

Heart & Health Reports™

Your trusted source for prevention, treatment, fitness and nutrition

November/December 2008

Vol 10/No 6

Many U.S. Leaders Afflicted

Heart Disease & The Presidency

THE election of Barack Obama signifies a new era in the history of the American Presidency. Political pundits are already comparing the incoming president to former Presidents Kennedy, Clinton, FDR and others. But it would serve all Americans if President Obama's term in office could avoid an affliction that has struck so many of our former leaders, namely cardiovascular disease.

More than just a historical curiosity, presidential illness can provide a dramatic illustration of the ravages of untreated hypertension, elevated cholesterol, obesity, and tobacco abuse. Some chief executives have set an example of health and fitness. More often, however, the medical histories of our 20th century presidents reveal a spectrum of cardiovascular risk factors and their consequences.

Theodore Roosevelt (26th President, 1901-1909).

Many think of Theodore Roosevelt as giant of a man, permanently affixed to Mount Rushmore



FDR was one of many U.S. presidents who suffered from serious cardiovascular illness.

with other American greats. In actuality, Roosevelt began life diminutive and sickly, his early years wracked by severe bouts of asthma and chronic gastrointestinal complaints. Instead of giving in to his ailments and small stature, Roosevelt became a devotee of regular and vigorous exercise. In his senior year at Harvard, he was 5 feet 8 inches tall and weighed 136 pounds. But his size belied his strength and stamina, and that summer he scaled the 15,000 foot Matterhorn. By the age of 38, Roosevelt was said to have the "strength of an ox, quick in movement and tough in endurance."

Roosevelt's emphasis on health and fitness continued after his election to the presidency. He set aside two hours each day for strenuous exercise and smoking was prohibited in the White House. In 1912, when Roosevelt was running for a third term, he survived an attempt

on his life, an assassin's bullet striking him in the chest. The bullet lodged in a rib and Roosevelt delivered his campaign speech as scheduled, stating he felt as "strong as a bull moose."

Roosevelt's health declined after leaving the presidency. In the winter of 1918, Roosevelt suffered from severe mastoiditis that required surgery at a New York hospital. At the same time, "inflammatory rheumatism" limited his activity. This likely predisposed him to the pulmonary embolism that complicated his hospitalization. While recuperating at home, he developed difficulty breathing, and later that evening, Roosevelt was found dead in bed. The cause of death was listed as a blood clot that detached from a vein and entered the lungs (deep vein thrombosis and recurrent pulmonary embolism). Roosevelt was 60 years old.

William Howard Taft (27th President, 1909-1913)

The size of the man who succeeded Theodore Roosevelt was in stark contrast to his predecessor. The diminutive Roosevelt was replaced by the corpulent Taft, who carried 350 pounds on a frame just less than 6 feet. His obesity undoubtedly contributed to the severe sleep apnea he suffered during his term. On many occasions Taft was seen to doze off during affairs of state, often snoring heavily. Taft had high blood pressure, commonly associated with sleep apnea, with a systolic pressure of 210 mmHg recorded in 1910.

(turn to page 4)

Inside This Issue

Our final issue	2
The Jupiter trial: An important new cholesterol study	3
If you take Ranexa	6
New food pyramid buying guide	7

Heart & Health Reports™

Editors

Franklin H. Zimmerman, MD
*Assistant Clinical Professor of Medicine
Columbia University*

Arthur E. Fass, MD
*Chief of Cardiology
Phelps Memorial Hospital, Sleepy Hollow, NY*

Dina R. Katz, MD
*Attending Cardiologist
Westchester Medical Center*

Medical Advisory Board
John A. Ambrose, MD
*Chief of Cardiology
University Medical Center, Fresno, CA*

William E. Boden, MD
*Chief of Cardiovascular Medicine
University of Buffalo*

Jeffrey L. Boone, MD
*Fmr. Director, Stress Medicine & Hypertension
The Cooper Clinic*

Edwin E. Ferguson, MD
*Professor of Medicine
University of Wisconsin*

Barry A. Franklin, PhD
*Professor of Physiology
Wayne State University*

William H. Frishman, MD
*Professor of Medicine and Chairman
New York Medical College*

Robert N. Levin, MD
*Director, Coronary Care Unit
William Beaumont Hospital, Royal Oak, MI*

Marvin Moser, MD
*Clinical Professor of Medicine
Yale University*

Mehmet Oz, MD
*Director, Cardiovascular Institute
Columbia Presbyterian Medical Center*

Richard J. Solomon, MD
*Professor of Medicine
University of Vermont*

David G. Wolinsky, MD
*Director of Cardiac Rehabilitation
St. Peter's Hospital, Albany, NY*

Contributors

Stefanie Schwartz, RD
Roberta Gershner, RD

Publisher

Laura Grant

HEART & HEALTH REPORTS (ISSN 1532-0243) is published 6 times a year by National Heart Communications LLC for the Heart and Health Education Foundation, 465 North State Road, Briarcliff Manor, NY 10510-1468. **Postmaster:** Send address changes to Heart & Health Reports, 465 North State Road, Briarcliff Manor, NY 10510-1468. **Subscriber Services:** Subscription Prices: US \$24.95; Single issues \$7; Foreign; \$49 plus shipping. Customer Service: **To subscribe:** call 1-914-762-5810, M-F 9am-5pm EST. Multiple copies are available at reduced rates. Occasionally we make our mailing list available to carefully selected parties. If you prefer not to receive such material, please write to us and enclose your mailing label. The material published in HEART & HEALTH REPORTS is provided for educational purposes only and is not a substitute for personal medical treatment by a health care professional. It may not reflect continuing research, or information from other sources. Responsibility is disclaimed for any loss incurred from the use of the material published herein including typographical errors. HEART & HEALTH REPORTS does not endorse any specific company or product. Expenses may be met by unrestricted educational grants and fees. Opinions expressed are not necessarily those of the editorial board or of National Heart Communications. Any reproduction or other use of the material published herein without written permission is forbidden. © 2008 National Heart Communications.

2008 Heart & Health Reports Index

Topic	Issue, page #	
Airplane emergencies	Mar/Apr, 1	HDL raisingJan/Feb, 1
American medicine at a crossroads	Mar/Apr, 2	Holiday heart attacks . . .Nov/Dec, 8
Aneurysm options	Jan/Feb, 3	HomocysteineMay/June, 3
Angina options	Jul/Aug, 3	Hyperbaric therapyMar/Apr, 1
Antibiotic prophylaxis	May/June, 8	Hypertension in elderly . .Jul/Aug, 3
Aquatherapy	Mar/Apr, 8	HytrinJan/Feb, 6
Atrial fibrillation	May/June, 8	Incidental CT findings . .May/June, 3
Ballpark foods		Juice products
buying guide	Jul/Aug, 7	buying guideMay/June, 7
Bariatric surgery	Jul/Aug, 1	Jupiter trialNov/Dec, 3
Beta blockers and lung disease	Jan/Feb, 8	Living to 100Jul/Aug, 2
Bystolic	May/June, 6	Off label prescribing . . .Sep/Oct, 2
Caffeine products		Peripheral vascular diseaseJul/Aug, 1
buying guide	Mar/Apr, 7	Presidential healthNov/Dec, 1
Cardiac rehabilitation	Sep/Oct, 1	Prostate health and poor sleepJan/Feb, 1
Catapres	Sep/Oct, 6	RanexaNov/Dec, 6
Cognitive decline		Red yeast riceJul/Aug, 8
post bypass surgery	Jul/Aug, 8	Sodium lossSep/Oct, 1
Constipation and heart medications	Mar/Apr, 8	Surgery in patients with coronary stents . .Sep/Oct, 3
Coronary syndromes	Jan/Feb, 3	TekturnaJul/Aug, 6
Diabetes cardiac risk	Mar/Apr, 3	Tim RussertMay/June, 2
Digoxin toxicity	May/June, 1	Vitamin D buying guide . .Jan/Feb, 7
Electrocardiography	May/June, 1	Vytorin studyJan/Feb, 2
Enlarged heart causes	Sep/Oct, 8	Web watchSep/Oct, 2
Food pyramid		Winter heart attacks . . .Jan/Feb, 8
buying guide	Nov/Dec, 7	ZebetaMar/Apr, 6
Halloween treats		
buying guide	Sep/Oct, 7	

A fond farewell and thank you

We regret to inform you that this will be the final issue of Heart & Health Reports in printed format. The current economy has affected us all, particularly a nonprofit entity like the Heart and Health Education Foundation, the parent organization of this publication.

For the past ten years, it has been our privilege to publish this newsletter. We have tried to provide you with the very best news and analysis of leading cardiovascular topics of the day. We are proud of our accomplishments and the awards we have won in recognition of our work.

We would like to thank you, our loyal readers, who have made suggestions that have improved the publication. Indeed, it has been most gratifying to answer the questions you have sent us. This input has also given us the best ideas for topics to write about. We would like to think that our publication has been unique, with subjects that interest our readers written from a perspective derived from many years of treating real patients.

We hope to continue our efforts on the Internet. All subscriptions end with this issue. We hope to contact you again about some exciting special projects. For now, we wish you a fond farewell and the best of health.

Franklin H. Zimmerman, MD

New horizons in preventive cardiology: The Jupiter trial

During the last half century, there has been an impressive decline in coronary disease and stroke mortality in the United States. While some of this improvement can be attributed to the use of procedures such as coronary bypass surgery, most of the change can be attributed to a population-wide effort to lower risk.

The public is now better educated in the need for regular exercise and weight control in disease prevention. There is greater awareness of the requirements of a healthy diet, and a willingness on the part of food processors and restaurant chains to modify their offerings in favor of less atherogenic fare.

For its part, the medical community has also placed a strong emphasis on risk factor modification. This has resulted in considerable success in lowering smoking rates, as well as improved diagnosis and treatment of high blood pressure. Perhaps the most important recent advance in coronary prevention, however, is a better understanding of the role of cholesterol in the genesis of vascular disease, and the availability of remarkable tools for managing its effects.

We have known for many years that there is a direct relationship between blood cholesterol levels and the risk of coronary artery disease. The risk resides mainly in the LDL cholesterol fraction, while HDL cholesterol is protective. The “statin” class of medications offered a potent new option for lowering LDL cholesterol in a relatively safe fashion. In addition, these agents appear to exert a variety of other beneficial effects including anti-inflammatory actions.

Numerous studies, beginning in the 1980’s have confirmed the protective effects of these drugs both in

patients with established vascular disease and healthy patients with elevated cholesterol levels. Given the unique advantages of these agents, could the benefits of “statins” be extended to patients who don’t meet the usual criteria for their use? This hypothesis was tested in a recently published study known as the Jupiter trial.

Over 17,000 apparently healthy men and women with LDL cholesterol levels of less than 130 mg/dl were chosen for the study. These patients would not have met the usual guidelines for statin therapy because their LDL cholesterol levels were in the normal range. All patients in the trial, however, had relatively high levels of C-reactive protein, a marker of inflammation felt to confer a higher risk of coronary events. Half the patients were treated with rosuvastatin (*Crestor*) 20 mg, the remainder received placebo.

The results were striking. Although the trial was planned to span five years, it was stopped after less than two years because of a decided advantage in the rosuvastatin treated patients. Rosuvastatin reduced LDL cholesterol levels by 50% and C-reactive protein levels by 37%. There were substantial decreases in heart attacks, strokes, and death from cardiovascular causes in the treatment group. Hospitalizations and revascularization procedures were reduced in the rosuvastatin group. Moreover, deaths from all causes were lower in the active treatment patients.

The medication was well tolerated with few side effects. There was no increased risk of myopathy (muscle damage) or liver injury with rosuvastatin compared to placebo. There was no decline in kidney function in the treatment group. There was a slightly increased frequency of physician-reported diabetes in the treated patients, but fasting blood glucose

levels were not different. The significance of this finding is unclear.

Analysis: Virtually all patients who have had a vascular event such as a heart attack are now treated with a statin. These patients understand the need for “aggressive prevention.” The decision is more difficult for an apparently healthy individual. Many healthy patients are reluctant to take long-term pharmacologic therapy for a problem that may never occur. The results of the Jupiter trial, however, proving the effectiveness and safety of the intervention, argue for treatment in many of these patients. Certainly, patients who have persistently elevated LDL cholesterol levels warrant treatment. Others with risk factors such as a strong family history of premature coronary disease are candidates for a statin. Measurement of the C-reactive protein might decide the issue in some cases.

In our experience, after overcoming initial reluctance, patients who start treatment are generally pleased about taking a more active role in disease prevention. This feeling is reinforced when blood testing shows a dramatic improvement in cholesterol levels.

— Arthur E. Fass, MD

Ridker PM, et. al. Rosuvastatin to Prevent Vascular Events in Men and Women with Elevated C-Reactive Protein. N Engl J Med 2008;359:2195-2207.

Presidential Health . . . *continued from page 1*

Eight years after leaving office, Taft was appointed chief justice of the United States. Although he lost eighty pounds, his health continued to decline with additional cardiovascular illnesses. Taft developed atrial fibrillation and was treated with digitalis. The former president developed “hardening of the arteries” with exertional angina, breathlessness and rising blood pressure. He complained of a progressively poor memory, even forgetting some of the lines from the oath of office that he administered at the inauguration of incoming president Herbert Hoover. Shortly before his death at the age of 73, Taft resigned from the Supreme Court, his doctors citing the former president’s “general arteriosclerosis and myocarditis.”

Woodrow Wilson (28th President, 1915-1921)

No American president endured as devastating an illness in office as Wilson, when he was stricken with a disabling stroke in October of 1919.

Wilson’s neurological history begins many years earlier. In May of 1906, Wilson awoke and found that he was blind in his left eye, the result of a retinal hemorrhage. He consulted two prominent Philadelphia physicians, who stated that he had arteriosclerosis and should live a “quiet and retired life.” Wilson’s ensuing political career contrasted with these recommendations. He developed high blood pressure and suffered from recurrent headaches. From 1906 to 1915, he experienced multiple episodes of transitory weakness and numbness of his right hand.

Wilson’s labors reached a pinnacle during World War I and its aftermath. He worked 18-hour days, developing the covenant for the League of Nations, simultaneously trying to overcome isolationist opposition in the U.S. Senate. In September of 1919, during a speaking trip to gain public support for

his efforts, Wilson suffered a headache, slurred speech and temporary left-sided weakness. He improved the next day, but cancelled his engagements and returned to Washington.

On the morning of October 2, Mrs. Wilson found her husband sprawled on the bathroom floor. His attack paralyzed his left arm and leg, leaving him unable to manage the duties of the presidency. Remarkably, the public was kept unaware of the president’s condition, with press releases stating that Wilson was suffering from “nervous exhaustion.” After several months, the president recovered to the point of walking with assistance. But Wilson remained physically frail, and for the remainder of his term, he was mentally and emotionally impaired.

Warren G. Harding (29th President, 1921-1923)

The administration of Warren Harding was a failure on many different levels. Scandals, both personal and political characterized his presidency. Perhaps Mrs. Harding best predicted the future president’s term before she became First Lady when she said, “I’ve seen the inside of the White House. The office is killing Wilson as surely as if he had been stabbed at his desk. Don’t ask Warren to run.” This proved prescient as ultimately, it was cardiac failure that caused Harding’s death while in office.

Harding had multiple risk factors for heart disease and showed signs of cardiovascular illness not long after taking office. He had high blood pressure, with systolic readings of 185 mmHg. He was overweight and used tobacco in all forms, including cigarettes, cigars, pipes and chewing tobacco. He suffered from depression. Harding became easily exhausted and periodically complained of chest and abdominal pain. His personal physician, Dr. Charles Sawyer, stated the

president’s symptoms were due to “indigestion.” In the fall of 1922, noted New York heart specialist, Dr. Emmanuel Liebman, met Harding at a dinner party, privately telling friends afterwards that the president would be dead of coronary disease within six months.

In the summer of 1923 Harding embarked on a trip to the west coast. During that trip he became increasingly exhausted and had difficulty sleeping unless propped up on pillows. He could not complete even nine holes of golf, complaining of extreme fatigue and shortness of breath. All were characteristic symptoms of congestive heart failure.

On July 27, 1923, Harding complained of abdominal discomfort. Dr. Sawyer again made a gastrointestinal diagnosis, stating the president had eaten some bad crabmeat salad. The president continued on to San Francisco and was put to bed in his hotel after suffering recurrent abdominal symptoms. On the evening of August 2, Harding died suddenly in bed. Dr. Sawyer said the cause of death was “a stroke of apoplexy.” A more plausible explanation is that Harding suffered cardiac arrest associated with a prior myocardial infarction, recurrent coronary insufficiency and congestive heart failure.

Franklin D. Roosevelt (32nd President, 1933-1945)

Franklin D. Roosevelt’s battle with polio is familiar to many. Fewer are aware of his severe hypertension and congestive heart failure.

Roosevelt was diagnosed with high blood pressure at the age of 55. He was a heavy smoker, and numerous photographs displayed the president with his characteristic cigarette holder. Because he often suffered from sinusitis, Roosevelt selected as his personal physician an ENT specialist, Admiral Ross McIntire. The doctor’s unfamiliarity with

cardiac disorders (or his unwillingness to recognize them) would delay the president's inevitable diagnosis for years.

At the end of Roosevelt's third term, his health began to decline, primarily from complications of untreated hypertension. In March of 1944, a cardiac specialist, Howard G. Bruenn was called in consultation. The extent of the president's illness was published in 1970 in the *Annals of Internal Medicine*. Dr. Bruenn described Roosevelt as tired, cyanotic (dusky), and breathless. The blood pressure was 186/108 mmHg, with evidence of fluid in the lungs and an enlarged heart on chest x-ray. A blowing, systolic murmur was present at the apex of the heart (suggesting mitral insufficiency). The electrocardiogram was abnormal with diffuse T wave inversions (suggesting hypertensive heart disease).

Over the next number of months Roosevelt was prescribed phenobarbital, codeine and digitalis. He was to have periods of rest and a salt-restricted diet. Cigarettes were to be "curtailed." These rudimentary efforts proved futile and the president's health steadily declined with blood pressure readings as high as 260/150 mmHg. At the pivotal meeting with Churchill and Stalin at Yalta in February 1945, Roosevelt appeared so ill that Churchill's physician Lord Moran said privately, "I give him only a few months to live." These words proved prophetic.

On April 12, 1945, Roosevelt complained of a severe headache in the back of his head. Within 15 minutes he was unconscious, lapsing into a coma from a massive cerebral hemorrhage from which he would not recover. His blood pressure was 300/190 mmHg shortly before he expired at the age of 63.

Dwight D. Eisenhower (34th President, 1953-1961)

The public persona of the legendary WWII general and president was that of a vigorous, healthy man.

Eisenhower boasted of his avoidance of illness, stating at a 1956 press conference, "I have been one of those fortunate creatures of good health." The truth is far different, for Eisenhower suffered serious medical illnesses, both before and after he became president.

Beginning in 1920, Eisenhower had recurrent and often severe abdominal pain. He endured his symptoms throughout his military service and into the presidency. In 1956, the president developed a bowel obstruction, requiring urgent surgery. It was then that Eisenhower was diagnosed with regional ileitis (Crohn's Disease), the probable cause of his longstanding abdominal complaints.

Cardiovascular illness proved to be the president's paramount medical concern. Eisenhower was a heavy smoker, consuming up to 4 packs per day. He also had sporadic high blood pressure and elevated cholesterol. While vacationing in Denver in September of 1955, Eisenhower noted "indigestion" during a round of golf. The following evening, he complained of chest pain and summoned his personal physician, Dr. Howard Snyder. The doctor administered oxygen, a form of nitroglycerin (amyl nitrate), morphine, a blood thinner (heparin) and a vascular dilator (papaverine). The president's blood pressure fell dangerously. Inexplicably, Dr. Snyder did not recommend hospitalization for the stricken president, advising Eisenhower's wife, Mamie, to slip into bed to keep the president warm. The next day, a cardiac specialist was summoned and an electrocardiogram confirmed that the president had suffered a "massive infarct."

Eisenhower was hospitalized at a nearby Army facility. When news of the president's attack emerged, the Dow Jones index fell by 6 percent, with a paper loss of \$14 billion, the largest single daily decline recorded since the crash of 1929. Noted Boston cardiologist, Paul Dudley White was called in consultation. In

contrast to recommendations of the day, Dr. White advised early ambulation. Over time, examinations suggested Eisenhower had developed an aneurysm of the left ventricle.

Ten months into Eisenhower's second term he suffered a neurologic event. The president had difficulty holding his pen and could not speak coherently. Over the next 24 hours, his symptoms improved, but he remained with a minor impairment for the rest of his term.

In 1960, while campaigning for Richard Nixon, Eisenhower developed atrial fibrillation. He was treated with quinidine sulfate, but had recurrent cardiac arrhythmias over the next few months. After leaving the White House, Eisenhower developed congestive heart failure and had a series of heart attacks. In 1968, he succumbed to cardiac arrest at the age of 78.

Cardiologist's comment:

The president of the United States may very well have the most stressful job in the world. It is not surprising then, that the holder of that office is at risk for cardiovascular illness. Presidents Coolidge, Johnson, Clinton, and Ford have all been affected. Many presidents suffered major cardiac illness while in office. Others succumbed to complications of heart disease after leaving the White House.

The chief executive has access to the best medical care our country can provide. But until the past few decades, few options were available to prevent complications of cardiovascular disease. In some instances, our presidents have suffered the consequences of missed diagnoses and substandard care. These examples of history provide lessons for us all.

Contemporary presidents have had the benefit of wonderful advances in prevention and therapeutics. One can only speculate how history would be different if these treatments were available during earlier administrations.

— *Franklin H. Zimmerman, MD*

Ranexa

(Ranolazine)

What kind of medicine is Ranexa?

Ranexa is the first of a new class of medication used for angina. It may be used alone, or in combination with other anti-anginal medications such as beta blockers, calcium channel blockers and nitrates. Unlike other medications for angina, Ranexa does not affect the heart rate or blood pressure.

How does Ranexa work?

Ranexa works by a unique mechanism that affects the body's regulation of sodium and calcium entry into the cells of the heart muscle. By restoring the normal balance of sodium and calcium, Ranexa improves blood flow to the heart muscle that occurs during the relaxation phase of cardiac contraction.

How will Ranexa help me?

Patients with coronary disease often have symptoms of angina. This may feel like a tightness, burning, or pressure in the chest, arms, back or jaw. Shortness of breath and fatigue during exercise may also be symptoms of inadequate blood flow to the heart. Ranexa helps to prevent angina from occurring and may allow you to perform physical activity that might otherwise cause discomfort.

How is Ranexa prescribed?

The recommended starting dose is 500 mg, twice daily. The dose may be increased to 1000 mg, twice daily as needed based on clinical symptoms.

What dosage sizes are available?

Ranexa is available in 500 mg, and 1000 mg extended release tablets. They may be taken either with food or on an empty stomach. The pills should be swallowed whole, and should not be split or crushed.

How long will it take to work?

Stable blood levels are obtained within three days. The onset of action should be similar.

What if I miss a dose?

Take the missed dose as soon as possible. Never try to "catch up" by taking an extra tablet.

Do food or other drugs affect this medicine?

Consuming large amounts of grapefruit products may increase the blood level of Ranexa. Limit the dose of Ranexa to 500 mg, twice daily. Otherwise, Ranexa is not usually affected by food.

Ranexa may interact with other medications.

- Ranexa levels may be increased with use of protease inhibitors (HIV drugs), ketoconazole and clarithromycin (*Biaxin*). Do not prescribe Ranexa in combination with these medications.

- Ranexa levels may be modestly increased with combined treatment with diltiazem, verapamil and erythromycin. Limit the dose of Ranexa to 500 mg, twice daily.
- Ranexa may increase the levels of digoxin and tricyclic antidepressants. The dose of these medicines may need to be reduced.
- Ranexa may raise blood levels of simvastatin. The dose of simvastatin may not need to be reduced, but use caution.

Who should use caution or not take this medicine?

- Pregnant women — there are no adequate studies to determine safety.
- Nursing mothers — it is not known whether Ranexa enters breast milk.
- Ranexa should not be used in patients with severe liver disease.

How will I feel while taking Ranexa?

Ranexa is usually well-tolerated. The most common mild, expected side effects include dizziness, constipation, nausea and headache.

Are there serious side effects to watch for?

Ranexa can cause an abnormal electrocardiogram (prolonged QTc) that may theoretically predispose you to serious abnormal heart rhythms. This has not been seen clinically. Inform your doctor if you have heart palpitations or fainting spells.

How does the doctor monitor my progress?

The doctor will inquire about your anginal symptoms and periodically perform an EKG and routine labs.

Do you have any special tips for me?

- Ranexa will not stop an acute attack of angina. Always carry nitroglycerin with you.
- Ranexa has a unique property that improves levels of glycosylated hemoglobin (Hb A1c). The clinical significance is unknown and Ranexa should not be considered a treatment for diabetes.
- Ranexa does not affect the heart rate or blood pressure and may be used in patients with all stages of congestive heart failure.
- For more information on the Internet, visit: www.Ranexa.com.

Ranexa® is a trademark of CV Therapeutics.

— Franklin H. Zimmerman, MD

The New Food Pyramid

THE Food Guide Pyramid, developed in 1992, is a tool used to translate nutrition recommendations into a food selection guide. The basic triangular guide is designed with the bottom portion filled with starch, the middle section meats and dairy, and the top fruits and vegetables. The number of recommended servings from the various food groups decreases as you move from bottom to top. Times have changed and so has the food guide pyramid. Over the years, nutrition professionals have realized that a “one size fits all” approach doesn’t work. The modern approach to healthy eating is a more customized strategy to help guide individuals through the basics of sound nutrition.

The new food guide pyramid is called “MyPyramid” and acknowledges that people of different ages, genders, sizes, and physical activity levels all have different nutritional needs. The new guide incorporates recommendations from the Dietary Guidelines for Americans, which was released in 2005 by the U.S. Department of Agriculture. It encourages variety and balance with respect to meal planning and physical activity.

Several new themes are included in the new recommendations. These include:

- **Moderation.** This is represented by the narrowing of each food group from bottom to top. The wider base stands for healthy foods with little or no solid fats, added sugars or caloric sweeteners. You should choose these foods most often to derive the most nutrition from the calories you consume.
- **Variety.** You should choose foods from all groups represented by the six colored bands.
- **Personalization.** Everyone’s needs are different and can be determined using an interactive tool.
- **Proportionality.** The different width of each band suggests the proportion from each group you should choose, relative to each other.
- **Physical activity.** The figure climbing the steps is a reminder to incorporate physical activity into your nutritional program.

- **Gradual improvement.** This is represented by the slogan “Steps to a Healthier You” and reminds you that even small steps can improve your lifestyle and can lead to greater gains.

The current pyramid expands the previous four food groups into six, represented by the six vertical stripes. These include grains, vegetables, fruits, milk products, meat and beans, and oils. The width of the stripes signifies the relative amount one should consume from each group. MyPyramid also emphasizes the importance of daily physical activity. Current recommendations suggest 30-60 minutes of moderate to vigorous physical activity on most days of the week.

To acquire an individualized profile, go to www.mypyramid.org and enter your age, gender, and physical activity level. For example, if you indicate that you are a 35 year-old male who is moderately

active, your recommended calorie level is 2000. These 2000 calories are distributed among the food groups as follows: 6 ounces of grains; 2 ½ cups of vegetables; 2 cups of fruits; 3 cups of milk; and 5 ½ ounces of meat or beans. In addition to “MyPyramid Plan,” there are a host of interactive activities that offer a customized approach to balanced nutrition. For example, MyPyramid Tracker provides you with a detailed assessment of your current eating and physical activity

habits. The tool will ask you to enter a daily food and activity log, allowing you to compare your habits to those suggested by current dietary guidelines.

MyPyramid is part of an overall food guidance program based on recommendations that connect diet and good health. Its user-friendly, interactive tools allow for a personalized approach to good nutrition right at your fingertips.

— Emily Kratz, MS, RD

Ms. Kratz is an outpatient nutrition coordinator at Phelps Memorial Hospital, Sleepy Hollow, NY.



