potent at producing sustained elevations in serum 25-OH vitamin D (25-OHD) than vitamin D3 (cholecalciferol). A study of 50,000 IU doses of each drug, demonstrated the area under the curve for D2 was <30% of that for D3 over 28 days. The relative potency for D3:D2 was calculated at 9.5:1.

The elderly respond as well as younger patients to oral replacement of vitamin D, but due to declining renal function and 1-alpha hydroxylase activity, they require a higher vitamin D level to suppress PTH. Most metabolic bone specialists recommend supplementation with 1000 IU per day for those at risk of deficiency. This can easily be obtained from OTC calcium plus D twice daily, plus a multivitamin. Alternatively monthly therapy with 50,000 units of vitamin D2 is an option. The goal when replacing a deficient patient is a 25 OHD level >30ng/mL.

During your discussions with your aforementioned "pill-averse" patient regarding health maintenance, remind them of the proven benefits of vitamin D and the intriguing results from case control studies. Screen your at-risk patients with a 25 OHD level, and aggressively replace those who are deficient with a short term course of high dose vitamin D2.

(Dr. Aspenson is a subspecialist in Endocrinology, Metabolism and Hypertension.)

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THE DOCTORS OF OKLAHOMA HEART INSTITUTE

Wayne N. Leimbach, Jr., MD, FACC, FSCAI, FCCP, FAHA

Dr. Leimbach is a subspecialist in interventional cardiology, including cardiac catheterization, coronary angioplasty and related interventional procedures such as stents, atherectomy, laser, intravascular ultrasound imaging and direct PTCA for acute myocardial infarction. He is Director of the Cardiac and Interventional Laboratories at Hillcrest Medical Center. Dr. Leimbach is Co-Director of the Lipid and Wellness Clinic at Oklahoma Heart Institute. He is Director of the James D. Harvey Center for Cardiovascular Research at Hillcrest Medical Center, as well as Director of the Oklahoma Heart Research and Education Foundation. He also serves as Clinical Associate Professor of Medicine at the University of Oklahoma College of Medicine – Tulsa. Dr. Leimbach completed a Clinical Cardiology Fellowship and a Research Fellowship at the University of Iowa Hospitals and Clinics. He also completed his Internal Medicine Internship and Residency programs at Iowa, where he was selected Chief Resident in Medicine. He received his medical degree from Northwestern University in Chicago and his Bachelor of Science degree from the University of Michigan.

Board certified in Internal Medicine, Cardiovascular Disease and Interventional Cardiology

Robert C. Sonnenschein, MD, FACC, ASE, RVT

Dr. Sonnenschein specializes in echocardiography and noninvasive peripheral vascular imaging. He is Director of Peripheral Vascular Ultrasound Imaging at Hillcrest Medical Center and Oklahoma Heart Institute and serves as Clinical Associate Professor of Medicine at the University of Oklahoma College of Medicine – Tulsa. He completed his Cardiology Fellowship at the State University of New York Upstate Medical Center in Syracuse, where he also completed his Internal Medicine Internship and Residency programs. Dr. Sonnenschein received his medical degree from Rush Medical College in Chicago and his Bachelor of Arts degree from the University of Pennsylvania.

Board certified in Internal Medicine, Cardiovascular Disease, and Adult Echocardiography
Registered Vascular Technologist

Robert E. Lynch, MD, FACC

Dr. Lynch is a specialist trained in noninvasive and invasive cardiology. He is former Chief of Cardiology at Hillcrest Medical Center, where he also has served as Chief of Medicine and President of the medical staff. Dr. Lynch is Co-Director of the Lipid and Wellness Clinic at Oklahoma Heart Institute and Director of the Executive Health Program. He is also a Clinical Assistant Professor at the University of Oklahoma College of Medicine – Tulsa. He completed his Cardiology Fellowship, as well as his Internal Medicine Internship and Residency, at the University of Oklahoma Health Sciences Center. Dr. Lynch received his medical degree from the University of Oklahoma School of Medicine and his Bachelor of Science degree from the University of Tulsa. Before establishing his practice in Tulsa, he served as Chief of Medicine at the U.S. Army Hospital, Bangkok, Thailand.

Board certified in Internal Medicine and Cardiovascular Disease

James J. Nemec, MD, FACC

Dr. Nemec is a subspecialist in echocardiography, stress echocardiography and nuclear cardiology. He serves as Director of Nuclear Cardiology for Oklahoma Heart Institute. Dr. Nemec has served as Assistant Professor of Internal Medicine, Division of Cardiology, at Creighton University and as Assistant Professor, Department of Radiology, also at Creighton University. He completed his Clinical Cardiology Fellowship at the Cleveland Clinic Foundation and his Internal Medicine Internship and Residency at Creighton University. Dr. Nemec also completed a year of training in pathology at the University of Missouri, Columbia, MO. He received his medical degree from Creighton University, where he also received his Bachelor of Arts degree.

Board certified in Internal Medicine and Cardiovascular Disease

John G. Ivanoff, MD, FACC, FSCAI

Dr. Ivanoff specializes in interventional cardiology, including cardiac catheterization, coronary angioplasty and related interventional procedures such as stents, atherectomy and direct PTCA for acute myocardial infarction. He is Director of the Catheterization Laboratories at SouthCrest Hospital. Dr. Ivanoff serves as Clinical Associate Professor of Medicine at the University of Oklahoma College of Medicine – Tulsa. He has also served as Assistant Professor of Medicine at the Medical College of Pennsylvania, as well as Associate Director of the Coronary Care Unit and Assistant Professor of Medicine at Hahnemann University Hospital, where he also completed his Cardiology Fellowship. He completed his Internal Medicine Internship and Residency at the Medical College of Pennsylvania, where he served as Chief Resident. Dr. Ivanoff also received his medical degree from the Medical College of Pennsylvania. He completed his Masters degree in biochemistry at Columbia University and received his Bachelor of Arts degree from the University of Pennsylvania.

Board certified in Internal Medicine, Cardiovascular Disease and Interventional Cardiology

Gregory D. Johnsen, MD, FACC

Dr. Johnsen is an interventional cardiologist with expertise in cardiac catheterization, angioplasty and related interventional procedures, such as stents and atherectomy. He is Director of Cardiac Rehabilitation at Hillcrest Medical Center and Director of the Hillcrest Exercise and Lifestyle Programs. He completed his Clinical Cardiology Fellowship at the University of Oklahoma – Oklahoma City, where he then finished an extra year of dedicated training in interventional cardiology. He completed his Internal Medicine Internship and Residency training at the University of Oklahoma – Oklahoma City, where he also received his medical degree. Dr. Johnsen received his Bachelor of Science degree from Oklahoma StateUniversity.

Board certified in Internal Medicine, Cardiovascular Disease and Interventional Cardiology

Alan M. Kaneshige, MD, FACC

Dr. Kaneshige is a noninvasive cardiologist with expertise in adult echocardiography, stress echocardiography and transesophageal echocardiography. He is Chief of Cardiology at Hillcrest Medical Center. Dr. Kaneshige is also the Director of the Adolescent and Adult Congenital Heart Clinic at Oklahoma Heart Institute and Director of the Congestive Heart Failure C.A.R.E. Center at Hillcrest Medical Center. Dr. Kaneshige completed his Internal Medicine Internship and Residency at Creighton University School of Medicine, where he also received his medical degree. He received a Bachelor of Science in chemistry at Creighton University. Dr. Kaneshige completed his Clinical Cardiology Fellowship at Creighton, where he also served as Chief

Cardiology Fellow for two years. He completed an additional Cardiac Ultrasound Fellowship at the Mayo Clinic in Rochester, MN. Dr. Kaneshige served as Assistant Professor of Medicine at Creighton University School of Medicine, where he was Director of the Noninvasive Cardiovascular Imaging and Hemodynamic Laboratory.

Board certified in Internal Medicine and Cardiovascular Disease
Board certified in Adult and Transesophogeal Echocardiography

Ernest Pickering, DO, FACOI

Dr. Pickering is a cardiology specialist trained in noninvasive and invasive cardiology with subspecialty expertise in cardiac catheterization and angioplasty. He is Chief of Cardiology at SouthCrest Hospital and past Chief of Cardiology at Tulsa Regional Medical Center. He completed a Cardiovascular Disease Fellowship at Baylor College of Medicine in Houston, TX. Dr. Pickering's Internal Medicine Residency was completed at Oklahoma Osteopathic Hospital in Tulsa. He received his medical degree from Philadelphia College of Osteopathic Medicine and his Bachelor of Science degree from Shelton College, Ringwood, NJ.

Board certified in Internal Medicine and Cardiovascular Disease

James A. Coman, MD, FACC

Dr. Coman is a subspecialist in cardiac electrophysiology, ablation therapy and pacemakers. He is Director of Electrophysiology at Hillcrest Medical Center. Dr. Coman also serves as Clinical Associate Professor of Medicine at the University of Oklahoma College of Medicine – Tulsa. He completed an Electrophysiology Fellowship at the Cleveland Clinic Foundation, where he was Chief Fellow. His Cardiology Fellowship was also performed at the Cleveland Clinic Foundation. Dr. Coman's Internal Medicine Internship and Residency training were completed at the University of Alabama, Birmingham, AL. He received his medical degree from the University of Alabama School of Medicine and his Bachelor of Science degree in biomedical engineering from Vanderbilt University, Nashville, TN.

Board certified in Internal Medicine, Cardiovascular Disease and Electrophysiology

Edward T. Martin, MS, MD, FACC, FACP, FAHA

Dr. Martin is a noninvasive cardiologist with subspecialty expertise in non-invasive imaging. He is Director of Cardiovascular Magnetic Resonance Imaging at Oklahoma Heart Institute, SouthCrest Hospital and Hillcrest Medical Center. Dr. Martin is also Director of Nuclear Cardiology at SouthCrest Hospital. In addition, he is a Clinical Associate Professor of Medicine at the University of Oklahoma College of Medicine – Tulsa. Dr. Martin has specialty training in Nuclear Medicine, as well as additional training dedicated to Cardiovascular Magnetic Resonance Imaging. He completed his Cardiology Fellowship at the University of Alabama. Dr. Martin's Internal Medicine Internship and Residency training were performed at Temple University Hospital in Philadelphia. He received his medical degree from the Medical College of Ohio. Dr. Martin completed his Master of Science degree in mechanical engineering at the University of Cincinnati and his Bachelor of Science degree in physics at Xavier University. Dr. Martin is a founding member of the Society of Cardiovascular Magnetic Resonance and is an editorial board member of the Journal of Cardiovascular Magnetic Resonance.

Board certified in Internal Medicine and Cardiovascular Disease

Roger D. Des Prez, MD, FACC

Dr. Des Prez is a noninvasive cardiologist with subspecialty expertise in echocardiography, nuclear cardiology and transesophageal echocardiography. He is Director of Echocardiography and Peripheral Vascular Ultrasound Imaging at SouthCrest Hospital. Dr. Des Prez received his medical degree and Bachelor of Arts degree from Vanderbilt University. He completed his Residency in Internal Medicine and Pediatrics at University Hospital of Cleveland. Dr. Des Prez practiced for six years as an internist with the Indian Health Services in Gallup, NM. He returned to Vanderbilt University as a member of the Internal Medicine Faculty, at which time he also completed his cardiology training. In addition to noninvasive cardiology, Dr. Des Prez is interested in outcomes research and computers in medicine.

Board certified in Internal Medicine, Pediatrics, Critical Care and Cardiovascular Disease

Board certified in Adult and Transesophageal Echocardiography

Christian S. Hanson, DO, FACE

Dr. Hanson is a specialist in Endocrinology, Metabolism and Hypertension at Oklahoma Heart Institute with expertise in diabetes, lipids and hypertension. He also serves as Clinical Associate Professor of Medicine in the College of Osteopathic Medicine – Oklahoma State University. He completed a Fellowship in Endocrinology, Metabolism and Hypertension at the University of Oklahoma in Oklahoma City. Dr. Hanson's Internal Medicine Residency and Rotating Internship were completed at Tulsa Regional Medical Center. He received his medical degree from Oklahoma State University and his Bachelor of Science degree from Northeastern Oklahoma State University in Tahlequah.

Board certified in Internal Medicine
Board certified in Endocrinology and Metabolic Diseases

Rebecca L. Smith, MD

Dr. Smith is a noninvasive cardiologist with subspecialty expertise in transesophageal echocardiography, intra-operative echocardiography, stress and pharmacological echocardiography and contrast echo-cardiography. She completed an Advanced Cardiac Imaging Fellowship at the Cleveland Clinic Foundation and her Cardiology Fellowship at the University of New Mexico Health Sciences Center, Albuquerque, NM. Dr. Smith's Internal Medicine Internship and Residency training were performed at the University of Arizona Health Sciences Center in Tucson. She received her medical degree from the Medical College of Ohio. Dr. Smith completed her Bachelor of Science degree at Cleveland State University.

Board certified in Internal Medicine

Board eligible in Cardiovascular Disease

Tobie L. Bresloff, MD

Dr. Bresloff is a specialist in Endocrinology, Metabolism and Hypertension, with expertise in diabetes, lipids, hypertension and thyroid diseases. She also serves as Assistant Professor in Clinical Medicine at the University of Oklahoma College of Medicine - Tulsa. She completed an NIH Fellowship in Endocrinology and Metabolism at Vanderbilt University in Nashville, TN. Dr. Bresloff's Internal Medicine Internship and Residency were completed at Sinai Hospital of Detroit, Detroit, MI. She received her medical degree from Wayne State University School of Medicine in Detroit and her Master of Science and Bachelor of Science degrees at the University of Michigan, Ann Arbor, MI.

David A. Sandler, MD

Dr. Sandler is a cardiologist with subspecialty expertise in electrophysiology. He completed his Cardiac Electrophysiology Fellowship and his Cardiovascular Medicine Fellowship at New York University Medical Center, New York, NY. Dr. Sandler's Internal Medicine Internship and Residency were performed at Mount Sinai Medical Center, New York, NY. He earned his medical degree from Georgetown University School of Medicine in Washington, DC. Dr. Sandler received his Bachelor of Arts degree at the University of Pennsylvania in Philadelphia.

Board certified in Internal Medicine and Cardiovascular Disease
Board certified in Cardiac Electrophysiology

Raj H. Chandwaney, MD, FSCAI

Dr. Chandwaney is an interventional cardiologist with expertise in cardiac catheterization, coronary angioplasty and related interventional procedures such as coronary stents, atherectomy, intravascular ultrasound and peripheral vascular interventional procedures. He completed his Clinical Cardiology Fellowship at Northwestern University Medical School in Chicago, IL., where he also completed an Interventional Cardiology Fellowship. Dr. Chandwaney's Internal Medicine Internship and Residency were performed at Baylor College of Medicine in Houston, TX. He received his medical degree from the University of Illinois at Chicago. Dr. Chandwaney completed his Master of Science degree at the University of Illinois at Urbana Champaign, where he also received his Bachelor of Science degree.

Board certified in Internal Medicine and Cardiovascular Disease
Board certified in Interventional Cardiology

D. Erik Aspenson, MD, FACE, FACP

Dr. Aspenson is a subspecialist in Endocrinology, Metabolism and Hypertension at Oklahoma Heart Institute, with expertise in diabetes, lipids, hypertension and thyroid diseases. He completed a Fellowship in Endocrinology at Wilford Hall Medical Center, Lackland AFB, Texas. Dr. Aspenson's Internal Medicine Internship and Residency were completed at David Grant Medical Center, Travis AFB, California where he served as Chief Resident. He received his medical degree from the University of Oklahoma and his Bachelor of Science degree at Oklahoma State University.

Board certified in Internal Medicine
Board certified in Endocrinology and Metabolic Diseases

Frank J. Gaffney, MD

Dr. Gaffney is an invasive and noninvasive cardiologist with subspecialty expertise in transesophageal echocardiography. He completed his Cardiovascular Medicine Fellowship at Scott & White Memorial Hospital in Temple, Texas. Dr. Gaffney completed his Internal Medicine Internship and Residency at Brooke Army Medical Center in San Antonio. He then remained on staff at Scott & White Memorial Hospital for several years, before entering his Fellowship in Cardiovascular Medicine. Dr. Gaffney earned his medical degree from New York Medical College, Valhalla, New York, and he received his Bachelor of Arts degree at Hofstra University in Hempstead, New York.

Board certified in Internal Medicine
Board certified in Cardiovascular Disease

Yunus A. Moosa, MD, FACC, FACP, FSCAI

Dr. Moosa is an interventional cardiologist with expertise in cardiac catheterization, coronary angioplasty and related interventional procedures such as coronary stents, atherectomy, intravascular ultrasound and peripheral vascular interventional procedures. He is Director of Peripheral Vascular Services at SouthCrest Hospital. Dr. Moosa completed his Clinical Cardiology Fellowship at Howard University in Washington, DC, where he also completed his Internal Medicine Internship and Residency. Dr. Moosa received his medical degree from the University of Ibadan, Nigeria. He obtained his Bachelor of Science degree at the University of Durban-Westville, South Africa.

Board certified in Internal Medicine and Cardiovascular Disease
Board certified in Interventional Cardiology

Michael J. Fogli, MD

Dr. Fogli is a subspecialist in magnetic resonance imaging, nuclear cardiology, echocardiography, stress echocardiography and transesophageal echocardiography. He completed a fellowship in Advanced Cardiac Imaging at the University of Texas, Southwestern Medical Center in Dallas, TX. His Cardiology fellowship was also performed there, as were his Internal Medicine Internship and Residency. Dr. Fogli earned his medical degree at the University of California, San Francisco School of Medicine and his Bachelor of Arts degree at the University of California, Berkley.

Board certified in Internal Medicine
Board certified in Cardiovascular Disease

Eric G. Auerbach, MD

Dr. Auerbach is a subspecialist in magnetic resonance imaging, nuclear cardiology, echocardiography, stress echocardiography and transesophageal echocardiography. He completed his Cardiovascular Magnetic Resonance Imaging fellowship at Oklahoma Heart Institute, Tulsa, OK. His Cardiology fellowship was performed at the University of Miami/Jackson Memorial Hospital in Miami, FL. Dr. Auerbach's Internal Medicine Internship and residency were also completed at the University of Miami/Jackson Memorial Hospital in Miami. Prior to that, he performed a Surgery Internship at New York Hospital/Cornell Medical Center, New York, NY. Dr. Auerbach earned his medical degree at the University of Miami School of Medicine, Miami, Florida and his A.B. degree at Princeton University, Princeton, New Jersey.

Board certified in Internal Medicine
Board certified in Cardiovascular Disease

Kelly Flesner-Gurley, MD

Dr. Flesner-Gurley is a subspecialist in Endocrinology, Metabolism and Hypertension at Oklahoma Heart Institute, with expertise in diabetes, lipids, hypertension and thyroid diseases. Prior to joining Oklahoma Heart, she was at St. John Medical Center in Tulsa. She completed her fellowship in Endocrinology at the University of Texas at Galveston. Her Internal Medicine Internship and Residency were completed at the University of Texas in Houston, where she also received her medical degree. She earned her Bachelor of Science degree at Texas A&M University in College Station, TX.

Board certified in Internal Medicine and Endocrinology, Diabetes and Metabolic Diseases

RESEARCH CORNER

by **Wayne N. Leimbach, Jr., MD, FACC, FSCAI, FCCP, FAHA**

To date, lipid-lowering strategies have focused on reducing LDL-C. The effectiveness of LDL cholesterol reduction has been demonstrated in many large clinical trials.

Recently attention has shifted to other potential therapeutic strategies to reduce coronary heart disease. Oklahoma Heart Institute is involved in three major multi-center clinical trials looking at complimentary strategies for correcting lipid and metabolic abnormalities.

One trial looks at the drug Torcetrapib, which is used to raise HDL cholesterol levels (the good cholesterol). Patients are randomized to receive either Atorvastatin 80mg q day and placebo or Atorvastatin 80mg per day plus Torcetrapib.

Another trial looks at the use of an HDL mimetic. The HDL mimetic augments HDL in the removal of LDL cholesterol from the body. It is given as an infusion once a week for 4 weeks. Animal studies suggest it may produce regression of plaque within weeks instead of years.

The third trial (the Stratevarius Trial) uses the new endocannibinoid receptor antagonist rimonabant to produce weight loss and improve several metabolic parameters. The purpose of this study is to see if the progression of atherosclerotic vascular disease can be stopped.

All of these studies use the technique of intravascular ultrasound to measure plaque volume at baseline and at 18 to 24 months. The format of the trials is similar to that of the REVERSAL trial, which showed that aggressive LDL cholesterol lowering with Lipitor 80mg a day was able to halt progression of atherosclerotic plaque in coronary arteries within just 18 months of therapy.

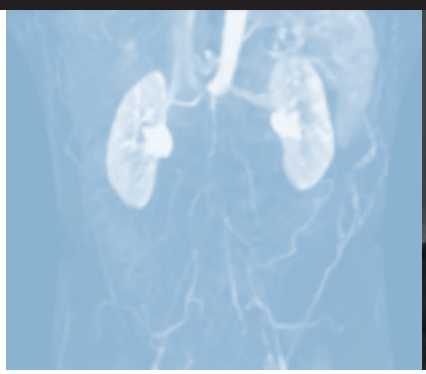
Patients who have lipid abnormalities and would like to participate in research trials involving the newer therapeutic agents can be referred to Oklahoma Heart Institute.

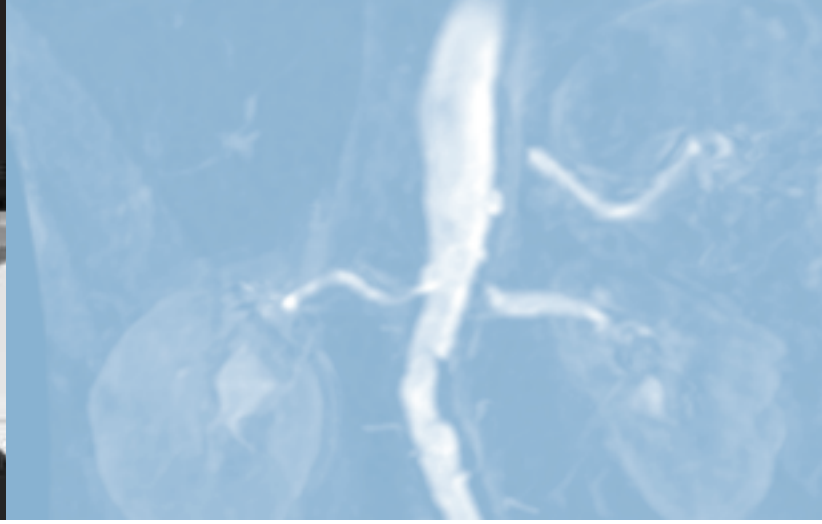
In addition, patients with abdominal obesity and evidence for the metabolic syndrome are also eligible for the trial using rimonabant. For more information, call 592-0999, and ask to speak with one of the Research Nurses.

(Dr. Leimbach is Director of the Oklahoma Heart Institute Research and Education Foundation.)

Oklahoma Heart Institute

by Edward T. Martin, MS, MD, FACC, FACP, FAHA





Cardiovascular Magnetic Resonance

Cardiovascular magnetic resonance (CMR) continues to expand its diagnostic capabilities since its inception in the 1980s. Recent technological advances have allowed the addition of myocardial stress testing and the evaluation of myocardial viability and patent foramen ovale to its diagnostic armamentarium. Stress testing can be performed with either dobutamine or adenosine, and results compare favorably to radionuclide and stress echocardiogram testing. Patent foramen ovale evaluation is performed in "real time" using the contrast agent gadolinium to diagnose the defect, as one would use agitated saline in echocardiography. In the last few years, delayed enhancement CMR has surpassed positron emission tomography (PET) imaging as the new gold standard for myocardial viability. The current clinical applications (Figures 1 and 2) in vascular and cardiac imaging are varied, allowing CMR to perform the tasks done by echocardiography, nuclear testing, as well as the invasive vascular labs. This versatility has led imaging professionals to proclaim it "the one-stop-shop".

Figure 1 **Clinical Indications for Cardiovascular MRI**

Cardiac

- Right and left ventricular cardiomyopathies
- Valvular function (qualitative/quantitative)
- Pericardial disease
- Para and intracardiac masses
- Congenital heart disease
- Intracardiac shunts (atrial/ventricular septal defects and vascular shunts)
- Patent foramen ovale
- Poor quality echocardiograms
- Dobutamine and vasodilator stress MRI
- Myocardial viability
- Syndrome X

Figure 2 **Clinical Indications for Cardiovascular MRI**

Vascular

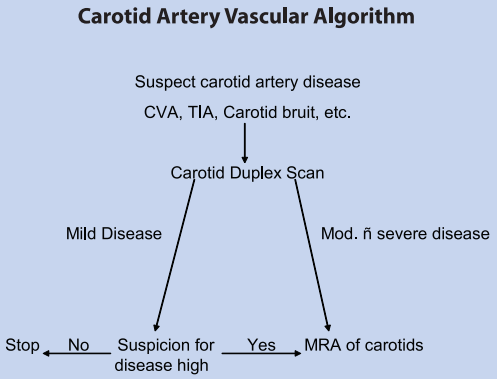
- Aortic dissection, aneurysm and intramural hematoma
- Aortic arch anomalies
- Aortic branch vessel disease
- Carotid artery disease
- Subclavian artery stenosis
- Renal artery disease
- Iliofemoral disease
- Popliteal and infrapopliteal disease
- Anomalous coronary arteries
- Coronary artery bypass graft patency
- Pulmonary emboli

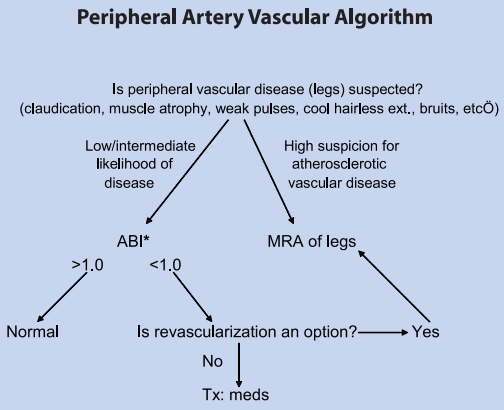
With CMR, all diagnostic vascular evaluation can be done noninvasively with minimal risk, which allows Magnetic Resonance Angiography to fit nicely into a routine cardiovascular medical practice (Figure 3). During the evaluation, an IV is placed and gadolinium is given to "light up" the arteries. There are no arterial punctures, and, so, there is minimal risk to the patient. Gadolinium is "kidney friendly" and can be given safely to diabetics and patients with renal disease.

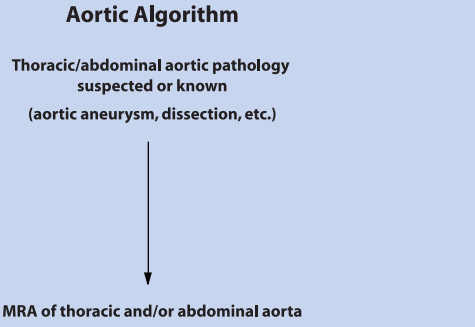
CMR may be limited by certain contraindications (Figure 4). Prosthetic heart valves and vascular stents are safe. Previously, pacemakers and implanted cardioverter defibrillators (ICDs) have been a strict contraindication to MR testing. However, OHI cardiologists have led the research in this area finding that undergoing MRI with these devices is not as detrimental as once thought. Because of this research, patients with devices who previously were denied MRI can have the test performed.

The Cardiovascular MRI Center at Oklahoma Heart Institute has recently begun its seventh year of serving Oklahoma with noninvasive cardiac and vascular diag-

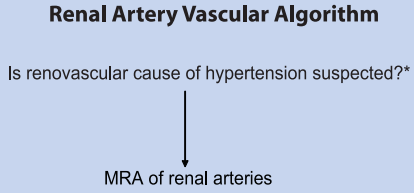
Figure 3 Magnetic Resonance Angiography Vascular Algorithms







Note: MRA of these regions should also be used to follow chronic dissection/aneurysm



*Onset of diastolic hypertension after age 55
Exacerbation of previously well controlled hypertension
Resistant hypertension
Epigastric bruit
Unexplained azotemia
Azotemia while receiving ACE inhibitors
Discrepancy in size of kidneys

Note: Use as the initial diagnostic test

Algorithms published in: Auerbach, E and Martin, E. Am Heart J 2004;148:755-633

nostics. In addition to clinical diagnostics, the center participates in multiple research projects and is an internationally recognized CMR training center for physicians and technologists. Current research project areas include stress myocardial perfusion, myocardial viability, ICD/pacemaker and MRI interactions, left ventricular hypertrophy and peripheral vascular disease.

To schedule a patient for a clinical study or to inquire about a research project call 592-0999.

(Dr. Martin is a noninvasive cardiologist with subspecialty expertise in noninvasive imaging. He is Director of Cardiovascular Magnetic Resonance Imaging at Oklahoma Heart Institute.)

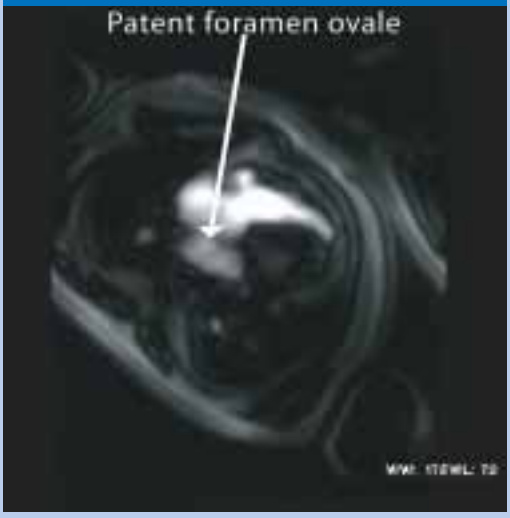
Figure 4 Contraindications to Cardiovascular MRI (partial list)

- Implanted cardiac devices (pacemakers, ICDs...)*
- Neurostimulators
- Cochlear implants
- Bone growth stimulators
- Intracranial aneurysm clips
- Foreign body near vital structures
- Pre-6100 series Starr Edwards heart valves
- First trimester of pregnancy

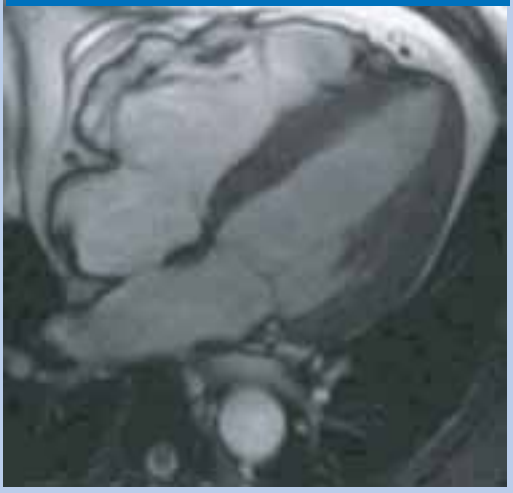
*Performed at Oklahoma Heart Institute under a research protocol

Clinical Cardiovascular Magnetic Resonance Images

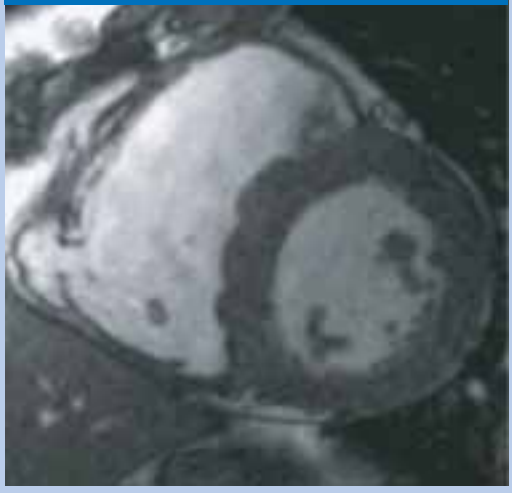
PFO Sequence



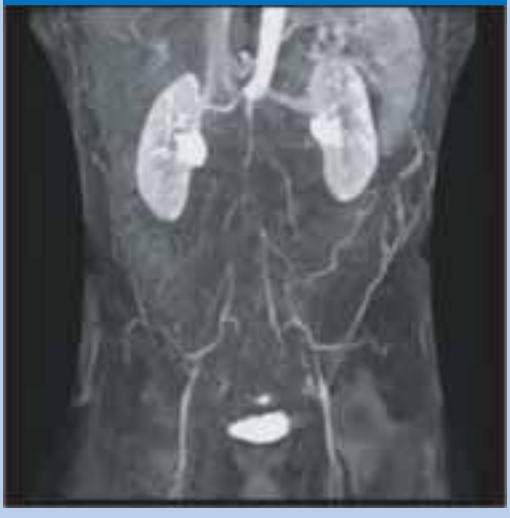
Arrythmogenic RV Dysplasia 4 Chamber view



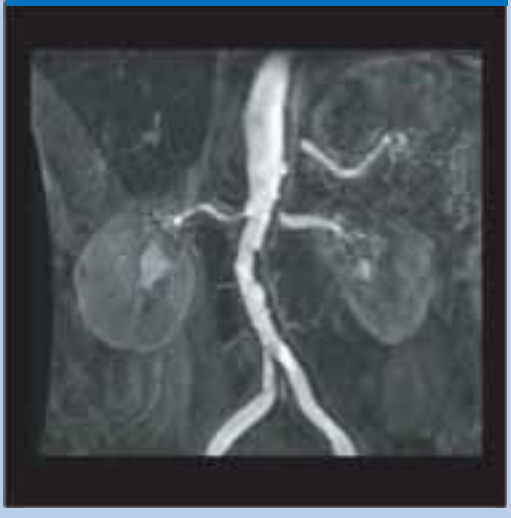
Arrythmogenic RV Dysplasia Short Axis view



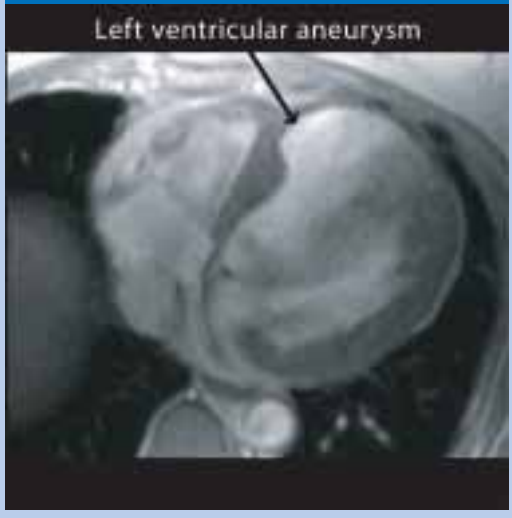
Occluded Aorta - Leriche Syndrome



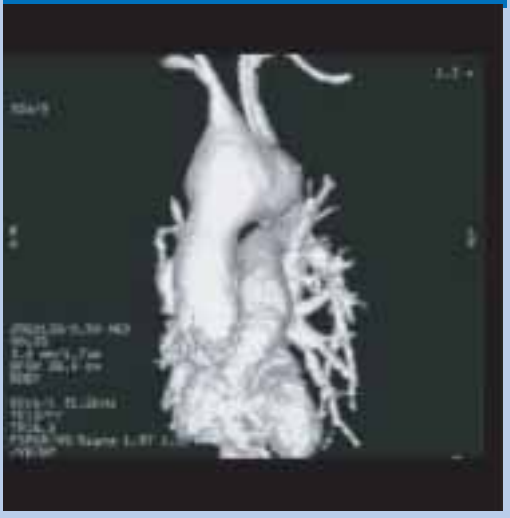
Left Renal Artery Stenosis



Ischemic Cardiomyopathy

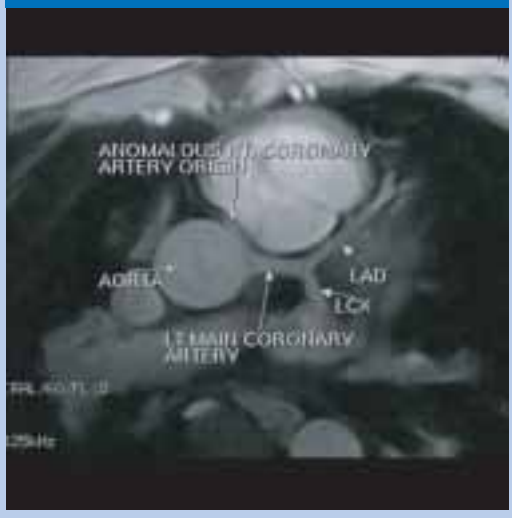


Thoracic Aortic Aneurysm

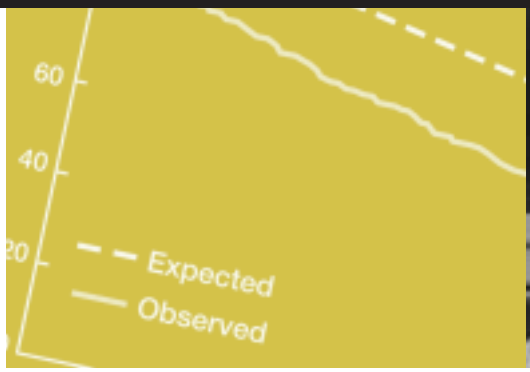


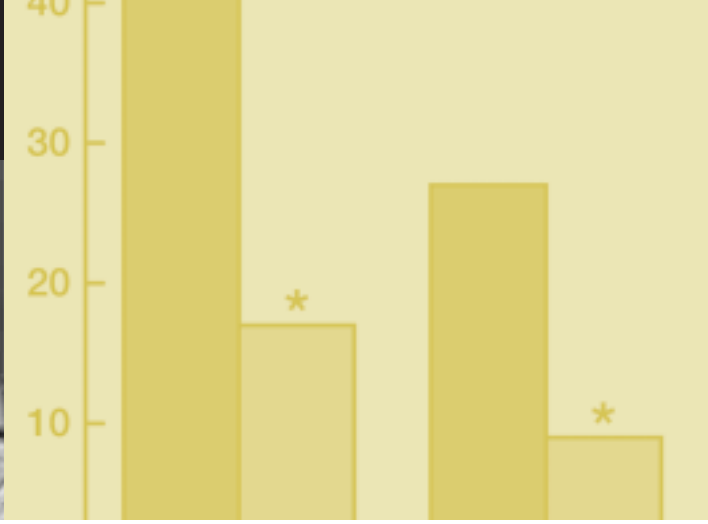
Thoracoabdominal Aortic Dissection

Anomalous Right Coronary Artery



■ *by* **Gregory D. Johnsen, MD, FACC**





The Benefits of Cardiac Rehabilitation

Coronary heart disease is the leading cause of death for both men and women in the United States. It is also a major cause of physical disability, particularly in the rapidly growing population of elderly persons.[1,2] The prevention of future coronary events in these patients and maintenance of optimal function status will be critically important to ensure they can resume active and productive lives.

According to the World Health Organization, cardiac rehabilitation can be defined as the "sum of activity required to ensure cardiac patients the best possible physical, mental, and social conditions so that they may by their own efforts regain as normal as possible a place in the community and lead an active life." Major objectives of cardiac rehabilitation include not only an improved functional capacity and quality of life, but also a reduction in mortality and morbidity.[3]

A simpler definition holds that cardiac rehabilitation has two aims: to return the cardiac patient to the best possible health and to reduce to the minimum the risk of recurrence of cardiac illness. The first aim must take into account all aspects of health, not only physical well being, but also psychological health, employment status, and social and economic recovery. To achieve this requires a truly multidisciplinary approach. The second aim is the maintenance of good health in the long term; this means ensuring that revascularization is performed when appropriate, that medication is optimal and that the patient understands all the relevant risk factors and the importance of continuing an appropriate lifestyle.[4]

Fifty years ago, patients who survived a myocardial infarction were confined to bed rest for 2 months or longer and then urged to limit their physical activity indefinitely. Avoidance of physical activity was likewise advocated for those with angina. The realization that bed rest hindered recovery and contributed to complications radically altered the rehabilitation of cardiac patients.[5] Vilifying extended bed rest as something that "saps morale, provokes desperation, unleashes anxiety, and ushers in hopelessness of the capacity of resuming a normal life," Levine advocated limited

Figure 1
Traditional Terminology for the Phases of Cardiac Rehabilitation.

Phase I	Inpatient rehabilitation usually lasting for the duration of hospitalization. It emphasizes a gradual, progressive approach to exercise and an educational program that helps the patient understand the disease process, the rehabilitation process, and initial preventive efforts to slow the progression of disease.
Phase II	Multifaceted outpatient rehabilitation, lasting 2 to 3 months. Emphasizes safe physical activity to improve conditioning with continued behavior modification aimed at smoking cessation, weight loss, healthy eating, and other factors to reduce disease risk. Initiate an exercise prescription.
Phase III	Supervised rehabilitation lasting 6 to 12 months. Establishes a prescription for safe exercise, and continues to emphasize risk factor reduction.
Phase IV	Maintenance, indefinite.

Figure 2
Efforts of Exercise-Based Cardiac Rehabilitation on Study End Points*.

Outcome	Mean Difference, %	95% Confidence Limit	Statistical Difference
Total Mortality	-20	-7% to -32%	p=0.005
Cardiac Mortality	-26	-10% to -29%	p=0.002
Nonfatal MI	-21	-43% to 9%	p=0.150
CABG	-13	-35% to 16%	p=0.400
PTCA	-19	-51% to 34%	p=0.400

Mean difference is the percentage of difference between exercise-trained and usual-care control group. MI indicates myocardial infarction; CABG, coronary artery bypass graft; and PTCA, percutnaeous coronary angioplasty.

*Data are derived from Taylor et al.[2]

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early activity after myocardial infarction.[6,7] Inpatient cardiac rehabilitation was formally introduced in the 1960s when researchers discovered that cardiac patients who moved about with early activity, rather than staying in bed for extended periods, had better recoveries.

Early mobilization came to be called phase I or inpatient cardiac rehabilitation. Comprehensive rehabilitation programs eventually grew to include four phases (see figure 1).[5]

Medicare will cover cardiac rehabilitation for patients with a clear medical need, who are referred by their attending physician and have a documented diagnosis of acute myocardial infarction (MI), who have had coronary artery bypass surgery or have stable angina.

Concerning patients with stable angina, if the symptoms of angina have been stabilized with intervention or medication, the patient is not eligible for Medicare coverage for cardiac rehabilitation.

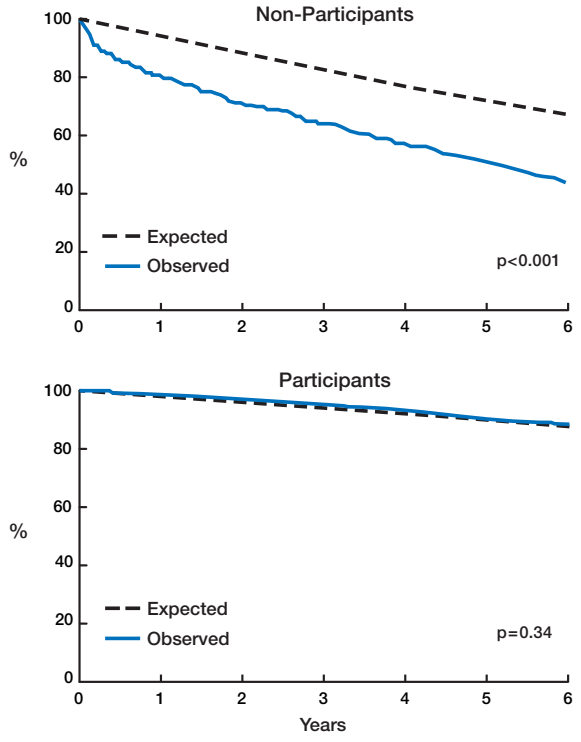
Despite multiple studies that have demonstrated the benefits of cardiac rehabilitation, only 10-20% of appropriate candidates participate in a formal supervised cardiac rehabilitation program.[8] This low participation rate is due to lack of physician referral, poor patient motivation, logistical constraints, and inadequate third-party reimbursements for services. Sometimes, the patient does not live near a cardiac rehab program and is unable to commute to the nearest program.

Several studies have demonstrated the benefits of cardiac rehabilitation. A meta-analysis of 21 randomized, controlled trials performed in the 1970s and 1980s, involving a total of 4,554 patients demonstrated a 20% reduction in overall mortality and in cardiovascular mortality at three years.[9] A recent meta-analysis involving 48 randomized trials and 8,940 patients reported similar findings with a 20% reduction in overall mortality and a 26% reduction in cardiac mortality[10] (see figure 2).

A recent study from the Mayo Clinic examined participation in cardiac rehabilitation after myocardial infarction in Olmsted County, Minnesota.[11] Participants in cardiac rehabilitation exhibited a marked survival advantage compared with non-participants, with three-year survival of 95%, compared with 64% among non-participants (p value <0.001).[11] The three-year survival among non-participants was significantly lower than the expected survival of the Minnesota total population at 83% (p <0.001). Conversely, there was no difference between the three-year survival among participants and the 95% expected survival of the Minnesota total population (p=0.34) (see figure 3).

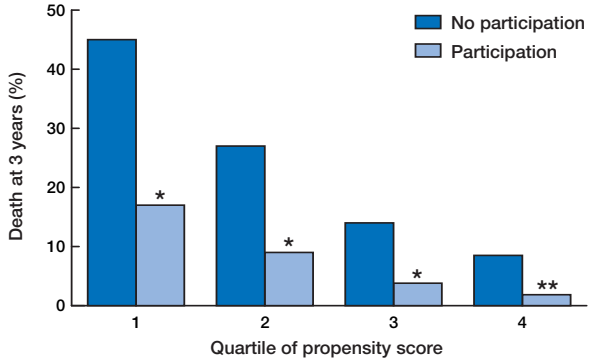
When the attributable risk of death related to non-participation was calculated by quartiles of the propensity to participate, the attributable risk was largest among individuals with the lowest propensity to participate in cardiac rehab (see figure 4). After adjusting for propensity to participate, participation in cardiac rehabilitation was associated with a 56% improvement in survival post MI (RR 0.44; 95% C1 0.36 to 0.54; p<0.001) which was similar across the age and gender groups.[11] There was also a 28% reduction in the risk of recurrent MI after adjustment for propensity score, age, and gender (p=0.049). In this community-based cohort study, approximately half of the patients participated in cardiac rehabilitation after MI, and the use of rehabilitation did not increase over time. Women and elderly patients were less likely to participate in

Figure 3
Expected and observed survivial by participation in cardiac rehabilitation.



Witt et al
JACC 2004; 44:988-96

Figure 4
Mortality within three years after myocardial infarction by participation in rehabilitation stratified by quartile of propensity to participate.



*p<0.001 for comparison between participation and no participation
**p<0.005

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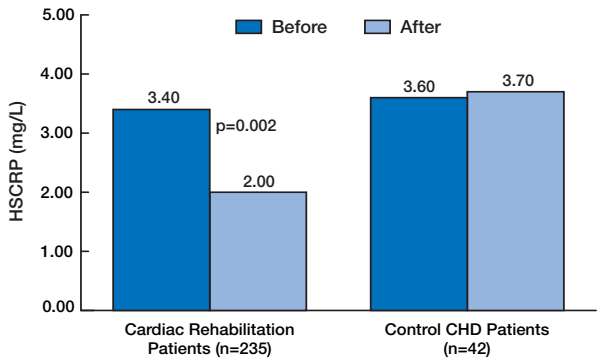
September 1, 2004:988-96

Figure 5
Median changes in high-sensitivity C-reactive protein (HSCRP) in cardiac rehabilitation and in control patients with coronary heart disease (CHD).



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cardiac rehabilitation. Participation in cardiac rehabilitation after MI was associated with large survival benefits in both genders and all age groups even after adjustment for the propensity to participate.[11]

Exercise training has numerous beneficial physiological effects. Exercise training leads to a lower heart rate and lower blood pressure. It also leads to reduced body weight, lower serum triglycerides, increased HDL cholesterol, reduction in circulation levels of norepinephrine, decrease in platelet adhesiveness and enhanced fibrinolysis. Exercise training increases endothelium-mediated coronary vasodilatation. It improves insulin sensitivity and glucose homeostasis, which has been show to reduce the risk of the type 2 diabetes mellitus in individuals with glucose intolerance. Thus, aerobic exercise can favorably modify all of the components of the metabolic syndrome.[12]

A recent study demonstrated the beneficial decrease in high-sensitivity C-reactive protein (HSCRP) levels with a 3-month phase II cardiac rehabilitation and exercise training program.[13]

HSCRP is an inflammatory marker, which provides for a global assessment of cardiovascular risk.[14]

Several prospective studies have shown that plasma levels of HSCRP are a strong independent predictor of risk of future vascular events.[15,16] This study analyzed plasma levels of HSCRP in 277 patients with CHD (235 consecutive patients before and after formal phase II cardiac rehabilitation and exercise training programs and 42 "control" patients who did not attend cardiac rehabilitation. Rehabilitation patients had a 41% reduction (p=0.002) in HSCRP levels (see figure 5). Patients on statin therapy had a 42% reduction in HSCRP (3.45 to 2.0 mg/L p=0.002) which is similar to that observed in statin-naïve patients (3.2 to 2.0 mg/L p=0.003) (See figure 6). This suggests that the effects of the rehabilitation program are unique and incremental over those of statin therapy. They also observed similar reductions in HSCRP regardless of variation of weight, suggesting that other aspects of the intervention program, including dietary modification and exercise training, may have played a prominent role in reducing inflammation[13] (see figure 7).

Exercise training improves psychological functioning. Longitudinal studies have shown that exercise training reduces depression in patients with cardiac disease. It improves self-confidence and self-esteem, attenuates cardiovascular and neurohumoral response to mental stress, and reduces some type A personality behaviors.[17]

Cardiac rehabilitation is safe. The occurrence of major cardiovascular events during supervised exercise in contemporary programs ranges from 1 per 50,000 to 1 per 120,000 patient-hours of exercise, with only 2 fatalities reported per 1.5 million patient-hours of exercise.[18]

Cardiac rehabilitation is cost-effective at $2130-4950 per life-year saved, which compares very favorably with other accepted treatments for coronary heart disease such as statin therapy ($9630) and coronary artery bypass graft surgery ($8500-114,000).5 Only smoking cessation programs are uniformly more cost-effective ($220-728 per life-year saved) than cardiac rehabilitation.

A crucial point is not to consider cardiac rehabilitation as exercise training only, but as a program aiming to improve the quantity and quality of life by reduction of the classic risk factors, such as smoking and cholesterol levels, modification of dietary habits, increase and maintenance of endurance training, psychological support and guidance on returning to work.[19]

Oklahoma Heart Institute is committed to provide state-of-the-art care for patients who have experienced a myocardial infarction or have had coronary artery bypass graft surgery. Cardiac rehabilitation is a crucial component to ensure that patients can return to the best possible functional status and to reduce to a minimum the risk of recurrence of a cardiac event.

(Dr. Johnsen is an interventional cardiologist with expertise in cardiac catheterization, angioplasty and related interventional procedures such as stents and atherectomy.)

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Figure 6

Change in high-sensitivity C-reactive protein (HSCRP) following cardiac rehabilitation and exercise training in patients taking statins and in patients not taking statins.

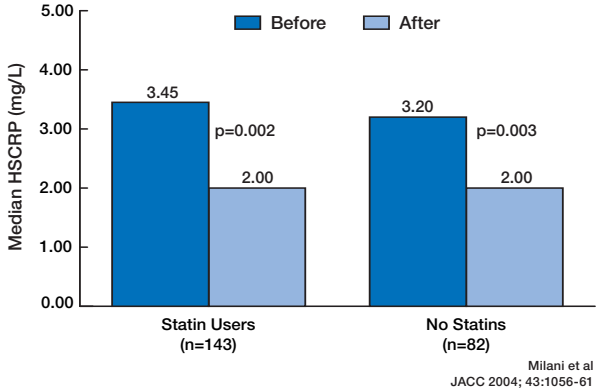
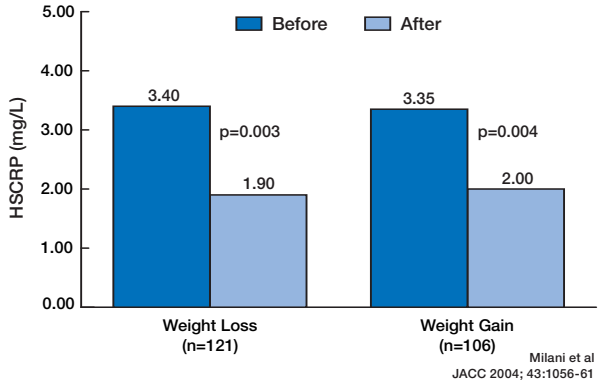


Figure 7

Changes in high-sensitivity C-reactive protein (HSCRP) following cardiac rehabilitation and exercise training in patients who achieved weight loss vs. patients who gained weight.



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