Paul F. McDonagh, PhD, has been appointed The Allan C. Hudson and Helen Lovaas Chair of Vascular Biology and Coagulation.

“Because of his strong basic and applied research background, the appointment of Dr. McDonagh is a move of strength,” says Jack G. Copeland, MD, a co-director of the Sarver Heart Center and chief of cardiovascular and thoracic surgery at the UA. “He will make major contributions immediately.”

Dr. McDonagh, who splits his time between teaching and research, is active in exploring vascular biology and coagulation as they apply to a broad spectrum of vascular diseases. This focus allows for collaboration with many other Sarver Heart Center members. For example, Dr. McDonagh’s research in inflammation and coagulation as they apply to artificial hearts, mechanical circulatory support devices and heart-lung machines provides an opportunity for collaborative research with the Sarver Heart Center’s basic scientists, cardiothoracic surgeons, pharmacologists, physiologists and cardiologists. These combined efforts to improve the bio-compatibility of blood and materials used in cardiovascular devices will lead to new and better medical and surgical approaches to the management of advanced heart failure.

In another area—the mechanisms of the inflammatory response to ischemic injury to the heart and brain, caused by restricted blood flow—his research will have far-reaching beneficial effects in the care of patients with a wide variety of cardiovascular illnesses, from heart attacks to strokes.

Gordon A. Ewy, MD, director of the Sarver Heart Center, noted that these projects will involve collaboration between Sarver Heart Center basic scientists, cardiologists, pharmacologists and neurologists and represent “state-of-the-art” research.

Dr. McDonagh will have a broad spectrum of responsibilities in the Sarver Heart Center related to cardiovascular research, Dr. Ewy added. He will represent the Center on the

Continued on page 2
Almost All You Need to Know About Heart Failure

One of my pathology professors in medical school used to say: “I’m going to talk about 90 percent of what you need to know about such and such.” What followed was always concise and useful. I hope to do the same for you with regard to heart failure.

Heart failure is present in more than 5 million people in the United States and increases by 550,000 each year. Fortunately, the vast majority of these individuals can be treated with medical therapy. Unfortunately, according to a study by the National Institute of Medicine, about 65,000 people per year in our country fail medical therapy and need “cardiac replacement,” more commonly known as a heart transplant.

Heart failure is caused by conditions that harm the heart muscle – called cardiomyopathies. Common causes of cardiomyopathies include myocardial infarction (heart attacks), blockage of the coronary arteries and high blood pressure. Less common, but equally important, causes include viral infection, pregnancy, alcoholism and congenital heart disease. Medical and surgical therapies have improved the outcomes of patients with both acquired (adult) and congenital (pediatric) heart disease. This means that more people are living longer with heart failure. But more may become so sick that they need cardiac replacement.

About 2,000 patients receive heart transplants in the United States each year. Over 6,000 are on the “waiting list” over the course of one year. Those lucky recipients have a 94 percent one-year and a 50 percent 12-year survival in our program, compared with an expected one-year survival of less than 50 percent without cardiac replacement. Obviously, there is a huge discrepancy between the 2,000 transplanted patients and the 65,000 people per year who need heart replacement to survive, raising the question of artificial hearts.

Artificial hearts may soon play a major role. The “Rematch Study” of a left ventricular assist device (an implantable device that helps the heart pump) showed that LVAD patients had better survival than a matched group of patients on medical treatment alone, but there were some issues that would lead most people to conclude that the LVAD is not “ready for prime time.” Durability of the device and infection were two major limiting problems.

I believe that such problems will be solved and that in the next five to 10 years mechanical circulatory support devices will play a major role in helping patients who fail on medical therapy.

The future of heart failure also includes new prevention strategies, more aggressive attempts to identify and treat patients with decreased heart function before they develop heart failure, improved medical therapy for heart failure patients in specialized heart failure clinics, more palliative operations done in the setting of stand-by with artificial heart support, and finally adding a mechanical option to the current standard of heart transplantation.

Gordon A. Ewy, MD, and I and other members of the Sarver Heart Center are busy with plans for a statewide program in all of these areas and will keep you posted as we make progress. ♥

McDonagh
from page 1

College of Medicine Dean’s Research Council. He also will spearhead a new program to obtain National Institutes of Health supported Sarver Heart Center research fellows and postdoctoral training programs. He also will develop a “pre-review” and mentoring program that will improve funding from outside granting agencies, including the NIH.

Being appointed to the endowed chair is “an honor and a privilege,” Dr. McDonagh said. “I look forward to working with my colleagues to further develop the overall research environment in the Sarver Heart Center.”

The endowment, he added, will help us to develop a stronger, more comprehensive research program aimed at inflammation-induced thrombosis in cardiovascular disease. A better understanding of the underlying causes of a problem allows for the development of improved treatments.”

Dr. McDonagh will collaborate with Leslie Ritter, RN, PhD, and Paul E. Nolan Jr., PharmD.

Dr. McDonagh has a bachelor’s degree from Worcester Polytechnic Institute, a master’s degree from Columbia University and a doctorate in physiology from the University of California at Davis. He was a postdoctoral fellow at the UA College of Medicine. He is a member of the Departments of Surgery and Physiology. ♥

By Jack G. Copeland, MD
Galloway Accepts Diabetes Award for ACC

James M. Galloway, MD, accepted an award on behalf of the American College of Cardiology (ACC) in recognition of his and the group’s efforts to raise awareness about the relationship between diabetes and cardiovascular disease.

The American Diabetes Association presented Dr. Galloway with the 2003 C. Everett Koop Award for Health Promotion and Awareness at the ADA’s annual meeting, held recently in New Orleans.

Dr. Galloway has been a leader in the successful “Make the Link: Diabetes, Heart Disease and Stroke” public service campaign. The campaign is a collaborative effort between the ADA and the ACC aimed at increasing awareness – among physicians and patients – of the link between diabetes and heart disease.

The campaign was launched in 2002 with a phone survey that returned some surprising results. Despite significantly higher rates of heart disease and stroke in people with diabetes, more than half of diabetics surveyed felt they were at little or no risk of a heart attack. Sixteen percent could not name one important thing they could do to reduce their risk.

Based on the survey results, “Make the Link” developed a significant education campaign for health professionals and patients, stressing the importance of the ABCs of diabetes: Hemoglobin A1c (blood sugar) control, Blood pressure control and Cholesterol control.

Dr. Galloway, an assistant professor of clinical medicine at the University of Arizona College of Medicine, has been involved with the campaign for the past year.

As director of the Native American Cardiology Program, Dr. Galloway knows well the cardiovascular consequences of diabetes. American Indian communities are experiencing an epidemic of diabetes and cardiovascular disease.

The Native American Cardiology Program is a unique collaboration between the Indian Health Service, the UA, University Medical Center, the Flagstaff Medical Center and the Southern Arizona VA Health Care System.

The program is associated with the UA Sarver Heart Center and is dedicated to the prevention and treatment of cardiovascular disease among American Indian communities.

Copeland Discusses Transplantation, Artificial Heart at Vienna Conference

University Medical Center’s experience with the CardioWest total artificial heart was the topic of a presentation given by Jack G. Copeland, MD, at an international conference this spring.

Dr. Copeland, chief of the Section of Cardiovascular and Thoracic Surgery at the University of Arizona, gave a talk titled “The Total Artificial Heart – Nine-year Experience in 61 Patients” at a conference of the International Society for Heart and Lung Transplantation, held in Vienna.

UMC has been the main center for the study of the CardioWest, a total artificial heart that has been used as a “bridge to transplant” for patients waiting for heart transplants.

Dr. Copeland also participated in a debate on the role of the surgeons and cardiologists in heart transplant programs.
Hold Your Breath
A New Way to do CPR is Coming

The survival from out-of-hospital cardiac arrest is very poor. In contrast to the high survival rates of patients with cardiac arrest on television shows like "ER," survival from out-of-hospital cardiac arrest in real life is rare. In most communities throughout the world, survival is less than 10 percent.

We believe it is time for dramatic changes in the way CPR has been performed over the past 40 years. Recent findings by the Sarver Heart Center’s CPR Research Group are pointing to a new and easier way to do CPR that could save countless more lives.

We know that one of the biggest problems with standard CPR (15 compressions followed by two mouth-to-mouth breaths) is that most people are reluctant to administer the breaths, and therefore do nothing to help the victim until paramedics arrive.

About a decade ago, laboratory studies done by the SHC CPR Research Group showed that "continuous chest compression" CPR—meaning without interruptions for mouth-to-mouth breathing—is far better than doing nothing at all.

Further research found that continuous chest compression CPR was as effective as "perfect" CPR, which is 15 chest compressions followed by a four- to five-second break to give two quick breaths.

Recently, SHC researchers learned that it actually takes the average person about 15 or 16 seconds to give the two breaths, roughly the same amount of time it takes to do 15 chest compressions. That means that chest compressions are being performed less than half the time, depriving the brain and other organs of blood and oxygen.

Colleagues in Europe have made many of the same findings. But because international CPR guidelines are revised only every five to seven years and the last update was issued three years ago, we feel we can’t wait to get the word out about continuous chest compression CPR so that people and organizations can start following it now.

In some instances, mouth-to-mouth breathing still will be absolutely necessary. These include suspected drowning and drug overdoses, and respiratory failure in children.

In the vast majority of witnessed adult collapses, ventricular fibrillation is to blame. Ventricular fibrillation is an electrical disturbance of the heart where the individual heart muscle fibers in the main pumping chamber are not contracting in unison. As a result, the ventricles—the main pumping chambers of the heart—stop pumping blood. The blood pressure abruptly falls and the victim rapidly loses consciousness and collapses. Even though the person may gasp, someone should begin CPR immediately.
There are different phases of cardiac arrest, and different approaches are needed for each. During the first phase – the electrical phase, which occurs during the four or five minutes after the collapse – an automated external defibrillator may be all that is needed to resuscitate the victim.

Studies have shown that AEDs on commercial airlines and in casinos (as demonstrated by Sarver Heart Center researchers Terence D. Valenzuela, MD, and Lani L. Clark) markedly improve survival.

The second phase is the hemodynamic phase and lasts for several additional minutes. Since paramedics rarely arrive within five minutes after an out-of-hospital collapse, most patients are in this phase when help arrives. Studies by the CPR group have shown that in this phase, prompt forceful chest compressions prior to defibrillation are essential for improved survival. Researchers from Sweden have confirmed these findings.

Karl B. Kern, MD, and Gordon A. Ewy, MD, recently presented the groundbreaking work of the SHC CPR group at the London CPR symposium. They reported that studies done by the CPR Research Group indicate that survival for out-of-hospital cardiac arrest would be much better with continuous chest compressions than with standard CPR. Further, standard CPR (with delays of 15 to 16 seconds between chest compressions for ventilation) results in a survival rate of 13 percent, compared with 70 percent to 80 percent for continuous chest compression CPR with no pauses for breaths.

It is of interest that Tucson’s survival rate for out-of-hospital witnessed cardiac arrest due to ventricular fibrillation has been 13 percent to 14 percent over the past five years.

Many studies have shown that bystanders are more likely to perform CPR if they don’t have to give mouth-to-mouth, and that survival chances are far worse when no CPR is administered. Eliminating the requirement for mouth-to-mouth breathing not only improves survival, but also increases how often bystanders give CPR.

In coming months, the Sarver Heart Center will be working with the medical and lay community to improve the city’s cardiac arrest survival rates. We believe our efforts will bring forth significant changes in both CPR and dramatic increases in survival after cardiac arrest.

In addition to Drs. Ewy, Kern and Valenzuela and Clark, the Sarver Heart Center CPR Research Group includes: Robert A. Berg, MD; Ronald W. Hilwig, DVM, PhD; Charles Otto, MD; and Arthur B. Sanders, MD.

We feel we can’t wait to get the word out about continuous chest compression CPR so that people and organizations can start following it now.
Cholesterol lowering drugs, known as statins, have been shown to save lives. A better understanding of how the drugs work could yield discoveries of new treatments for patients with heart failure, giving them a new tool with fewer side effects.

Heart failure is among the most debilitating and costly diseases today. It affects more than 65 million Americans and the care of these patients cost an estimated $105 billion in 2001.

A common cause of heart failure is the blockage of coronary arteries. The heart muscle that is fed by those arteries dies and those cells are replaced with scar tissue, which is unable to contract. This results in decreased heart function and, eventually, heart failure.

Researchers have found that arteries in heart failure patients are stiffer due to a defect in the cells that line the artery walls.

A research team headed by Kathryn Bates, DO, has found that one statin—simvastatin—helps the arteries relax, which decreases the resistance against which the heart has to pump.

Some of the team’s findings:
- Simvastatin enhances arterial tone and relieves the heart from pumping against stiffer arteries.
- Statins achieve this by increasing the levels of a protein called nitric oxide synthase, which maintains tone in artery walls.

The team continues to study how nitric oxide synthase is regulated and how simvastatin might be involved in that process.
medical students were taught both and then tested immediately after training, at six months and at 18 months.

Among the findings:
- Students took longer to start CPR when doing the standard version.
- Students took much longer than expected to give each breath.
- Many of the ventilations (mouth-to-mouth breaths) were done incorrectly and little air reached the victims’ lungs.

Using these and other findings, the CPR Research Group is focused on developing a new type of CPR that would be easier to perform and would save more lives. (See article on pages 4-5.)

The Murcott Cardiovascular Research Award
Modulation of Impaired Nitric Oxide Mediated Endothelial Dependent Vasorelaxation in Congestive Heart Failure Through \( \beta_3 \)-Adrenergic Receptor Activation
Hoang Thai, MD

One element of decompensated heart failure (heart failure accompanied by fluid retention and swelling) is elevated peripheral vascular resistance (PVR), which makes it more difficult for the heart to pump blood to the extremities.

Scientists have discovered that heart failure symptoms can be dramatically improved by using certain drugs to modulate the activation of the renin angiotensin aldosterone and \( \beta \) adrenergic systems. These approaches have become the cornerstone of contemporary heart failure therapy.

But scientists remain unclear on how exactly these drugs improve mortality. One possibility is that they help relax the arteries by increasing levels of nitric oxide.

Hoang Thai, MD, and other researchers examined a \( \beta \) receptor called \( \beta_3 \)-AR to see if blocking the receptor increased nitric oxide levels, thus improving the pressure levels in the arteries.

Their study compared the use of: no drugs; BRL 37344 (a \( \beta_3 \)-AR blocker); and Carvedilol, which is a \( \beta_{1+2} \)-AR blocker.

The researchers found that both beta blockers produced significant improvement of arterial pressures, apparently due to increased levels of nitric oxide.

The study points to a new way to decrease vascular resistance in patients with heart failure.

The William J. “Billy” Gieszl Endowment for Heart Research
Gene Expression Profiling During Heart Valve Development Using cDNA Microarray Analysis
Ian Jongewaard, PhD

Using new technology to shed light on the genes involved in the formation of the human heart could yield important information about the prevention and treatment of heart defects.

A research team led by Ian Jongewaard, PhD, used DNA microarrays to learn about the genes that are involved in embryonic heart valve development.

Before microarray technology, heart development could be examined only a few genes at a time.

“The knowledge gained through this application may have tremendous potential for the development of new and more effective diagnostic, preventive and therapeutic approaches, as well as provide new avenues for research,” Dr. Jongewaard said. ♥
Fact or Fiction?
A look at two common misconceptions

By Gordon A. Ewy, MD
UA Sarver Heart Center Director

Health myths are rampant and often arise from the spark of an observation or theory, then are fanned into a flame by the enthusiasm of the discoverer and by entrepreneurs who stand to profit from the sale of an item or an approach. They spread like wildfire by ordinary people who want an easy way to solve a difficult problem or by desperate people looking for miracles.

Some of these myths sound very plausible and may cite seemingly credible sources. The old rule usually applies: If it sounds too good to be true, it probably is.

Can coughing save my life if I have a heart attack while I am alone?

Let’s take a recent myth that has been circulating via e-mail. As the Sarver Heart Center has been active in the science and application of defibrillation and cardiopulmonary resuscitation for years, it eventually made its way to my in-box.

At first I was amused. But then it kept resurfacing and patients continued to ask me about it. Since it is a colossal myth, it’s worthwhile to set the record straight.

The myth comes in an e-mail with a subject line stating something like “How to survive a heart attack alone.”

The idea being touted here is that if you are alone and have a heart attack, you can save your life by coughing. The example given is someone who is driving a car when they are struck with chest pain. It claims that repeated and vigorous coughing at the onset of a heart attack will keep your heart beating and help it to regain a normal rhythm.

There are so many misconceptions in this well intentioned e-mail that it is hard to know where to start. First, what you should do if you start experiencing severe pain in your chest that starts to radiate out into your arm and up into your jaw – the classic signs of a heart attack due to blockage of one of the coronary arteries – is to pull over to the side of the road and call 911 on your cell phone (or ask a passerby to make the call). You should inform 911 that you think you are having a heart attack and give your exact location. Then take an aspirin if you have one (not a Tylenol or ibuprofen, but aspirin). Lie down and try to be calm, but do not start coughing – that just increases the work of the heart and may cause complications. If you start to lose consciousness, as you might if you are having a cardiac arrest (the heart stops pumping blood) from your heart attack, you might cough but it is not recommended. Rather you should hope that help is on its way and that there is a bystander willing to do chest compressions and use an automated external defibrillator (AED) while waiting for paramedics.

Where did this idea of so-called “Cough CPR” come from?

Interestingly, it can be traced back to the hospital – the cardiac catheterization laboratory to be specific.

On occasion, when contrast material (dye) is injected into a coronary artery (usually the right coronary artery, as it most often supplies the sinus node or “spark plug” of the heart) the heart may stop beating for a few seconds. The heart almost always resumes beating again on its own as the contrast material supplying the “spark plug” is replaced by blood.

In an effort to shorten the period when the heart is not beating because of the dye in the coronary artery, the cardiologist often asks the patient to cough. If the heart beat stops, the cardiologist will say “cough” at a rate of once every two or three seconds, usually necessary for two or three coughs. The cough increases the pressure inside the chest, and consequently inside the aorta, and this increased pressure in the aorta pushes the dye through the coronary artery faster. As the injected dye in the coronary artery is replaced by blood, the heart resumes beating normally. This has been standard practice for at least 30 years.
Over two decades ago, there was a case in which a patient not only developed asystole (heart stopped beating), but then ventricular fibrillation – the rhythm that is the major cause of cardiac arrest. The cardiologist was having the patient cough in a rhythmic fashion. The rhythmic coughing kept the patient conscious for one minute. The patient then became unconscious. The cardiologist then got the defibrillator and shocked the patient’s rhythm back to normal.

On another occasion the defibrillator did not work so the patient was asked to continue coughing until the nurse ran to the next room for a second defibrillator. The patient was able to maintain consciousness for almost one minute during ventricular fibrillation by rhythmic coughing. This was reported as “Cough CPR” and is well known to cardiologists. However, the longest anyone has been able to maintain consciousness by coughing during ventricular fibrillation is one minute! Therefore it is a clear myth that one can maintain consciousness by rhythmic coughing if one has ventricular fibrillation. And there is no reason to start coughing if you think you are having a heart attack.

If you are having a heart attack or anything that seems like a heart attack, the approach is to call 911 as soon as possible. Save your breath and your energy – do not cough!

The American Heart Association addresses this myth on its website: www.americanheart.org. Visit the “Heart & Stroke Encyclopedia” and look under “Cough CPR.”

Will an ibuprofen a day keep the doctor away?

Aspirin therapy is recommended for anyone not allergic to it who has cardiovascular disease (heart attack, coronary artery bypass surgery, angioplasty, stent placement, angina, stroke, transient ischemia attack, peripheral vascular disease) and anyone at high risk for heart attack or stroke. Those at risk include people with a family history of early cardiovascular disease and people with elevated cholesterol, hypertension, diabetes, a smoking habit and/or newer risk factors such as an elevated high sensitivity C-reactive protein.

The reason is that aspirin has an anti-inflammatory effect and decreases the ability of the platelets to initiate the clotting process, a common final step in blocking arteries and causing symptomatic cardiovascular disease.

Ibuprofen is one of the many nonsteroidal anti-inflammatory agents, and it also has an antiplatelet effect, but only for the duration it is in the bloodstream—about four hours. Therefore ibuprofen cannot be substituted for an aspirin a day. So if you are taking ibuprofen for arthritis or other aches and pains and you are at risk (as outlined above) you also need to take aspirin. However, they cannot be taken together as ibuprofen interferes with the beneficial effects of aspirin.

Aspirin is prescribed because it attaches to the platelet receptor and disables the platelets so that they do not function normally and the blood is less able to clot. Because ibuprofen diminishes this effect, aspirin should be taken at least 12 hours, and preferably longer, from the time the ibuprofen is taken.

The amount of aspirin one needs to take is controversial but, in general, it is about two baby aspirin (81 mg each) a day or one adult aspirin (325 mg) every other day. Many people undoubtedly can be protected with as little as one baby aspirin a day. But to be sure, one would have to measure the aspirin’s effect, which is rarely done. ♥
Modifier Genes Shed Light on Heart Condition

Scientists are covering new ground and making important discoveries about a heart condition that has genetic origins and, in half of all cases, is passed down family lines.

The condition is hypertrophic cardiomyopathy, which is one of three main types of cardiomyopathy. While coronary artery disease attacks the arteries and valvular disease strikes the valves, cardiomyopathy affects the muscle of the heart. In general, it causes the left ventricle to enlarge. It also sometimes causes enlargement of the septum, the wall that separates the right and left sides of the heart.

Scientists know that hypertrophic cardiomyopathy is caused by a mutation in the sarcomeric protein. But more clues about how the condition emerges might be found through study of “modifier” genes, which are at the center of emerging research into the condition, says Eugene Morkin, MD. Modifier genes are not the chief culprits, but rather genes that somehow affect the process that determines the physical appearance of the heart.

The good news about hypertrophic cardiomyopathy is that it is less deadly than previously thought. However, its consequences—congestive heart failure and sudden cardiac death—can be fatal.

Below are the three types of cardiomyopathy plus some of the features of each.

**Hypertrophic cardiomyopathy**
- The muscle of the left ventricle is enlarged.
- The wall between the two ventricles (the septum) is enlarged and, in some cases, can contribute to obstruction of blood flow from the left ventricle.
- More than half of these cases are hereditary.
- Most common in young adults, but can be diagnosed at any age.
- Symptoms include shortness of breath on exertion, dizziness, fainting and chest pain.
- Greater chance of sudden death in patients who were diagnosed before the age of 30 and who have a family history of sudden cardiac death.

**Dilated (congestive) cardiomyopathy**
- Most common form.
- Heart cavity is enlarged and the heart does not pump well.
- Leads to congestive heart failure in most patients.
- Poor prognosis, but 20 percent of patients improve.
- In the worst cases, heart transplantation may be needed.

**Restrictive Cardiomyopathy**
- Least common form.
- The ventricles become very rigid, making it harder for them to fill with blood between heartbeats.
- Symptoms include fatigue, swollen hands and feet, difficulty breathing with exertion.
- Usually caused by systemic disease.

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**Types of Cardiomyopathies**

RA = Right Atrium  RV = Right Ventricle  LA = Left Atrium  LV = Left Ventricle

Normal  Hypertrophic  Dilated  Restrictive
A Tribute to Courage and Selflessness

It was not until Therese Velasco Berg stood on the shore of Ellis Island and looked out across the vast expanse of ocean that she realized the depth of respect she felt for her mother and aunt.

“I stood there in the shadow of the Statue of Liberty and my eyes became teary thinking about the courage of these two teen-age girls leaving their home in Germany to travel to the United States.”

At ages 18 and 17, her mother and aunt settled in Germantown in Philadelphia, until about 1906, when they traveled west. Her aunt, Marie Schreiner, eventually moved to Victoria, British Columbia, and her mother, also named Therese, settled in Los Angeles. Both cooked and cleaned for doctors and their wives and became central to the lives of the families for whom they worked.

In 1911, Therese met Charles Velasco, who worked as a chauffeur for the family next door. Born and raised in Tucson, Charles also was from a family tradition of courage and selflessness. His father, Carlos Ygnacio Velasco, a lawyer from Hermosillo, Sonora, Mexico, came to Tucson in the 1860s. In 1870, he started El Fronterizo, a Spanish-language newspaper. He also was the principal founder of the Alianza Hispano-Americana, a mutual aid and benefit society dedicated to ensuring the care of Hispanic citizens and their families.

After marrying in Los Angeles in 1916, Charles and Therese moved to Tucson, setting up their home in the old Fronterizo building at 475 S. Stone Ave., which had been converted into apartments by Charles’ mother. They later purchased a home at 601 S. Stone Ave., where daughter Therese was born on Dec. 30, 1919.

“My mother was completely surprised at the state of Tucson in 1916. Stone Avenue was cobblestones, but the rest of the streets were dirt.”

Charles Velasco was the proprietor of a thriving auto supply and service station business. Like many other businesses, it failed during the Depression. After campaigning for a winning candidate for sheriff, he became a respected and much-loved deputy sheriff, and held the job until he was 72.

Mrs. Berg continues to be inspired by her parents, who cared for her and ensured that she received the best care available to treat the polio she contracted at age 3. At age 9, she underwent corrective surgery at the Shriners hospital for children in San Francisco; she returned at age 11 for another operation. After returning to Tucson, she began swimming regularly—an activity she pursues still today. A retired educator, she attended the University of Arizona, receiving her undergraduate degree in 1942 and her master’s degree in 1954. She continued to study and take courses until 1978. She retired in 1994 after a 40-year career.

Mrs. Berg has established a number of gift annuities with the University of Arizona Foundation and those annuities ultimately will create a number of special endowments in units across campus. “My gifts are not altogether charitable, as they benefit me until I am gone and then they will be used for the purpose I have specified,” she says.

As a tribute to her beloved parents and her cherished aunt, Mrs. Berg has established the Therese S. and Charles Velasco and Marie Schreiner Endowment for Heart Research.

The permanent fund will be a memorial to all three, all of whose lives ended due to stroke or heart disease. Mrs. Berg notes, “I care about this endowment because it will mean that all the good things possible through research will continue when I am gone and other families will be helped.”
With each summer comes the departure and arrival of doctors training in the Sarver Heart Center’s cardiology fellowship program.

We wish the best of luck to the three fellows who just completed the three-year training and have left the Sarver Heart Center to pursue fellowships in interventional cardiology: Abdul Memon, MD (Arizona Heart Institute); Alex Vasquez, MD (Minnesota Heart Institute); and Lyndon Xavier, MD (Arizona Heart Institute).

We welcome the five who just began the program: Gurpreet Baweja, MD; Jon Brower, MD; David Cragun, MD; Michelle Dew, MD; and Frank Molls, MD.

They join continuing fellows Raymond Huang, MD, Ming-He Huang, MD, Brian Mitchell, MD, Ranjan Dahiya, MD, and Bao Do, MD.

And we say “welcome back!” to three others: Kathryn Bates, DO, a past cardiology fellow who is returning for a one-year fellowship in cardiac imaging; Raghu Kamineni, MD, who just completed the cardiology fellowship and now is in a one-year fellowship in interventional cardiology; and Julia Indik, a former fellow in cardiology and electrophysiology who has joined the faculty of the UA College of Medicine as an assistant professor of medicine. ♥
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SHC Establishes Northern Advisory Board

With more and more friendships developing in the central and northern parts of Arizona, the Sarver Heart Center has established a Northern Advisory Board to help guide fund-raising and public education efforts beyond Southern Arizona.

The group of volunteer leaders has been assembled to increase the representation of the entire state and to foster better relationships and a more significant presence in Maricopa County, Flagstaff, Prescott and other northern communities.

Jacquelyn Michelson, a member of the primary SHC Advisory Board and a longtime resident and volunteer leader in Maricopa County, will chair the Northern Advisory Board.

She will spend the next several months identifying and recruiting board members who will be helpful in supporting the statewide mission of the Sarver Heart Center.

“Having lost both my parents to heart disease as a teen-ager, finding a cure for the number one killer of men and women in America is of vital importance to me as I assume this leadership role as chair of the northern board,” Michelson says.

For information about how to get involved, please call (520) 626-4146.

New Equipment to Benefit Research in Heart Failure

A critical instrument used in a lab that studies heart failure has been replaced thanks to donations to the Sarver Heart Center’s equipment fund.

The research team, led by Douglas F. Larson, PhD, relies on the use of a spectrophotometer to conduct its investigations into heart failure, the mechanisms of the aging heart and viral infections of the heart. The team’s previous spectrophotometer was in disrepair and could not be fixed due to lack of parts.

A spectrophotometer is used to analyze tissues at the molecular level, using color to describe what is taking place in the sample. Dr. Larson’s lab uses the equipment to gather information about growth and immune factors and the rate of cellular growth.

The equipment also will benefit research in cardiac resuscitation.

Tammy Struiksma, RN, won the women’s division of the Tour of the Tucson Mountains bicycle race this summer, setting a course record at the same time.

Struiksma, a senior research coordinator for Paul Fenster, MD, crossed the finish line of the 58-mile race at 2:10:13. The previous women’s record was 2:12:38.

It was the first time that Struiksma, a triathlete, had ever won a race.

Struiksma, who began training for triathlons in 1997, set her sights on winning the race back in 1998 when she saw its unique copper trophy inside a bicycle shop.

“I asked the guy at the counter, ‘Where did you get that?’ He said, ‘Well, you have to win El Tour de Tucson or Tour of the Tucson Mountains to get the trophy,’” Struiksma recalls. “I secretly said to myself, ‘I want one of those.’ And five years later I got one!”

Struiksma attributes her win in part to the fact that she followed the advice she gave a friend at the starting line: Go out hard and don’t stop even if you think you can’t hang on.

“Once I crossed the finish line, I just couldn’t believe I had won.”

Tammy Struiksma recently accepted a position with skin care company Medimorphosis.
SHC Research Grants, Awards and Honors

Gordon A. Ewy, MD, has been nominated as an International Health Professional of the Year by the International Biographic Centre of Cambridge, England.

Dr. Ewy was one of a small group of people chosen from thousands of biographies.

Dr. Ewy is chief of cardiology at the University of Arizona College of Medicine and director of the Sarver Heart Center.

The following awards were given this summer in recognition of service and contributions during the 2002-03 academic year.

The Charles W. Hall Jr. Memorial Award, in recognition of outstanding medical residents, selected by cardiology faculty:
- Leona Downey, MD
- Sarah Ferguson, MD

The Faculty Awards, selected by cardiology fellows:
- Teaching: Sam Butman, MD
- Mentoring: Steve Goldman, MD

The Zenas B. Noon Award for Excellence in Cardiology, in recognition of an outstanding medical student, selected by cardiology faculty:
- Arlynne Eisner

The following grants were awarded to Sarver Heart Center researchers this summer. Presentations of the research results will be given in the future.

Inflammation-induced thrombosis in mechanical circulatory support device patients
Pei H. Tsau, MD

Effect of treatment with DITPA and captopril on myocardial gene expression in the rat post-infarction model of heart failure
Cynthia Adamson, PhD

Development of an in vitro model to study α-opioid agonist mediated cardioprotection
Eva Varga, PhD

Plasma brain natriuretic peptide level as a new determinant in predicting success of restoring normal sinus rhythm in heart failure patients with new onset atrial fibrillation
Raymond Huang, MD

Molecular mechanisms of trichloroethylene-induced heart malformations
Ornella Selmin, PhD

Green Valley

Green Valley Speaker Series
Desert Hills Social Center, 2980 S. Camino del Sol
All lectures begin at 10 a.m.

Oct. 3 • Oct. 31 • Dec. 5 • Jan. 9 • Feb. 6 • March 5

Tucson

Healthy Heart public education conference
University Medical Center, 1501 N. Campbell Ave.
Feb. 14, 2004 • 9 a.m. - 3 p.m.

For more information, please call Pila Martinez at (520) 626-4083
UPDATE

Over 425 Donors Contribute to Chair Honoring Copeland

Thank you for your gifts toward completion of The Jack G. Copeland, MD, Endowed Chair in Cardiothoracic Surgery!

The Sarver Heart Center announced in the Spring 2003 Newsletter that patients, friends and colleagues of Dr. Copeland established an endowed chair in his name to recognize his 25 years as a leader in cardiothoracic surgery. A minimum of $2 million is needed to fund the chair.

To date, 425 families have contributed the following:

- **Immediate gifts**: $397,604
- **Pledged gifts**: $596,500
- **Planned gifts**: $189,000
- **Total**: $1,183,104

Please note that all donors will be invited to a special gala celebration on Friday, Nov. 7, to mark the completion of the $2 million goal.

For more information, call the office of Development at (520) 626-4146.

To see past issues of the Sarver Heart Center Newsletter, please visit our web site at www.heart.arizona.edu. You’ll also find information about cardiovascular disease risk factors, your healthy weight range, exercise tips and more.

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New SHC Members

- Gurpreet Baweja, MD  
  Fellow, Cardiology
- Jon Brower, MD  
  Fellow, Cardiology
- Katharine Burleson, MD  
  Fellow, Integrative Medicine
- David Cragun, MD  
  Fellow, Cardiology
- Michelle Dew, MD  
  Fellow, Cardiology
- Frank Molls, MD  
  Fellow, Cardiology
- Daniel T. Redford, MD  
  Clinical Anesthesia
- Mark Sharon, MD  
  Cardiology  
  Southern Arizona VA Health Care System
- Christy Smith, MD  
  Fellow, Cardiovascular and Thoracic Surgery
- Vincent Sorrell, MD  
  Cardiology
- Jack Wilmore, PhD  
  Professor Emeritus  
  Texas A&M
Perhaps the most pleasant administrative task of my position as director is the appointment of an outstanding individual to an endowed chair. And so I am very pleased to announce that Paul McDonagh, PhD, will occupy The Allan C. Hudson and Helen Lovaas Endowed Chair of Vascular Biology and Coagulation.

As outlined on the front page of this issue, Dr. McDonagh’s research interests will have a major impact on the interdisciplinary research collaboration and research training efforts of the Sarver Heart Center. He will attract young investigators to work in his and other laboratories to help solve some of the important scientific and medical questions of the day. These questions involve the role of inflammation and coagulation, contributors to atherosclerotic cardiovascular disease, and the major cause of heart attacks and strokes.

One significant area in which he will contribute is the study of cardiovascular complications in diabetes. This is an important subgroup to study, as diabetics are at especially high risk. While 40 percent of Americans will die of cardiovascular disease, 75 percent of diabetics will die of cardiovascular disease.

Dr. McDonagh’s work also will focus on improving the safety of artificial heart assist devices and artificial hearts.

And last but certainly not least, he will continue to play a strong role in training the cardiologists, cardiovascular surgeons and research scientists of tomorrow.

We are delighted to have Dr. McDonagh fill this leadership role in the Sarver Heart Center.

Sincerely,

Gordon A. Ewy, MD
Director, UA Sarver Heart Center