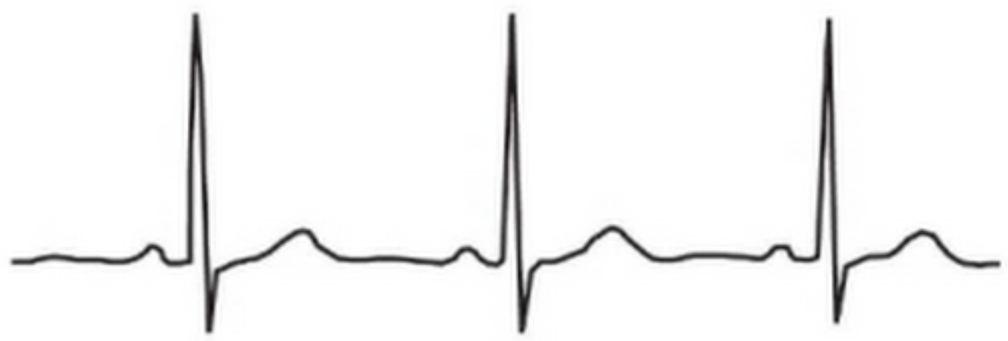
Heart 411

THE ONLY GUIDE TO HEART HEALTH YOU'LL EVER NEED



* The medical tests you need (and those to avoid!) * Choosing between stents, surgery, and pills * The truth about red wine * How your emotions affect your heart * Facts and fictions about heart-healthy eating

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The Only Guide to
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This book contains general information and advice relating to heart disease. It is not intended to replace personalized medical advice and should be used to inform patients and supplement the regular care of a physician. We strongly recommend that you consult with your doctor about questions and concerns specific to your heart health. The authors and publisher expressly disclaim responsibility for any adverse effects that may result from the use or application of the information contained in this book.

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CONTENTS

HEART 411

SECTION I

HEART HEALTH AND DISEASE: THE BASICS THAT YOU MUST KNOW

CHAPTER 1

SAVING YOUR LIFE

"Why me?"

We've heard those words thousands of times, repeated again and again by our heart patients over the years.

Lying in the hospital bed staring at the white ceiling of his cubicle in the Cleveland Clinic coronary care unit, the forty-eight-year-old man didn't have time for a heart attack. He needed to finish a few things at work before leaving to take his son on a whirlwind college tour, planning to hit seven schools in nine days. He had a schedule, but another wave of chest pain brought him back to today's reality. Looking with dread at his already thick hospital chart with our names—Nissen and Gillinov—emblazoned on the spine, he realized that he was having a heart attack. His plans with his son would have to wait. We had a radically different plan for him.

Had he been able to peer around the corner from his room, he would have seen us, his cardiologist and heart surgeon, conferring. The coronary care unit (CCU) is where we meet. Twenty feet from the bedside, we studied a computer screen that displayed images of the patient's coronary arteries.

Steve and I easily identified the problem—severe blockages of all three coronary arteries, meaning a trip to the operating room was imperative.

Sleep-deprived and responsible for a twenty-four-bed ICU filled with every manner of complex heart patient, Steve was clearly pleased to have a quick disposition in this case. The treatment plan was clear, and success was almost certain. He could move on to the next bed, which served as a temporary home to yet another patient with a life-threatening cardiac condition.

Meanwhile, I needed to figure out how to fi t another surgery into an already full OR schedule. Making my way past the busy nurses, technicians, and doctors, I called the operating room and told them to cancel a scheduled elective surgery to make room for the emergency. The nurses had an OR ready, and the anesthesia team would be down to pick him up in fifteen minutes.

The bypass surgery went well, and our patient was discharged from the hospital in six days with a good prognosis. We never learned what happened with his son's college trip. By the time the forty-eight-year-old heart attack victim left the hospital, Steve had seen fifty new patients in the CCU, and I had operated on ten more. Together, we focused on that case for a grand total of only a few hours and then moved on to the next one. It was a monumental event for our patient and his family, but for us, it was a routine part of a hectic day on the cardiac service at the Cleveland Clinic.

Between the two of us, working separately or together, we have cared for more than 10,000 cardiac patients. Over the last twenty years we have successfully made use of EKGs, stress tests, and cardiac catheterizations to diagnose heart problems, and medicines, angioplasty, and heart surgery to treat the diseases we have discovered. Along the way, we have helped thousands of patients and saved thousands of lives. We have basked in the warm and fl attering glow created by successful procedures and grateful families. The rewards of a career in heart medicine have been tremendous.

But over time, we have come to recognize a key limitation to the way we practice medicine. Our experience with the forty-eight-year-old heart attack victim plays out over and over. We hurry from one patient to the next, doing our best to treat their diseased hearts. But we see our patients too late, after they already have heart disease or established risk factors for developing it—toxic diets, abandoned exercise programs, dangerous supplements, ill-advised combinations of prescription medicines, and failure to manage emotional stress are among the notations we record in patients' charts again and again. There seems no end to the flow of patients who

require complex procedures to fix their hearts—not to mention the ones who show up year after year for another bypass operation, or one more stent. Like the mythological Sisyphus condemned to spend eternity pushing a boulder up a mountain only to have it roll back down every night, we feel the frustration associated with trying to prevent a monumental, recurring problem. But unlike Sisyphus, as doctors, we have the opportunity to achieve success.

The solution to our dilemma becomes clear when we revisit the classic ideal of the physician from generations past. You know the type: the kindly, unhurried, gray-haired gentleman with a white coat, a black bag, and a stethoscope. Decades ago, doctors could not imagine peering into the body with real-time, three-dimensional MRI scanners or dream of preventing heart attacks by propping open the heart's two-millimeter-wide arteries with tiny metallic stents. With limited technology, what did these physicians do? *They communicated with their patients.*

Doctors of old took the time to listen and to talk. When they came to a hospital bed, they saw whole patients, often surrounded by generations of people whom they had also treated. In today's whirlwind of technological marvels and breakthrough cures, these aspects of medicine have all but disappeared. We heart doctors tend to "fix" the plumbing problem of the moment and then move on rapidly to the next one. All too often, patients become "cases" ("Can you check on the 80 percent left main coronary artery obstruction in cath lab number 4?") rather than people in desperate need of advice and counseling.

The fact that we are missing an important piece of the puzzle really hit home a few years ago as we prepared to do some cardiac plumbing on our fourth patient from a single family. Periodically, we treat two or three members of the same family, and we have even performed his-and-hers heart surgeries for married couples. But the Whelton family's cardiac problems were like an annuity for heart doctors. Their story forced us to reexamine our long-held assumptions of what it means to be a successful doctor.

THE WHELTONS: A FAMILY PLAN FOR HEART DISEASE

This time it was Jim Whelton's turn. Sitting in the consultation room with Jim were his father, Sam (triple bypass surgery, 1998), brother, Rick (quadruple bypass, 2007), sister Noreen (two coronary artery stents, 2005),

and sister Susan (no cardiac procedures to date). Their mother had died of a heart attack four years earlier. When we entered the room, it was a warm reunion. Sam, Rick, and Noreen enthusiastically reported that they were all feeling great and doing well. Jim, on the other hand, did not look particularly thrilled: he was facing his turn with coronary artery bypass surgery.

We talked about the procedure, explaining how we would open his chest and reroute the blood flow around his heart's blocked arteries. With each detail, Jim lost a bit more color; he would soon blend in perfectly with the white hospital walls. His brother and father tried to cheer him up. They assured Jim that it would be a piece of cake. He would do fine.

And he did—technically, Jim's surgery was a success. But a conversation with his younger sister, Susan, the day before Jim left the hospital made us realize that it was also a failure. Susan cornered us in the hall outside Jim's room, fi dgety and obviously worried. Picking up on her distress, we assured her that everything was fine. Jim's EKG and echocardiogram revealed that his heart had weathered the surgery well, and he would be going home within twenty-four hours. For doctors, a patient's hospital discharge after heart surgery always represents a victory.

As Susan talked, though, we quickly realized that we had missed the point completely. She was glad Jim had done well, but *his* health wasn't what was keeping her up at night. She was the only member of the family who had not yet developed heart disease. Wasn't there some way to avoid it? Should she try coenzyme Q_{10}, chelation therapy, or one of the antioxidants she read about on the Internet? Maybe she should add blueberries, Cheerios, and POM Wonderful to her diet? Wouldn't these keep her arteries clean? At fi rst we were amazed. How could she not know that coronary heart disease is preventable? Wasn't everybody conversant with its risk factors, including diets too rich in calories and saturated fat, high blood pressure, elevated cholesterol, smoking, family history and all the other usual suspects? But then we turned the question back on ourselves. Where could she get the information she needed to avoid a trip to the cardiac cath lab or operating room? The answer was obvious: from us.

The next question followed quickly, though it was not one that we really wanted to dwell upon. We had been taking care of the Whelton family for more than a decade, seeing them through operations and writing endless prescriptions for aspirin, Lipitor, and Plavix. We had talked to them at length about these how these medical therapies can help with coronary heart disease. But had we failed to give them the relevant information they

needed to manage their heart health and even prevent heart disease in the first place? Painfully, we concluded that our efforts had fallen short.

Susan and the rest of the Whelton family did not see us as failures. We had fixed their hearts! But our follow-through was too limited, and our plumbing efforts came too late, at a stage when they already had severe cardiac problems that required high-tech and invasive solutions.

Susan wanted information, not an operation. Realizing that we could not tell her everything she needed to know in a three-minute discussion outside her brother's hospital room, she asked us to point her to the best website. Googling "heart disease" the night before had presented her with more health information than is contained in all of the world's medical libraries put together. Surely one of these sites had it all, including plans for prevention for her and disease management for her family. We promised to look into this and get back to her.

We checked, and checked again, and found no such resource. The volume of available material was tremendous, but no single site or book contained all of the critical information. Many websites contained recommendations that were shockingly wrong and even dangerous, touting ultra-low-fat diets and unregulated supplements that promised to make artery-clogging plaque melt away. Sensational headlines and unrealistic promises abounded, but sound advice was elusive. So we decided to provide that advice. That's the reason we wrote this book.

EVIDENCE-BASED ANSWERS VERSUS UNFILTERED INFORMATION

As a society, we crave medical information. Eight out of ten Internet users search the Web for health information. We are hungry for the facts and for a to-do list that will make us healthy. But most available information is unvalidated and unsorted. Even when the information is correct, how can you determine how to use it? A website may describe a new medical study extolling the virtues of aspirin for heart attack prevention and conclude that an aspirin a day is generally good for most people. But are you "most people"? Do *you* need an aspirin a day?

Adding to the confusion and anxiety, pharmaceutical-company-sponsored Internet sites try to brand medical conditions and convince people they are suffering from them. We're all familiar with this approach. An

animation shows blood clots forming in the heart. Ringing with authority, a deep voice proclaims, "This could be happening in *your* chest right now!" The solution—run out and "ask your doctor" about taking Plavix. As a consequence of this tactic and the huge volume of information on the Web, we have developed a new medical condition, cyberchondria—hypochondria fostered by too much time spent on medical websites. Search any symptom and you will wade through medical jargon and conflicting recommendations, encountering nightmare scenarios involving people who seemingly had exactly the same problem as you.

Susan Whelton, her family, and just about all of us—both heart patients and those who want to avoid becoming heart patients—need reliable information we can act on. The challenge of filling this gap created an opportunity to expand our definition of what it means to be a good doctor. It gave us a chance to move beyond fixing cardiac plumbing that is already broken to arming people with the critical information that will enable them to care for their hearts and perhaps avoid seeing us altogether.

We approached this project much the same way that we manage a patient with heart disease. Medical decision making should be evidence-based. When we treat a patient, we choose only those therapies that are supported by the scientific data. But such rigor is lacking in much of the medical content on the Internet or the overflowing shelves in the health section of your local bookstore. Weekly health headlines skim the surface of the medical ocean—take fish oil, pass on heart scans, throw away your aspirin—but what do the studies *really* say? What is the message for you and your heart? We decided to explore the science behind the headlines and to explain clearly but completely the evidence supporting our plan for your heart health.

WHY NOW?

The threat has never been more dire, the need for action never so urgent. After decades of progress, a new tidal wave of heart disease is building. Look around you. Fast food and supersized meals have replaced healthy choices and appropriate portions. Video games and iPods have edged out exercise as sources of entertainment. Smoking remains stubbornly entrenched, and our waistlines are expanding at an alarming rate. Today two-thirds of us are overweight or obese, and nearly everybody harbors one or more risk factors for heart disease.

The statistics concerning cardiovascular disease in general and heart disease in particular are staggering. Eighty-two million American adults have cardiovascular disease, a broad group of disorders that includes coronary heart disease, stroke, high blood pressure, heart failure, and cholesterol abnormalities. Each year, 800,000 Americans will have a new heart attack, while 500,000 will suffer a second (or third, or fourth) one. This amounts to a heart attack every twenty-five seconds. When we combine the two most serious cardiovascular problems—coronary heart disease and stroke—we find that these conditions account for one of every three deaths in the United States. If we could eliminate all forms of cardiovascular disease, life expectancy would increase by seven years. In comparison, eliminating cancer would add only three years.

When it comes to heart disease, ignorance and complacency are the enemies. Countless recent media reports reiterated an important but potentially misleading statistic from the American Heart Association: "From 1997 to 2007, the death rate from cardiovascular disease declined 27.8 percent." Good news, but how does that help the million-plus people who will have a heart attack this year and the 900,000 who will die from cardiovascular disease? We can be gratified by the progress, but we should not be satisfied. When we are operating on somebody's heart, we don't relax when we have merely slowed the bleeding. We keep working until we have it completely stopped.

People affected by heart disease come from all strata of society, from celebrities such as Robin Williams, Barbara Walters, David Letterman, and Bill Clinton to teachers, firefighters, athletes, and even heart doctors. It is the proverbial elephant in the living room: heart disease can attack anybody—young and old, male and female—and its prevention must begin early.

Therefore, one of our key initiatives addresses the causes of cardiovascular disease. Heart disease is not like breast cancer and prostate cancer, which often seem to strike unfairly and indiscriminately. We know what causes heart disease, and you do, too: high blood pressure, smoking, elevated cholesterol, diabetes, obesity, and family history. Except for family history, each of these factors is modifiable—meaning that to a very great extent, you can control it. Right now we are doing a poor job of managing our risk factors. Much of the problem stems from what seems to be our society's motto: eat more, exercise less.

This society-wide increase in cardiac risk factors is shifting the ground beneath us, preparing to create a tsunami of cardiovascular disease. The

number of cardiovascular operations and procedures done annually—attempts to fix the plumbing damaged by our unhealthy lifestyles—has increased from 5.4 million to 6.8 million over the last decade. Our society can't afford to pay for this. The economy and health care budgets are buckling under the strain of a $167 billion annual price tag to treat cardiovascular disease and an additional $119 billion in lost productivity caused by the illness. If we don't make headway, these costs will triple by 2020, dragging down the economic well-being of every American.

The solutions are not all that complicated. We know how to prevent cardiovascular disease and better treat existing heart problems. The government cannot solve the problem with legislation, and your employer can't fix it by removing soda from the vending machines at work. Solutions must begin on an individual level—with you. Once you learn how to help yourself, you can rescue your family and friends from a future diminished by cardiovascular disease.

CARDIOVASCULAR DISEASE IN MUMMIES

Atherosclerosis, or hardening of the arteries, is pervasive today, but disease-causing, cholesterol-filled plaques have actually been around for a long, long time. Scientists recently reported the results of high-tech, whole-body CT scanning on twenty-two mummies in the Museum of Egyptian Antiquities in Cairo, Egypt. While the "patients" were not waiting for their test results, the medical community was curious. In fact, the results generated so much interest that they were reported in the *Journal of the American Medical Association*, a leading medical journal, and a few days later in the *New York Times*.

Of sixteen mummies who lived between 1981 BCE and 334 BCE, the CT scans showed that nine had either definite or probable atherosclerosis. The most ancient mummy with arterial disease was Lady Rai, nursemaid to Queen Nefertari, who died in approximately 1530 BCE. One of the study authors quipped, "She went in a relic. She came out a patient."

This intriguing study demonstrates that the history of cardiovascular disease extends thousands of years into the past. Our plan is to limit its impact on our future.

What if you have moved beyond the risk factor stage and already have coronary heart disease? We have critically important information for you, too. There is good news from the front lines for the millions of Americans who have existing cardiovascular problems. In addition to increasing our

understanding of the risk factors for heart disease, our pills and procedures for diagnosing and treating it have never been better. But there is a catch—you must ensure that you receive the *right* treatments.

When it comes to medicines for high blood pressure and high cholesterol, strategies to open blocked arteries and urgent measures to manage heart attacks, the difference between an okay treatment and the best treatment can mean the difference between life and death. Coronary heart disease and its management are not mysterious. The obstacle faced by Susan Whelton and millions of others is simply their lack of good information. Which brings us back to our mission.

MAKING A PLAN

Whether you are young or old, male or female, already a parent or planning to start a family, you need to understand how to keep your heart and your family's hearts healthy. Our plan to ensure your heart health is simple. We will cover it all—from how factors such as cholesterol and genetics cause coronary heart disease to the role of high-tech procedures such as CT scans and even heart transplants. Explaining the science and the studies behind today's headlines, we will provide you with the tools you need to judge for yourself the next time the media report on "a startling new medical breakthrough." You will be able to distinguish fact from fiction and understand what the new information means for your health. Armed with the right information and sound, scientifically validated strategies, you, the intelligent and motivated reader, can grow from a passive patient into an active partner in your heart health.

CHAPTER 2

CORONARY HEART DISEASE: THE RISK FACTORS YOU KNOW AND THOSE YOU DON'T

THE BIG PICTURE

Coronary heart disease (also called coronary artery disease or coronary atherosclerosis) is characterized by cholesterol-filled plaques that block arteries and can cause chest pain (angina) or heart attacks. As with every medical condition, the development of coronary heart disease depends upon the presence of predisposing conditions or behaviors, known as risk factors. The more risk factors that you have, the greater the likelihood that you will end up in our offi ce with chest pain or a heart attack. On the other hand, if you recognize these risk factors and reduce them as much as possible, you may never have to meet us professionally.

While some basic risk factors are beyond your control—advancing age, a family history of heart disease—others are completely up to you. Most people are aware of the usual suspects leading to coronary heart disease: high blood pressure, diabetes, obesity, tobacco use, lack of exercise, and abnormal cholesterol levels. Managing these risk factors can go a long way toward reducing your risk of developing heart disease.

In the last few years, scientists have extended our understanding of the

genesis of heart disease. We now recognize a host of factors and conditions with previously unsuspected links to heart disease, including inflammatory diseases (such as rheumatoid arthritis), migraine headaches, and even living near a highway. In this chapter, we will look at heart disease from this wider perspective.

IT'S YOUR HEART, NOT YOUR ARTHRITIS

Sally Robinson was no stranger to doctors. Now fifty-one years old, she had been seeing her rheumatologist regularly for nearly thirty years, as he constantly adjusted Sally's medicines to treat the rheumatoid arthritis that threatened to take control of her life. For the most part, Dr. Frazier had been successful. Although constant low-grade knee pain made it hard to exercise, Sally could do almost anything she wanted and was only a few pounds overweight. Sally took her arthritis medicines religiously and suffered few side effects, although the steroids she needed to take caused slight elevations in her blood pressure and blood sugar.

One Sunday afternoon, Sally developed pain in her back and left shoulder, which she blamed on her arthritis. Although shoulder pain was new for Sally, she just increased her arthritis medications, which seemed to work. During the next week, the pain in the front of her left shoulder waxed and waned. By Friday, it was such a nuisance that she finally called Dr. Frazier, who gave her an appointment for the following Monday and advised her to take it easy over the weekend. When Dr. Frazier saw Sally, he pushed and prodded her shoulder but could not reproduce the pain. During his standard examination of Sally's lungs, Dr. Frazier suddenly grew serious. He told Sally that he wanted to get a chest X-ray to check things out, and walked her down the hallway to the radiology suite. Nervously, Sally watched as Dr. Frazier silently examined the chest X-ray and then asked his assistant to perform an EKG.

Dr. Frazier finally told Sally that he had heard "crackles" when listening to her lungs. These crackles, which sound like Rice Krispies when you pour in the milk, indicated a possible buildup of fluid in Sally's lungs, and the chest X-ray confirmed his suspicion. Although rheumatoid arthritis *can* cause lung problems, the combination of her left shoulder pain and the new lung findings pointed toward a problem with Sally's heart. Comparing her new EKG to a previous one done years earlier, Dr. Frazier saw changes indicating that Sally had suffered a heart attack, probably beginning at the onset of her left shoulder pain.

Sally was stunned. She believed that only older women, not those her age, developed heart disease. When Sally came to us to treat her heart problems, we told her that young women *can* develop heart disease, especially when they have risk factors. We explained that people with rheumatoid arthritis, which is characterized by whole-body inflammation, actually face an increased risk of coronary heart disease. Had Sally known this earlier, she might not have ignored her shoulder pain.

Fortunately, Sally's heart was not severely impaired. The damage was minor, and her cardiac catheterization showed that only a single small artery was blocked. She did not need surgery or stents—but she did require aggressive measures to manage her other risk factors, including her slightly elevated blood pressure and blood sugar and her extra weight. Today, Sally remains healthy and active, and neither her joints nor her heart slows her down.

HOW CORONARY HEART DISEASE BECAME OUR NUMBER ONE HEALTH PROBLEM

Coronary heart disease (CHD) currently looms as the greatest health threat to Americans. The rise of coronary heart disease to the top of our medical "to-do list" is relatively recent. In 1900, pneumonia was the leading cause of death in the United States, and the average life expectancy was only forty-seven. During the first half of the twentieth century, doctors and scientists focused on treating infectious diseases—for example, developing new drugs to cure pneumonia and virtually eradicate tuberculosis. These dramatic advances enabled people to live longer—and inadvertently opened the door to coronary heart disease.

By 1930, average life expectancy in America had risen to about sixty, and heart disease had become the number one cause of death. These statistics refl ect an important feature of CHD: the incidence of the disease increases strikingly with age. Longer life means more time for arterial plaques to develop and cause problems. The risk of an eighty-five-year-old man having a heart attack is twenty-five times that of a forty-five-year-old.

Longer life does not by itself cause CHD, but the combination of longer life and damaging lifestyles increases the risk of developing coronary heart disease. Contemporary lifestyles have created a minefield of risk factors for CHD. Liberated from the grip of infectious diseases, too many

of us fi ll our extra years of life with smoking, eating, and many excuses not to exercise.

THE BATTLE FOR YOUR ARTERIES' HEALTH: THE GENESIS OF CHD

Before we examine the risk factors for coronary heart disease, let's focus on your arteries and the mechanisms by which plaques form. The process begins with damage to the endothelium, a smooth, tile-like layer of specialized cells lining the inner walls of blood vessels through which blood flows on its way to our organs. More than simply a watertight seal to keep the blood inside the artery, the endothelial lining is biologically active, producing chemicals that prevent blood from clotting at its surface. In addition, the endothelium acts as a barrier to prevent toxic substances from entering the blood vessel's wall.

Many of the risk factors for CHD initiate and accelerate disease by damaging the endothelium. Smoking and air pollution increase levels of carbon monoxide and other toxic chemicals in the blood, triggering chemical reactions that assault and damage the endothelium. High blood pressure causes the blood to act like a battering ram, attacking and disrupting the endothelium.

Over the last few years, scientists have recognized that inflammation also damages endothelial cells and contributes to plaque formation. The term *inflammation* is derived from the Latin meaning "to set on fire." Inflammation is not always bad; it actually represents the body's normal response to injury and infection, and in the appropriate setting it restores health. But when it occurs inside blood vessels, inflammation can initiate plaque formation.

Regardless of the source of injury, when the endothelium is damaged, LDL cholesterol can breach its defenses and enter the artery wall. When LDL molecules oxidize—that is, when they combine with oxygen in the blood—they become particularly hazardous. (See page 45 to learn more about LDL cholesterol.) As oxidized LDL makes its way past the damaged endothelium and enters the artery wall, the body misinterprets the event and responds as it would to an infection, sending white blood cells called macrophages to the area. The macrophages ingest the oxidized LDL, which augments the infl ammatory reaction and causes more damage to the endothelial lining. The cycle continues, and over time, the collection

of white blood cells, cholesterol, and inflammatory proteins forms a large plaque within the artery wall.

Breaking this cycle by removing the sources of inflammation and endothelial damage allows the artery to heal, and we have the tools to make this happen. Statins can help by reducing the concentration of LDL, and they also inhibit infl ammation. Treating high blood pressure prevents further damage to the endothelium. Quitting smoking lowers blood levels of carbon monoxide and other harmful chemicals, preventing these noxious substances from damaging the endothelium.

The battle for your arteries' health begins early in life. Plaques form in the walls of the coronary arteries over the years, initially causing no signs or symptoms to signal their presence. Although people rarely suffer heart attacks in their twenties and thirties, the plaques are already present. Autopsies performed on young soldiers who died during the Korean and Vietnam wars revealed early plaque buildup in many of their arteries. Similar studies by Cleveland Clinic physicians uncovered plaques in the coronary arteries of young trauma victims—by age thirty, more than half of them had measurable atherosclerotic plaques in their coronary arteries.

Does this mean that we should go looking for these early plaques in young people? Probably not. Detecting coronary plaques during their silent phase can be very diffi cult. Some physicians advocate a test known as calcium scanning to try to detect the early development of CHD, but there are many downsides to this procedure (including radiation exposure and false positive results), and we don't recommend it. Instead, we believe that the best approach is to gather intelligence by screening patients for the risk factors that cause CHD and then aggressively treating those risk factors to prevent the disease from developing or progressing.

Notice that many of the most important risk factors for coronary heart disease are modifi able, meaning they are under your control. Several recent studies suggest that up to 90 percent of CHD could be prevented by addressing these treatable risk factors! The most important modifiable risk factors are increased cholesterol, high blood pressure, smoking, diabetes, abdominal obesity, and absence of regular physical activity.

Risk Factors for Development of Coronary Heart Disease

RISK FACTOR	STRENGTH OF EVIDENCE FOR AN ASSOCIATION*	CAN YOU CHANGE IT?
The Usual Suspects		
Family history	++++	No
Advanced age	++++	No
High blood pressure	++++	Yes
Diabetes	++++	Yes†
Cholesterol/lipid abnormalities	++++	Yes
Tobacco use	++++	Yes
Obesity	+++	Yes
More Recently Identified Risk Factors		
Inflammatory diseases		
Rheumatoid arthritis	+++	No
Psoriasis	++	No
Lupus	++	No
Sleep apnea	++	Yes
Periodontal/gum disease	++	Yes
Air pollution	++	Yes
Emotional stress	++	Yes
Migraine headaches	+	No

*++++ Conclusive evidence
+++ Strong evidence
++ Moderate evidence
+ Weak evidence
† Type 2 diabetes can be treated with lifestyle changes in many patients

THE RISK FACTORS YOU KNOW

Cholesterol

We cover the problem of high cholesterol and its treatment thoroughly in Chapter 3. Although the science is complicated, the key message is clear:

a combination of diet and drugs can treat high cholesterol in nearly all affected individuals. Yet despite the wide range of effective options available, we still see many patients with a first heart attack who didn't know they had an elevated cholesterol level. Some of these individuals had a strong family history of premature CHD and yet chose not to have a cholesterol test. We cannot emphasize enough: you *must* know your cholesterol levels.

Every adult should have a cholesterol test (lipid panel) in his or her twenties. If this initial blood test reveals normal cholesterol levels, repeat it every fi ve to ten years, because cholesterol tends to rise with age. Don't make the mistake of assuming that a normal lipid panel at age twenty-five comes with a fi fty-year guarantee. On the other hand, we don't agree with those pediatricians who advocate cholesterol screening in all children. We prefer a selective and thoughtful approach, testing children only if they suffer from obesity or have a strong family history of early CHD. Finding a high cholesterol level in a child or young adult does not necessarily lead to drug therapy, but it should always serve as a wake-up call leading to significant lifestyle changes. Achieving normal cholesterol levels dramatically reduces the likelihood of a first heart attack or stroke.

When discussing cholesterol with our patients, we focus on LDL cholesterol. As you'll learn in Chapter 3, the lower the LDL, the lower the risk of heart attack and stroke. There is no threshold or LDL value that is too low: like the old adage that you can be never be too rich or too thin, you can never have too low an LDL. We also measure HDL, which is *inversely* related to the risk of CHD—that is, the higher your HDL, the lower your risk of heart disease. However, the relationship between CHD and low levels of HDL is not as strong as the relationship with high LDL levels. Finally, triglycerides appear to be weakly associated with an increased risk of CHD, but the relationship is controversial because people with high triglycerides also tend to have low HDL, making it difficult to prove that triglyceride levels are the actual culprit. Because of the weaker evidence for relationships between CHD and HDL or triglycerides, these lipids are considered secondary, not primary, targets for treatment.

Some doctors routinely order advanced cholesterol tests that measure the levels of apolipoprotein B (also called ApoB), a protein associated with LDL, and apolipoprotein A (or ApoA), a protein associated with HDL. While some suggest that these measurements help to determine the risk of developing CHD, we don't find them particularly useful in most people. Similarly, fancy tests that measure the size of LDL particles have become very popular, but they are expensive and add little to the risk factor picture.

If you know your LDL, HDL, and total cholesterol levels, you have most of the information you need to accurately assess your risk for CHD and track your treatment progress.

High Blood Pressure

High blood pressure (hypertension) is a powerful and modifiable risk factor for development of CHD. We divide hypertension into two categories: primary and secondary. More than 90 percent of patients have primary hypertension, which means that we cannot identify a specific medical cause for the increased blood pressure. In the early years of the twentieth century, physicians thought it was normal for blood pressure to rise with aging, but now we know they were wrong. Hypertension isn't a normal part of aging, and it can be deadly.

Secondary hypertension accounts for only 5–10 percent of cases, but it is very important because in these patients the elevated blood pressure is caused by some other medical disorder. Treating the underlying condition can return the blood pressure to normal. Certain hormonal imbalances can cause secondary hypertension, but kidney problems are the most common culprit. When atherosclerotic plaques narrow kidney arteries, reducing blood fl ow to the kidneys, these organs attempt to counteract it by releasing a hormone that triggers blood vessels to tighten, thereby raising blood pressure. For cases of blocked kidney arteries, stenting or surgery can dramatically lower blood pressure. Rarely the kidney itself is abnormal, rather than its artery; in such cases, surgical removal of the affected kidney may be necessary to control blood pressure.

If you have high blood pressure, do you need to worry that it might be caused by an uncommon kidney issue or a rare hormonal imbalance? Probably not. But if your blood pressure is very high and multiple medicines fail to control it, you and your doctor should consider blood tests and scans to look further.

What is a normal blood pressure? For many years, physicians were taught that anything lower than 160/90 mm Hg was acceptable. However, careful randomized clinical trials demonstrated that this threshold was too high, so doctors adopted 140/90 as the cutoff for normal blood pressure. Alas, they were wrong again, as new evidence suggested that 140/90 is still too high, leaving people at increased risk for heart attack and stroke. To arrive at the truth, scientists studied non-industrialized populations,

where diets were low in salt and primarily vegetarian, and people were always active and not obese. Most of these people had a blood pressure less than 120/80, which remains our "normal" value today. If your blood pressure is between 120/80 and 140/90, you are considered prehypertensive. Although you probably don't need to begin taking blood-pressure-lowering medicines, you should lower your salt intake, follow a special diet (the DASH diet; see Chapter 5), and lose weight. With these steps, blood pressure often returns to normal.

The number of people with hypertension in developed countries is staggering and increases sharply as the population ages. According to the American Heart Association, more than 76 million Americans have high blood pressure. The rate of hypertension reaches 50 percent for individuals ages fi fty-five to sixty-four, rising to more than 70 percent by age seventy-five. The incidence of hypertension is substantially higher in certain subgroups, especially African Americans and people with diabetes, indicating the need for closer vigilance. Hypertension has even reached our schools, where obese children are developing high blood pressure at alarming rates.

A key challenge to lessening the toll from hypertension is early recognition. Like high cholesterol levels, hypertension generally manifests no symptoms until it causes a major problem such as a heart attack or stroke. You should know your blood pressure and ensure that every member of your family has his or her blood pressure checked. We have excellent medicines and strategies to treat high blood pressure, but we are less effective at repairing the damage it can cause.

While the relationship between blood pressure and the risk of heart attack is strong, the link between high blood pressure and stroke risk is even more dramatic. In both cases, the systolic pressure (the top number) exerts the strongest impact on the risk. High-quality randomized trials show that a five-point reduction in systolic blood pressure can reduce heart attacks by 15 to 20 percent and strokes by 25 to 30 percent. Current guidelines suggest reducing blood pressure to less than 140/90 for most patients and less than 130/80 for diabetics. However, the exact target levels for systolic blood pressure remain controversial, because many studies do not show benefi t from reducing blood pressure substantially below 140/90. Unlike LDL cholesterol, where there is no lower threshold for benefit (lower is always better), blood pressure reduction has a definite range in which benefit occurs. In fact, too low a blood pressure can actually harm certain patients. Fear of complications related to low blood pressure may be one explanation for the fi nding that physicians are frequently not aggressive enough, often

BLOOD PRESSURE AND THE BARBER

African American men have the highest death rate from hypertension of any group in the United States. Compared to a white man, an African American man faces a threefold increase in the risk of dying from hypertension. As scientists have examined this threatening situation, they have discovered that diagnosis and medical access are the key problems. This realization sent Texas researchers straight to the barbershop.

Because the barbershop presents a comfortable and nonthreatening environment, doctors reasoned that African American men might allow barbers to take their blood pressure and might follow their recommendations if the barbers informed them it was too high and a visit to the doctor was in order.

In a randomized, controlled clinical trial in seventeen black-owned barbershops in Dallas County, Texas, barbers were trained to take blood pressure readings, and screened their clients when they came in for a haircut. The barbers identified 1,300 men with hypertension. Half of the hypertensive men received pamphlets about blood pressure control while the other half received more intensive intervention, including blood pressure checks with every haircut and physician referrals when hypertension was detected.

The barbers did a great job. Average blood pressure fell in both groups, with a slightly larger decrease among those who had more intense screening and treatment. Next up: hairdressers for women.

tolerating unacceptably high blood pressure levels. The optimal blood pressure in the elderly remains controversial because overly aggressive lowering can cause adverse consequences.

Therapy for hypertension always starts with lifestyle modifications (reduced salt intake, exercise, and weight loss) but often progresses to drugs. For patients with severe hypertension (blood pressure greater than 160/100) many physicians will start with two drugs, because a single drug is unlikely to adequately lower blood pressure. You may not feel well when you start medical therapy, but don't be too quick to abandon your pills; as your body adjusts to a lower blood pressure, symptoms such as dizziness tend to resolve. And don't let yourself run out of your blood pressure medications. Suddenly stopping anti-hypertensive drugs can cause a sudden rebound in which blood pressure spikes to dangerous levels.

For patients with hypertension, purchasing an automated blood pressure

cuff is a great investment. These simple instruments typically cost less than $100 and provide accurate blood pressure measurements. Always take your blood pressure in the same position (usually sitting) after a few minutes of relaxation. Keep a log of your blood pressure and share it with your doctor.

Diabetes

Diabetes mellitus is an important potentially modifiable risk factor for CHD, but reducing the influence of diabetes on heart disease is not always simple.

Insulin is needed by all the body's tissues to metabolize sugar (glucose). In type 1 diabetes (formerly known as juvenile diabetes), which accounts for about 10 percent of cases, specialized cells in the pancreas fail to produce enough insulin. This can lead to an abrupt rise in blood glucose levels, which can be fatal if not recognized and treated promptly. Although people with the far more common form known as type 2 diabetes (previously called adult-onset diabetes) do have elevated blood sugar levels, their problem is not inadequate insulin production. Rather, their problem is insulin resistance—the pancreas produces sufficient insulin, but the body's tissues do not respond properly, allowing sugar to build up in the bloodstream. Although the pancreas initially compensates by producing more insulin, eventually, blood sugar rises. The current obesity epidemic in the United States and other developed countries has produced a corresponding explosion of type 2 diabetes.

Patients with insulin resistance who have not yet exhausted the ability of the pancreas to respond (by making more insulin) are considered "prediabetic." Remarkably, if the causes of insulin resistance—obesity and a sedentary lifestyle—are recognized and corrected early enough, full-blown diabetes can be prevented. In one study, a program of intensive weight loss and increased physical activity reduced the incidence of type 2 diabetes by more than two-thirds, while the medication metformin reduced diabetes incidence by only about one-third. The earlier that weight loss is initiated, the more likely this strategy will prevent the development of type 2 diabetes. Even in patients who already have the disease, weight loss brings their blood glucose levels under better control and may enable them to reduce the amount of medication required or even eliminate the need for insulin injections. If recognized early, the equation for prevention of type 2 diabetes is clear:

WEIGHT LOSS = DIABETES PREVENTION

Diabetes affects nearly every organ in the body, from the kidneys to the eyes to the heart. Patients with either type of diabetes are much more likely to develop coronary heart disease than their non-diabetic counterparts. The increase in CHD risk for diabetics depends upon the duration of the diabetes (longer is worse), with most studies indicating that long-term diabetics face an approximate doubling of the rate of CHD. Heart attack or stroke is the cause of death in approximately 65 percent of diabetics.

GASTRIC BYPASS CAN REVERSE DIABETES

Gastric bypass surgery is proven to cause substantial weight loss in extremely obese people. Now the International Diabetes Federation advocates this therapy for certain obese diabetics. When obese diabetic patients undergo gastric bypass surgery, blood sugar often begins to drop within hours to days of the operation, well before actual weight loss sets in. In some cases, patients requiring 100 units of insulin per day no longer require any insulin at all by the time of hospital discharge. While the precise mechanism for this rapid benefit is not completely clear, it appears to relate to changes in blood levels of certain chemicals produced by the intestine. This therapy shows promise, but physicians advise caution, favoring weight loss through lifestyle changes as the initial step to try to reverse type 2 diabetes.

How does diabetes cause or accelerate CHD? We don't yet completely understand the precise mechanisms, but we have identified several factors that play important roles. Patients with diabetes typically have lower levels of HDL and higher triglyceride levels than non-diabetics. In addition, as we discussed above, a very strong association exists between diabetes and hypertension. Diabetics also have increased blood levels of markers of inflammation, suggesting an important role for inflammation in their CHD.

It would be natural to assume that good control of blood sugar would lower the risk of CHD in diabetics, but it turns out that this is only a small part of the answer. We assess long-term blood sugar control by using a blood test called HbA1c (commonly called A1c), which measures how much of the blood's hemoglobin contains glucose. Normal HbA1c levels

are less than 6 percent, and diabetes is usually defined as a level greater than 6.5 percent. There is a moderate relationship between the A1c level and the risk of developing coronary heart disease, but it's not nearly as strong as the relationships for other common risk factors, such as LDL cholesterol or high blood pressure. Lowering blood sugar levels only modestly reduces the risk of CHD. But good glucose control is still very important, because it reduces the risk of other diabetic complications such as kidney failure, nerve damage, and blindness.

If controlling blood sugar is not the key to CHD prevention in diabetics, what are the most effective strategies? High-quality trials demonstrate that blood pressure control has a dramatic effect on rates of CHD in diabetics. In fact, the national guidelines for hypertension set a more aggressive target for blood pressure levels in diabetics (130/80) compared with nondiabetics (140/90). Use of statins to lower LDL cholesterol also confers substantial benefi t in diabetics, even when they have normal LDL levels. Therefore, many practitioners believe that *all* patients with diabetes, regardless of cholesterol level, should take a statin.

Despite all our knowledge and corresponding treatment targets, study after study demonstrates that most diabetics don't achieve optimal LDL, blood pressure, and glucose levels. Know your goals and partner closely with your doctor to achieve these targets. For most people with diabetes, these are reasonable targets:

• LDL cholesterol	100 mg/dL or less
• Blood pressure	Lower than 130/80
• A1c	7.0–7.5 percent or lower

Smoking

Despite more than fi fty years of warnings, smoking remains a depressingly common cause of CHD. Make no mistake: people who smoke double their risk of developing coronary heart disease and cut their life expectancy by an average of eight to eleven years. Smoking exhibits a dose-response effect: the more cigarettes you smoke and the more years you smoke, the greater your risk.

Smoking causes heart disease by multiple mechanisms, but the most important factor appears to be damage to the endothelium that lines the insides of our arteries. Remember, endothelial cells protect against the

entry of cholesterol into the blood vessel wall. Carbon monoxide and other chemicals in tobacco smoke damage the endothelium, breaking the barrier and enabling plaque-forming oxidized LDL to penetrate the arteries. The effect of tobacco is so powerful that even exposure to secondhand smoke by living or working near a smoker elevates the risk of developing CHD. And don't fool yourself into thinking that smokeless tobacco represents a safe alternative; it too increases the risk of coronary heart disease.

Despite these well-known risks, 50 million American adults (21 percent of the population) continue to smoke. These sorry statistics actually represent progress. The number of smokers increased dramatically during and immediately after World War II, reaching a peak of 42 percent of the population in 1965. We have cut the percentage of smokers in half, but we still have a long way to go.

People continue to smoke, despite extensive health warnings and graphic images of what smoking can do, because nicotine is addictive. Within seconds of taking a drag on a cigarette, nicotine reaches the smoker's brain, triggering a cascade of chemical reactions that produce relaxation and euphoria. Addiction experts place nicotine's addictive strength in the same class as illicit drugs such as cocaine and heroin. As with those drugs, addiction has economic implications, meaning that there is money to be made from selling it. Consequently, tobacco companies have a history of manipulating the nicotine content of cigarettes to promote and maintain addiction.

We see the powerful and tragic effects of tobacco addiction in our daily practice. Almost every cardiologist has had some version of this poignant exchange when interviewing a patient the day after a heart attack:

> *Physician:* "Do you smoke?"
> PATIENT: "No."
>
> *Physician:* "Did you ever smoke?"
> PATIENT: "Yes."
>
> *Physician:* "When did you quit?"
> PATIENT: "Last night."

With the crushing pain and terror of the heart attack fresh in his memory, the patient has decided to quit, convincing himself that he is no longer a smoker. But in many cases, this situation changes shortly after hospital discharge. We see many patients who resume smoking soon after returning home from a hospital stay, even after suffering a heart attack or undergoing

heart surgery. You might expect that someone who has just experienced the discomfort and stress of open heart surgery would be scared straight, frightened by the very idea of resuming smoking. Unfortunately, the power of the addiction drives people to light up "just one more" cigarette. We do our best to help them understand that the stakes are high, while the benefits of quitting are real and occur quickly.

The cardiac damage caused by smoking does not need to be permanent. Remarkably, the risk of developing coronary heart disease in smokers declines quickly once a patient stops smoking. About half of the excess CHD risk disappears within one year of quitting, and the risk continues to fall over time; after ten years of tobacco abstinence, the ex-smoker has a risk virtually identical to that of a non-smoker. However, as any former (or trying-to-be-former) smoker will tell you, quitting is extremely difficult. As Mark Twain said, "Quitting smoking is easy. I've done it hundreds of times."

Today's smoker has more options available to help than did Twain. While we recognize that people are different and no single strategy works for everyone, one strategy is nearly always doomed to failure: resolving to cut down gradually. Studies demonstrate that nearly all smokers can relatively easily reduce the number of cigarettes they consume, and at first blush that fi nding sounds promising. However, when measuring the urine concentration of nicotine breakdown products, scientists found that they actually remain constant in patients even as they reduce their cigarette consumption. How can this be? The question was answered when researchers realized that smokers who cut down on the number of cigarettes they smoked actually worked harder to get more out of each one: they inhaled more deeply, held the smoke in their lungs longer, and smoked more of the length of each cigarette, unconsciously maximizing their nicotine intake from each cigarette.

Many people need a little pharmacologic help to quit smoking, and we think that is just fi ne. Nicotine replacement therapy using gum or skin patches signifi cantly increases your chances of quitting. Once you stop smoking, it is usually not too difficult to gradually reduce the use of the nicotine gum or patches. A new drug called varenicline (Chantix) also helps some patients. With chemical effects similar to those of nicotine, varenicline reduces the craving for cigarettes. Studies confirm its effectiveness at helping motivated patients to quit. However, the drug is controversial because it causes some patients to experience serious psychological side effects, including anxiety, anger, and suicidal thoughts. The antidepressant

bupropion, rebranded as a smoking cessation drug (Zyban), also helps some patients quit. However, bupropion can increase blood pressure and may also cause behavioral changes and, rarely, seizures. Regardless of whether or not you use these drugs or therapies to help yourself quit, it is essential that you keep trying. Successful quitters often report many failed attempts before achieving long-term success.

Obesity and Its Friends: Metabolic Syndrome

While daily news reports warn that obesity (particularly abdominal obesity) is the single most important modifiable risk factor for CHD, the real threat probably comes from the bad company that obesity keeps. People with abdominal obesity frequently develop a constellation of risk factors that includes high blood pressure, low HDL cholesterol, elevated triglycerides, diabetes, and increased waist circumference. If a person has three or more of these risk factors in tandem, he or she is said to have metabolic syndrome. This is a controversial subject among physicians, with some doctors claiming that it is nothing more than a cluster of individual risk factors. Other doctors contend that these factors are synergistic in their impact on the development of coronary heart disease, meaning that their combination poses a greater threat to the patient than the sum of the individual conditions.

Why should increased waist circumference be a criterion for metabolic syndrome? Isn't all fat equally bad? When it comes to obesity, most scientific data show differences in health risks for patients with the "apple" and "pear" body shapes. People who carry their extra weight around the stomach (apple shape) face a greater risk of developing heart disease than do those people with large buttocks and thighs (pear shape). Although some new studies have cast doubt on this distinction, asserting that all body fat, no matter where it is distributed, is equally dangerous, we believe that such a distinction exists. Fat cells that accumulate in the abdomen are metabolically more active than those in other parts of the body, causing insulin resistance (the hallmark of type 2 diabetes) and producing substances that increase infl ammation. This explains why women with large thighs (a pear shape) but not a particularly large waistline are not as likely to develop CHD as women with an apple shape. To their misfortune, men tend to gain weight in their abdomen, the pattern more strongly associated with CHD.

If you carry excess pounds—no matter where they reside—we urge you to follow a sensible weight loss plan. Most fad diets don't work in the long term. A program of moderate calorie restriction coupled with exercise remains the best strategy for sustained weight loss.

Age

Although you can modify most of the classic CHD risk factors, you can't turn back the clock and change your age. The incidence of coronary heart disease increases with age. As we noted earlier, heart attacks rarely occur in men younger than thirty-five or women younger than forty-five. For both genders, heart risk increases steeply with age, but women have a lower overall incidence of heart disease than do men—that is, until menopause, after which they slowly catch up with their male counterparts. You don't need a blood test or the latest scan to assess this risk factor. Keep your heart healthy at all ages, and be especially vigilant as you reach middle age.

Family History

Family history remains one of the most important non-modifiable factors to assess your risk of developing coronary heart disease. If either of your parents or a sibling developed CHD before age fifty-five, your risk of CHD is increased approximately one and a half to two times. This increased risk associated with family history is independent of other risk factors, such as smoking, diabetes, cholesterol, or hypertension.

The precise link between family history and CHD is a subject of intense scientifi c investigation. There is no single "heart disease gene," although many genes appear to contribute to its development. If you have a strong family history of CHD, stay tuned, as we may someday have a genetic test that screens for the genes that contribute to CHD.

Our advice for individuals with a strong family history of heart disease is always the same: do everything possible to reduce the risk factors that you *can* modify. We recommend more aggressive preventive treatment in patients with a strong family history—for example, favoring the use of statins in patients who might otherwise be considered borderline candidates for cholesterol-lowering treatments. With vigilance and proper management, a family history of heart disease is not a death sentence. While

there may not be anything you can do about the red hair or freckles you inherited from your father's side of the family, you *can* signifi cantly alter your prognosis for heart health.

THE NEW KIDS ON THE BLOCK: RISK FACTORS YOU MAY NOT KNOW

Rheumatoid Arthritis and Coronary Heart Disease

We know that inflammation contributes to the development of artery-blocking plaque. Therefore, we are not surprised that scientists have established links between a variety of inflammatory diseases and coronary heart disease, including rheumatoid arthritis, psoriasis, inflammatory bowel disease, certain muscle disorders, and systemic lupus erythematosus. Of these, we have the strongest evidence supporting and explaining the association between rheumatoid arthritis and coronary heart disease.

Rheumatoid arthritis develops when the body makes a mistake and the immune system attacks the joints and surrounding tissues, causing inflammation, pain, and limited mobility. The disease usually strikes people over forty, and women are affected more often than men. More than 1 million Americans currently suffer from rheumatoid arthritis. Although these patients and their doctors focus on the complex medical management of their painful joints, they must not forget their hearts.

Like Sally Robinson, whom you met earlier in this chapter, people with rheumatoid arthritis face an increased risk of cardiovascular disease, including strokes and heart attacks. While scientists used to think that this risk became apparent only after years of fighting the disease, new research shows that increased heart and cardiovascular problems occur early: the risk of heart attack increases by 50 percent within one year of the diagnosis of rheumatoid arthritis. Because people often develop rheumatoid arthritis in their forties, cardiac vigilance must start early.

How do rheumatoid arthritis and other inflammatory conditions affect heart health? We don't know the precise mechanisms, but the link between infl ammation and blood vessel damage is strong. The inflammation associated with rheumatoid arthritis causes the release of proteins and activated cells into the blood; these can damage the inner lining of arteries and contribute to plaque formation.

What should the person with rheumatoid arthritis do about her heart?

We have two concrete steps to take: manage your other cardiac risk factors and be vigilant about any indicators of potential heart problems. Johns Hopkins researchers have suggested that the increased risk of heart problems is greatest in those patients who have both rheumatoid arthritis *and* traditional risk factors. Don't let yourself gain weight. Put down the salt-shaker. Don't smoke. Take your statin as prescribed. Ask your physical therapist for an exercise program compatible with your joints and your heart. And if you do develop symptoms that could be from your heart—pain in the chest, shoulder, neck, or back, shortness of breath, new fatigue—don't assume it's your arthritis.

Brush Your Teeth

Dentists (and parents) remind us to brush and floss at least twice a day. A clean mouth is a healthy mouth. If you take care of your teeth, you can avoid the high-pitched whine of the dental drill. But if you listen to your dentist, you'll enjoy an additional benefit—good dental hygiene may also help keep you out of our cardiac catheterization laboratory and operating room.

Large observational studies suggest that inflammation and infection of the gums increase the risk of developing coronary heart disease by 20 to 40 percent. About 25 percent of Americans have some periodontal (gum) disease, while 1 percent, or 3 million people, have severe inflammation and infection of the gums. As with most cardiac risk factors, the worse the gum disease, the greater the cardiac toll. In one widely publicized observational study, people who rarely or never brushed their teeth faced a 70 percent increase in their risk of suffering a heart attack or other serious cardiac event over an eight-year period. We suspect they also had really bad breath.

As with rheumatoid arthritis, inflammation is the likely link between gum disease and arterial disease. Gum disease is actually the most common chronic infl ammatory condition in the world. In an interesting study using positron emission tomography scans to detect inflammation, Harvard researchers found that when the mouth "lit up" on the scan, the carotid arteries in the neck were also affected, indicating simultaneous inflammation in both parts of the body. Studies also demonstrate that people with periodontal disease have elevated blood levels of C-reactive protein, an indicator of infl ammation linked to coronary heart disease. Perhaps as a consequence of infl ammation, patients with gum disease display other un-

favorable cardiac characteristics, including abnormal function of arteries and an increased tendency for blood to clot.

In addition to increasing inflammation, periodontal disease tends to "travel" with other conventional risk factors. People with poor dental hygiene often smoke cigarettes, eat junk food, and avoid exercise. In such patients, gum problems are just one part of the heart-attack-causing package. Of course, fixing these unfavorable behaviors enhances heart health. But can you help your arteries simply through better dental hygiene?

A preliminary report published in the *New England Journal of Medicine* answers yes to this question. In that study, 120 patients with periodontitis were randomly assigned to receive either usual or intensive gum care. Patients in both groups ended the six-month study with better oral hygiene and reduced infl ammation in their bodies, but only those receiving intensive treatment enjoyed the added benefit of improved blood vessel function. The study's message is intriguing: treating gum disease may have a positive influence on your arteries.

Still, some scientists argue that we have not firmly established causality. Proof of a cause-and-effect relationship would require a study in which we randomly assigned people to twice-daily brushing and flossing or to no dental hygiene or care for a year or more. We don't think we would find too many people who would be anxious to sign up for this study, which also raises serious ethical issues. Based upon the data that we have right now, we think that the evidence is strong enough to follow the American Heart Association's recommendation: don't smoke, eat right, and brush your teeth. And while you are in the bathroom, don't forget to floss!

Hold Your Breath: Air Pollution and Your Heart

Scientists fi rst recognized the health risks of air pollution in the 1930s. Exhaust produced by cars and factories contains hundreds of potentially harmful substances. Most people fear that air pollution will hurt their lungs, but it turns out that their hearts also face an increased risk.

Fossil fuel combustion from traffic, industry, and power generation releases particles of different sizes into the air. The smallest among them—tiny, invisible particles less than 2.5 microns in diameter, just a fraction of the width of a human hair—seem to pose a cardiovascular risk. Observational studies have suggested an association between high concentrations of these particles and unfavorable cardiovascular changes.

IS GOING TO THE DENTIST *RISKY* FOR YOUR HEART?

Most of us don't like going to the dentist, and a recent report published in the *Annals of Internal Medicine* claims that invasive treatments for gum disease actually increase the risks of heart attack and stroke. Analyzing records of 32,000 Medicaid patients, researchers identified a weak linkage between cardiovascular events and having had a dental procedure in the preceding four weeks. They suggested that the temporary inflammation associated with the dental procedures could explain the association. Previous studies confirm that dental work causes a brief inflammatory response. In theory, this response could cause a tiny, brief increase in the risk of cardiovascular problems. But the long-term benefits of good dental hygiene far outweigh this short-term effect.

A second possible culprit that might connect dental procedures to heart problems is the common practice of stopping aspirin before dental procedures. In patients with preexisting cardiovascular disease, interrupting aspirin therapy could increase the risks of heart attack and stroke.

A trip to the dentist does pose a potential risk for certain heart valve patients. During dental procedures—whether a simple cleaning or a complex root canal—bacteria enter the bloodstream. These bacteria have a tendency to infect artificial heart valves. A single dose of prophylactic antibiotics before the procedure can prevent this devastating complication in people who've had heart valve surgery.

What should you do if you are a heart patient and you need dental work? Tell your dentist if you have undergone heart valve surgery so that you can receive an antibiotic. If you take aspirin, don't stop taking it without asking your doctor first. But don't let unsubstantiated fears of cardiovascular harm cause you to avoid a needed visit to the dentist. In the long run, it will be good for your smile *and* good for your heart.

Los Angeles residents who live within 100 yards of a highway tend to have abnormal arteries compared to those with homes farther away. On the other side of the country, Bostonians who live closer to highways are more likely to have signifi cant coronary heart disease than those who reside near smaller roads. In a study using air quality data from the Environmental Protection Agency, scientists correlated poor air quality with a 10 percent increase in the risk of cardiac arrest in New York City. Other observational studies suggest that the fumes (and possibly the frustration) of a rush hour traffic jam trigger 7 percent of all heart attacks.

NOISE POLLUTION

We can't sense the tiny particles in the air that enter our lungs and assault our arteries. But we can certainly hear loud and often irritating noises transmitted through the air as sound waves. Recent observational studies suggest that like air pollution, noise pollution may have adverse cardiovascular consequences for some people.

While living near a highway produces multiple cardiovascular challenges, including particulate air pollution, clusters of fast-food restaurants, and a paucity of parks and places for outdoor exercise, a Danish observational study adds traffic noise to this list of risk factors, suggesting that the louder the traffic, the greater the risk of stroke. Along similar lines, studies from England and Switzerland correlate living under an airplane flight path with an increased risk of dying from a heart attack. Analogously, people who work in noisy environments such as factories are more likely to suffer from cardiovascular problems than people with quiet workplaces.

In the United States, 22 million people work in environments with a potentially dangerous noise level. Does this noise predispose them to cardiovascular disease? We don't know for sure, but we suspect that any excessive cardiovascular risk associated with noise is modest. Concurring with this conclusion, Canadian researcher Hugh Davies stated, "If this [noise] affects you, you could think about moving somewhere quieter. But you'd probably find equal heart benefit if you stopped smoking, ate a healthier diet, or exercised more."

How do tiny particles entering the lungs exert an effect upon the heart? As is the case with many of these unconventional risk factors, scientists have not yet pieced together the entire puzzle. Some believe that the particles are so small that they actually cross from the airways to the bloodstream, which then carries them to the heart's arteries, where they can cause damage. Experimental evidence supports the direct effects of air pollution on the cardiovascular system. Animal studies demonstrate that exposure to fi ne particulate air pollution can both initiate and accelerate atherosclerosis. It increases blood pressure, constricts blood vessels, and diminishes blood vessel function in addition to increasing blood clotting and infl ammation and contributing to the development of abnormal heart rhythms.

In a given individual, the increased cardiovascular risk posed by air

PLASTIC, BPA, AND YOUR HEART

A 2008 publication in the *Journal of the American Medical Association* worried doctors and stunned consumers with its conclusion that bisphenol-A, or BPA, is linked to heart disease. A ubiquitous component of polycarbonate plastic items such as baby bottles, food packaging, and the lining of food cans, BPA can contaminate the food stored in these containers.

Health experts and the FDA have long recognized potential neurologic toxicity from BPA exposure in babies. A published report extended this concern, suggesting that higher levels of BPA exposure in adults increase the risk of developing cardiovascular disease. A 2010 update of the study suggested a similar association.

While the media created a sensation from these reports, the scientific evidence that BPA actually causes heart disease is relatively weak. It's impossible to avoid BPA completely, but if you are worried, you can limit your exposure with a few simple steps:

- Avoid plastic containers with the number seven in the recycling symbol on the bottom, as these are most likely to contain BPA.
- Don't microwave polycarbonate plastic food containers.
- Avoid canned foods.

pollution is actually quite small: exposure to severe air pollution increases the odds of having a heart attack by less than 5 percent (by comparison, cocaine use increases the risk 230 percent). But when we consider the enormous number of people who regularly inhale polluted air, the overall impact is potentially huge. Taking all of the health consequences into consideration, the World Health Organization estimates that air pollution contributes to 800,000 premature deaths per year, making it the thirteenth-leading cause of worldwide mortality.

We agree with the American Heart Association's conclusion that fine particulate matter may be a modifiable risk factor that can contribute to cardiovascular disease. What can you do about it? If possible, try not to travel during rush hour. You can determine your local air quality by visiting the website www.airnow.gov: when the air quality index for particulate matter is in the unhealthy range, limit your outdoor activity, and try to schedule your outdoor workouts away from rush hour traffic.

obesity and diabetes. Short sleep reduces the production of hormones that suppress appetite, and this may contribute to weight gain. These associations may explain the increased burden of cardiovascular disease in short sleepers. (Another possible explanation for heart disease in long sleepers: they may stay in bed for more hours because they are already unwell.)

When it comes to monitoring your sleep, make sure that you don't have sleep apnea and do your best to get enough sleep, which should be no less than seven hours per night for most of us. Ask yourself two questions: Do you feel tired during the day? Do you snore loudly? If the answer to both questions is yes, ask your doctor to evaluate you for sleep apnea. Meanwhile, take standard measures to improve your sleep, including avoiding caffeinated drinks near bedtime, limiting alcohol, and finding time to exercise during the day. Also, turn off the computer, cell phone, BlackBerry, and television at least half an hour before going to sleep—studies have shown that the mental stimulation from these devices makes it harder to fall asleep and stay asleep. By taking these steps, you'll feel better, sleep better, and be doing your heart a favor.

RELAX IN THE HOT TUB OR SAUNA

Next to almost every hot tub or sauna you see a sign that reads, "Hot tubs and saunas may pose a risk to heart patients" or "If you are a heart patient, consult with your doctor before using a hot tub or sauna." Unless your doctor is with you at that moment, you are going to have trouble getting a permission slip from her. But you can get it from us.

Staying in a hot tub or sauna for too long can certainly cause severe dehydration and dangerous fluid and electrolyte problems. But a ten-minute session in the hot tub or sauna will not hurt you or your heart. Hot tubs and saunas do not cause heart attacks or heart problems or interfere with cardiac pacemakers. As you enter the sauna or immerse yourself in the hot tub, blood vessels near the skin dilate (enlarge), causing a slight drop in blood pressure; this is rarely dangerous. You can minimize the impact of this blood pressure change by getting in and out slowly, which gives your body a moment to adjust to the temperature change. Be careful as you approach the hot tub or sauna: your greatest risk is slipping on the wet tile as you get in or out. Watch your feet, but don't worry about your heart.

The Monday Morning Heart Attack

We all have internal clocks, or circadian rhythms, that direct many of our body's processes. These can exert important influences on cardiovascular function and cardiovascular risk.

Cardiac-wise, mornings are the worst time of day. Heart attacks, strokes, and blood clots in coronary artery stents occur most often in the morning. Many scientists blame this on variations in blood pressure, which tends to drop as you sleep and then increase upon awakening, reaching its highest level at midmorning. The high blood pressure associated with this time of day batters away at plaques in coronary arteries, increasing the chances the plaques will rupture and trigger a heart attack. In the mornings we also have an increased heart rate (which increases the heart's workload), increased blood viscosity (thickness) related to overnight dehydration, and an increased tendency for blood to clot. Together, these changes increase the potential for a heart attack.

Morning heart attacks tend to be large. A team of Spanish researchers recently reported that heart attacks occurring between 6:00 a.m. and noon caused 20 percent more heart damage than those that struck later in the day. The heart patient who likes to exercise first thing in the morning may face particular risk. If you exercise in the morning, hydrate yourself before beginning your workout, and give yourself a slow warm-up to prepare your heart for the stress of exercise.

If mornings are the most dangerous time of day, Monday mornings are the most dangerous time of the week. Doctors have long noted what they call "Monday morning heart attack syndrome," having observed that the risk of heart attack is increased by 20 percent on Monday. What's the problem with Mondays? Poor sleep is probably a factor. Many people sleep in over the weekend, which makes it difficult for them to fall asleep Sunday night; they begin their week with a short and restless sleep. And then there's work. Stresses associated with returning to work can exacerbate the normal morning increases in blood pressure and heart rate, adding to the load on the heart. Don't succumb to temptation and sleep in until noon on Sunday; if you get up at a reasonable hour, you'll be able to go to sleep at your usual time.

THE WEATHER FORECAST FOR YOUR HEART

The weather may influence heart attack risk. Heart attacks peak in winter, and the colder it gets outside, the greater the risk of having one. A recent observational study from England and Wales documented a 2 percent increase in the risk of heart attack for every 1°C (1.8°F) drop in temperature. The most vulnerable people included the elderly and those with a history of coronary heart disease.

How might the cold influence your heart? Exposure to cold temperatures causes increased blood pressure, which in turn results in an increased cardiac workload and an increased tendency for blood to clot. Fortunately, the solution is easy. If you have heart disease, stay indoors when it is very cold. If you must go out, bundle up in layers.

Emotion and the Heart

In Chapter 8 we provide an in-depth analysis of the fascinating and complex links between emotions, stress, and your heart. Depression, anxiety, and anger are associated with the development of coronary heart disease. Although the exact cause-and-effect mechanism is not yet established, strong emotions (such as intense anger) and stressful situations can trigger heart attacks in susceptible individuals. Scientists are just beginning to produce data suggesting that stress reduction techniques such as yoga, cognitive behavioral therapy, and even transcendental meditation can ease cardiovascular problems in some heart patients. Exercise also reduces stress, while improving traditional cardiac risk factors. Managing your emotional health is an important component of your personal cardiac program.

Headache and Heartache: Migraines and the Heart

Recently there has been great media interest in scientific studies reporting a potential link between migraine headaches and heart disease. Because abnormal blood vessel function is one cause of migraine headaches, an association between migraines and other cardiovascular issues seems plausible. For the 28 million Americans who suffer from migraines, this is a

critically important issue. Do they need to worry about heart attacks and strokes, too?

A recent observational study of nearly 20,000 people from Iceland sought to answer this question. Researchers found that compared to people without migraines, over a twenty-five-year period people who suffered migraines with aura had modest increases in their risks of dying from coronary heart disease or stroke. (Migraine with aura refers to migraine headaches preceded by visual or other sensory symptoms, which can include flashing lights, blind spots and tunnel vision.) Based upon their observations, the study authors concluded that migraine with aura is a possible marker for cardiovascular mortality, but it is weaker than other established risk factors such as high blood pressure, smoking, and diabetes.

At this point, we don't know if successful treatment of the symptoms of migraines reduces the cardiovascular risk associated with them. Therefore, our cardiac message for migraine sufferers is to focus on the usual suspects in order to ensure your heart health.

EDUCATION, MONEY, AND YOUR HEART

Health behaviors and cardiovascular outcomes are linked to education, social status, and money. Studies examining the impact of education find that the longer you stay in school, the better your cardiovascular health. Specifically, higher levels of education—from high school to college and beyond—correlate with reduced risks of diabetes, high blood pressure, and heart disease. On average, the person who attended graduate school has a blood pressure that is three points lower than that of the high school dropout. Studies suggest that the link between higher educational attainment and a healthy cardiovascular system is a healthier lifestyle.

Like education, income and social standing correlate with heart health. Across the United States and around the world, the poorest people have the greatest risk of cardiovascular disease. Those engaged in manual trades have a fourfold increase in the risk of dying from heart disease when compared to management. Once again, behaviors explain the gap. Exercise, a good diet, and general health consciousness tend to be more prevalent among managers and administrators. The solution is not to aim for advancement at work in order to protect your heart; rather, adopt the right behaviors as you climb the corporate ladder, so that you reach the top with a healthy heart.

Rx: CORONARY HEART DISEASE RISK FACTORS

Manage the usual suspects:

Cholesterol
High blood pressure
Diabetes
Smoking
Obesity

Recognize additional risk factors:

Inflammatory diseases (rheumatoid arthritis, psoriasis, lupus)
Gum disease
Air pollution
Sleep apnea
Emotional stress
Migraine

faced a future that might be dominated by heart disease. Irene had already experienced a heart attack and bypass surgery. Would her children have the same fate?

Thankfully, we were able to reassure her. We had diagnosed her kids early enough to delay or prevent significant cardiac consequences. We laid out a plan for cholesterol management that would control the LDL levels of the entire family. Explaining the role of a good diet (low in saturated fats, high in fi ber and whole grains), we also emphasized the importance of exercise. Most important, we immediately began all three children on a regimen of statin drugs. Initially Irene protested, worried about giving her kids such medicines. We convinced her that with her children's cholesterol levels so high, a statin could be lifesaving.

We kept our promise to treat Irene's cholesterol levels, and her family's future is bright. Irene always takes her medicines (a cocktail based primarily on a high-dose statin), and her LDL hovers near 100, an acceptable although not optimal value. The kids *usually* take their medicines (they're teenagers, after all), and their cholesterol levels, too, have come under better control. One small pill each day may well keep them out of our catheterization laboratories and cardiac operating rooms. We think that's a pretty good trade-off.

Familial hypercholesterolemia (Irene's disorder) has taught us much about the important role of cholesterol in the development of coronary heart disease. But most people who suffer the cardiovascular consequences of elevated cholesterol do not have this genetic condition and the strong family history that enable early detection and treatment. For the rest of us, vigilance is imperative. Cholesterol remains an important silent killer

High cholesterol has no symptoms; every day, we see patients who learn about their high cholesterol only *after* they arrive in the coronary care unit with a heart attack. Avoid this scenario. Have your cholesterol tested. If the results suggest you are at risk, review the information in the next few pages. It might just save your life.

CHOLESTEROL: WHY DO WE HAVE IT AND HOW DO WE MEASURE IT?

Cholesterol's Functions

What is cholesterol and what does it do? A waxy, yellowish white substance, cholesterol was fi rst isolated by an eighteenth-century French chemist who was studying gallstones. This first identifi cation of cholesterol suggested that its primary roles centered on illness and disease, but subsequent research proved this theory incorrect. It turns out that every cell in your body contains cholesterol, and you can't live without it.

Cholesterol is a key component of the cell membrane, the outer barrier between the cell and the rest of the body. Within the membrane, cholesterol molecules act like tollbooths, helping to regulate the passage of materials into and out of the cell. Cholesterol also serves as a building block for many important hormones, including estrogen, testosterone, and cortisone. Your body even needs cholesterol to manufacture vitamin D from sunlight.

When we talk about cholesterol, we are usually referring to cholesterol in the blood. The blood carries cholesterol to our cells and tissues, where it can be used to synthesize needed chemicals and hormones, broken down and removed from the body, or, under certain conditions, cause damage to the blood vessels themselves. When cholesterol travels in the blood, it is carried in packages of molecules called lipoproteins. We generally focus on two types of blood-borne cholesterol, which are distinguished by their attached lipoproteins: low-density lipoproteins (LDL) and high-density lipoproteins (HDL).

LDL is the major carrier of cholesterol in the blood. High levels of LDL or "bad" cholesterol are associated with development of plaques in arteries. Oxidation of LDL cholesterol in the blood enables it to enter the walls of arteries, leading to the buildup of plaques.

HDL or "good" cholesterol works in an opposite fashion, removing cholesterol from the arteries and returning it to the liver, where it is either broken down or removed from the body. By this mechanism, HDL retards the formation of artery-blocking plaques. Contemporary research demonstrates that some forms of HDL can actually clear the arteries, reducing the burden of plaque. Naturally occurring high levels of HDL are clearly associated with protection from heart disease.

If you want to infl uence cholesterol levels, you first need to know where it comes from. For the most part, you make it. Eighty percent of the body's cholesterol is made by the liver. While most people think that diet is the

most important factor in determining cholesterol levels, this is a myth. Only 20 percent of your cholesterol comes from your diet, which explains why it is so diffi cult to reduce blood cholesterol levels via dietary interventions alone. Theoretically, if you completely eliminated all cholesterol from your diet, you might reduce your total cholesterol level by 20 percent. In reality, dietary modifi cations are usually even less effective because the liver responds to reduced dietary cholesterol intake by increasing cholesterol synthesis.

Understanding the limitations of dietary interventions often helps people with high cholesterol levels accept the fact that they need to take cholesterol-lowering drugs. If your cholesterol level is 50 percent higher than normal, you are simply not going to reach your cholesterol goals by diet alone. In such a case, you need to combine a good diet with the right medication.

Measuring Cholesterol

When physicians want to determine cholesterol levels in patients, we obtain a laboratory test known as a lipid panel. Your lipid panel will include four important numbers: total cholesterol, LDL, HDL, and triglycerides. Many patients who come to us can recite only one of these numbers, the total cholesterol. But of the four numbers, total cholesterol is often the least important.

When it comes to predicting heart disease, the LDL level is the most useful. Study after study has confirmed the strong relationship between high levels of LDL and heart disease. In contrast, high HDL levels reduce the risk of cardiac events. Because these two forms of cholesterol have opposite effects on risk, total cholesterol has major limitations as a predictor of heart disease. For example, consider a healthy young woman with a very high HDL level (85) and a normal LDL level (125). Although her total cholesterol is elevated (above 200), that is mainly as a consequence of high HDL. In this case, we deem this a favorable lipid profile.

Similarly, we usually do not rely on the ratios of the different lipids to guide our treatment. We want to know the individual values, not their ratio; these are the numbers most closely tied to treatment and prognosis.

What are normal levels of LDL, HDL, total cholesterol, and triglycerides? That's a surprisingly difficult question to answer. "Normal" cholesterol levels have been a moving target throughout the years. In the

SPECIAL LIPID TESTS

In recent years, commercial laboratories have aggressively marketed complex special lipid tests to physicians who care for heart patients. Many self-described lipid experts order these tests in every patient with any hint of a lipid abnormality. The tests are expensive, costing hundreds of dollars, compared to a standard lipid profile, which typically runs about $25. Are they worth the extra money?

No. The special lipid tests are nearly worthless. The purported value of the test is the detection of "small dense" LDL cholesterol, a certain type of cholesterol that may be especially likely to cause plaque buildup in the arteries. Scientists debate the importance of small dense LDL, but we can assure you that you don't need an expensive blood test to look for it. A standard lipid profile provides all of the information that you need. If triglyceride levels are high, the small dense LDL is usually high, too. Don't waste your money on special tests. If your doctor orders a lipid profile or a cholesterol test, make sure that you just get the standard test.

1960s, American physicians decided that a total cholesterol of less than 300 was normal. A decade or two later, we changed our minds, deeming levels less than 240 acceptable. Most recently, experts have settled on the value of 200 as the upper limit of normal for total cholesterol. Why can't we decide?

The problem relates to our diets and our habits. Beginning after World War II, cholesterol levels rose rapidly as Americans consumed diets rich in meat and other sources of saturated fat. Scientists were misled into believing that the elevated cholesterol levels observed in the population were normal. However, we now know that such high levels are not healthy.

Current guidelines recommend that in adults without known heart disease or risk factors for heart disease, total cholesterol should be less than 200, HDL greater than 40 for men and greater than 45 for women, LDL less than 130, and triglycerides less than 150.

We need to emphasize a couple of important points here. The first relates to the relative importance of these different measurements. Although early attention centered on total cholesterol, remember that today we focus mostly on LDL cholesterol. The LDL level is the best predictor of the risk of heart attack and stroke, and the basic concept is simple:

lower is better. For example, an individual with an LDL of 125 has a higher risk of developing heart disease than someone of similar age and gender with an LDL of 90, even though both levels may fall in the normal range. In fact, this continuous relationship even extends to individuals with very low levels of LDL. Similar relationships hold true for HDL, where higher is better, although the strength of the relationship is somewhat less robust.

WHEN DO WE TREAT LDL CHOLESTEROL LEVELS?

Every time we bring a group of doctors together to determine when to treat LDL levels, we come away with new answers and recommendations. Today, most doctors rely on guidelines first developed in 1985 by the National Institutes of Health in collaboration with several professional medical societies known as the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP). The most recent update to the guidelines (ATP III) was released in 2004, and an entirely new version (ATP IV) is due soon. The targets of cholesterol levels and their treatment continue to move.

The basic idea behind treatment guidelines is to treat elevated LDL cholesterol levels in people who already have coronary heart disease or who face a high likelihood of developing it. We use simple calculations to identify people who have a high risk of developing coronary heart disease and who therefore require cholesterol treatment. To determine who should be treated, we use a method known as the Framingham Risk Score, which incorporates seven risk factors to estimate a person's risk of developing coronary heart disease (CHD) over the next ten years.

FACTORS IN THE FRAMINGHAM RISK SCORE

Age

Gender

Total cholesterol

HDL

Tobacco use

Systolic blood pressure (the first number in the blood pressure reading)

Use of blood pressure medications

which tend to improve the ratio between LDL and HDL cholesterol. Saturated fats (typically found in meat and full-fat dairy products), which promote the production of cholesterol by the liver, are strictly limited. In addition, avoid trans fats, which adversely affect the balance of production between LDL and HDL. The message here: eat the right fats to control your cholesterol.

ATP III Guideline Recommendations for TLC Diet

NUTRIENT	RECOMMENDED INTAKE
Saturated fat	Less than 7 percent of total calories
Polyunsaturated fat	Up to 10 percent of total calories
Monounsaturated fat	Up to 20 percent of total calories
Total fat	25–35 percent of total calories
Carbohydrate	50–60 percent of total calories
Fiber	20–30 grams/day
Protein	Approx. 15 percent of total calories
Cholesterol	Less than 200 mg/day
Total calories	Sufficient to maintain ideal body weight

How does the cholesterol you eat affect your body's own cholesterol levels? Surprisingly, when it comes to influencing your cholesterol levels, your intake of saturated and trans fats plays a greater role than dietary cholesterol does. Foods rich in cholesterol, such as eggs, produce only modest changes in blood cholesterol levels. Your diet *can* include eggs, but no more than one to three per week if you need to lower your LDL cholesterol.

Certain foods have a beneficial impact on your LDL cholesterol levels. Soluble fi ber, found in foods such as oatmeal, beans, and legumes, slightly decreases LDL levels in many people. National guidelines recommend eating foods (often margarines) fortified with certain plant stanols/sterols that slightly lower LDL. While many doctors support this recommendation, it is important to know that there is no strong scientific evidence documenting a clinical benefi t (e.g., fewer heart attacks). While it is probably okay to purchase these fortifi ed spreads, don't expect miracles.

There are no magic foods or supplements that can dramatically lower

LDL. Your dietary habits should be sensible rather than extreme. We do not recommend highly restrictive diets such as the Ornish, Esselstyn, or Pritikin diets: following these diets is challenging, and ultra-low-fat diets tend to have the unfavorable side effect of lowering levels of HDL, the "good" cholesterol.

Securing a favorable lipid profile also requires consistent exercise, maintaining an ideal body weight, and avoiding tobacco. Fortunately, each of these three factors helps to raise your HDL levels as well.

Lifestyle to Improve Your Cholesterol Profile

Diet	
Avoid saturated fats	Limit red meats, processed meats, full fat dairy products, lard
Favor monounsaturated fats	Olive oil, canola oil
Favor dietary fiber	Whole grains, oats and oatmeal, beans, barley
Exercise	Aerobic exercise at least 30 minutes per day
Ideal body weight	Achieved by decreasing calories and increasing exercise
Quit smoking	Enough said!

What should you expect from the TLC program of diet, weight loss, and exercise? The program's effects on LDL levels are measurable but often quite modest. Because most of your cholesterol is manufactured in your liver, a reduction of more than 10–15 percent in LDL through changes in diet alone is unusual. If your LDL level is 140, you can probably get it down to 115–125 by adopting a healthy lifestyle. On the other hand, if your LDL is more than 20–25 percent above the target goal, you are unlikely to reach an optimal level through lifestyle changes alone.

This doesn't mean that you should abandon the TLC approach. Rather, embrace it as part of a broader program to bring down your LDL cholesterol levels. Understanding the limits of lifestyle modification in controlling LDL is important because many patients have an inordinate fear of taking cholesterol-lowering drugs. Our patients constantly tell us, "I know I can lower my cholesterol with diet. I'll cut out the french fries! Please don't put me on drugs!" For high-risk patients with very elevated LDL levels, this perspective can be lethal. As you work to control your LDL, begin with

the TLC plan, but be prepared to take the next step to ensure your heart health.

THE STATINS ARRIVE

What Are Statins?

Because lowering LDL cholesterol is the primary goal of cholesterol management, drugs that reduce LDL levels are the big guns in our armamentarium. Statins, the most important class of LDL lowering drugs, have revolutionized modern cardiology. The first statin, lovastatin (Mevacor), reached the market in 1987, signaling a major breakthrough in the battle against CHD. Statins work by blocking a critical enzyme involved in the liver's production of cholesterol, reducing the amount of cholesterol the liver makes. Statins also increase the activity of receptors on the surface of liver cells, literally pulling cholesterol from circulating blood. As a consequence, blood levels of LDL and total cholesterol drop.

Lovastatin wasn't particularly potent by today's standards, but it was much more effective than anything previously available. Within a few years after the introduction of lovastatin, the FDA approved two more statins, pravastatin (Pravachol) and simvastatin (Zocor). Surprisingly, initial acceptance of statins by the medical community was relatively slow. Although these drugs were very effective at lowering LDL, there were not yet randomized clinical trials demonstrating that reducing LDL in patients with CHD would result in a reduced risk of future heart problems. The concept made sense, but a change in practice of this magnitude required solid evidence.

The proof came in 1994 with the publication of the landmark Scandinavian Simvastatin Survival Study, also known as the 4S Trial. A total of 4,444 patients with very high LDL levels who had suffered a heart attack were randomly assigned to receive either simvastatin or a placebo. After five years, simvastatin-treated patients had a 30 percent reduction in the risk of death due to all causes and a 42 percent reduction in the risk of death due to CHD. The risk of a second heart attack was also dramatically reduced, by 34 percent.

These results were stunning, and the medical community was positively giddy as a result of the 4S trial. Caught up in the enthusiasm, the two Nobel Prize winners who discovered the biologic mechanisms underlying the effects of statins wrote an editorial in the prestigious journal

Science predicting that by the turn of the twenty-first century heart attacks would be gone. Obviously, that prediction has not come to pass, but irrational exuberance notwithstanding, these drugs are incredibly useful. Today eight statins are marketed in the United States. Although all of these drugs substantially lower LDL cholesterol, they vary considerably in efficacy. The following table shows the most commonly used statins and the typical LDL-lowering effects that can be expected at various dosages.

HOW MUCH WILL A STATIN LOWER LDL CHOLESTEROL?

Drug	10 mg	20 mg	40 mg	80 mg
Pravastatin (Pravachol)	22 percent	28 percent	34 percent	40 percent
Simvastatin (Zocor)	28 percent	34 percent	40 percent	Not recommended
Atorvastatin (Lipitor)	34 percent	40 percent	46 percent	52 percent
Rosuvastatin (Crestor)	42 percent	48 percent	54 percent	Not available

Using this information, you can predict a particular statin's effect on your LDL level.

Does this mean that once you begin to take a statin, you can eat anything that you want? Don't make this mistake! By adhering to a heart-healthy, cholesterol-lowering diet, you can ensure that you can take a relatively low dose of your statin. Because statin side effects are dosage-related, this can represent a big advantage for you.

Diet is especially important in high-risk patients who may need to lower their cholesterol by 100 points or more. In these cases, no statin is likely to work on its own, but a combination of one of the most potent statins at the highest dosage and a heart-healthy diet will probably get you to your goal.

Patients often worry about the cost of statins. Statins need not be expensive. For many years statins were available only as brand-name drugs, and costs often reached $200 per month. In 2001, the first statin became generically available, but because lovastatin was not the most effective statin, branded drugs continued to dominate. In 2006, a medium-potency statin, simvastatin, became available as a generic, resulting in a gradual shift away from the more expensive brand-name drugs. An even more po-

tent statin, atorvastatin, became generically available in November 2011, thus enabling even more patients to reach their LDL goals at lower cost.

If your doctor tells you that you need a statin, get involved with the decision making in order to maximize the effect on your heart and minimize the impact on your wallet. With your LDL level in hand, look at the table on the previous page. If you can achieve your LDL goal using a generic statin, we think such an approach makes the most sense. If the table predicts that you can't achieve optimal results generically, you may need to take the last remaining branded drug, rosuvastatin, or an additional drug (combination therapy) to further lower your LDL cholesterol.

WOULD YOU LIKE FRIES AND A STATIN WITH THAT?

Recognizing our infatuation with fast-food meals, British cardiologists proposed a novel concept to mitigate the risks associated with our dangerous eating behaviors. Their solution? Take a statin tablet with your Quarter Pounder! They calculated that most statins are sufficiently powerful to offset the cardiovascular risk associated with eating a meal such as a Quarter Pounder with cheese and a small milk shake. So why not put them together? After all, if you are going to put yourself at risk by driving ninety miles per hour, you might as well wear your seatbelt to ensure some protection from your dangerous behavior.

Of course, the British doctors offered this proposal with tongue in cheek. We do not really know whether or not statins, or any medicine for that matter, can reduce the deleterious effects of dangerous behaviors that are repeated over years or decades. It is unlikely that the condiment station at McDonald's will any day soon include salt, pepper, ketchup, and a statin. The British authors suggest that "no tablet can completely neutralize the harm to your individual health from eating unhealthy. Better ways to reduce your risk of death from heart attack include: eating healthily, exercising, maintaining a healthy weight, and not smoking." We could not agree more.

Statin Side Effects

Few drugs have been studied as carefully as statins. Scientists have examined statins in clinical trials involving hundreds of thousands of patients. These drugs are among the safest drugs in all of medicine, but they do have side effects. Because statins affect metabolic pathways in the liver, doctors

RED YEAST RICE: A "NATURAL" STATIN?

For centuries the Chinese have used red yeast rice as a food preservative, food colorant (it gives Peking duck its red color), spice, and ingredient in rice wine. But outside of the kitchen, others have extolled the medicinal properties of red yeast rice, claiming that it improves blood circulation and alleviates indigestion and diarrhea. This compound needs to be kept in the kitchen and out of your medicine cabinet.

Produced by culturing yeast on rice, red yeast rice *is* chemically active. It contains a group of substances that work like statins in the liver. Sound familiar? Red yeast rice actually contains a naturally occurring form of lovastatin, the first statin. However, unlike a prescription statin, the amount of active ingredient in red yeast rice is neither standardized nor controlled. Some products contain potentially toxic dosages of lovastatin, while others contain none at all. One-third of all red yeast rice preparations actually contain chemicals that can be toxic to the kidneys.

The FDA has repeatedly attempted to ban these products, but it has been difficult to stop their sale. In 2008, Americans spent $20 million on red yeast rice supplements, an 80 percent increase from 2005. There are no advantages to taking this "natural" form of lovastatin, but there are plenty of risks. Get your statin the right way—with a doctor's prescription.

OTHER ANTI-CHOLESTEROL MEDICINES

Ezetimibe (Zetia) and Vytorin

What if you follow a good diet, exercise, take your statin, and your LDL cholesterol is *still* too high? What's your next step? This question comes up for doctors, their patients, and, of course, the big pharmaceutical companies that are searching for the next blockbuster heart drug.

The answer to the question is controversial. While statins work at the level of the liver, ezetimibe reduces absorption of cholesterol from the intestine, lowering LDL by an additional 15–20 percent. Ezetimibe is available in two forms, as a stand-alone drug (Zetia) and as a combination product packaged with simvastatin (Vytorin). Both Zetia and Vytorin are not available in generic form yet and are relatively expensive.

Although ezetimibe seems like a logical choice for lowering cholesterol,

STATINS, INFLAMMATION, AND C-REACTIVE PROTEIN

While many people consider coronary heart disease a straightforward problem of arterial blockages caused by cholesterol, it is far more complicated. A variety of other factors contribute to its development, including inflammation. Boston researcher Paul Ridker confirmed the link between coronary heart disease and inflammation when he demonstrated that high blood levels of C-reactive protein (CRP), a substance related to inflammation, are associated with an increased likelihood of suffering a heart attack.

The higher your blood CRP level, the greater your risk of cardiovascular disease. We consider a value of 1 ideal, values of 1 to 2 intermediate, and values greater than 2 indicative of high cardiovascular risk. Fortunately, we can treat high CRP levels, and it turns out that the best treatment is the same as the treatment for high LDL cholesterol levels: statins. In addition to decreasing LDL cholesterol levels, statins lower CRP levels, and many scientists believe that this anti-inflammatory effect of statins contributes to their effects on cardiovascular health.

Do you need to know your CRP level? Maybe. Based upon the landmark findings of a study called the Jupiter Trial, we measure CRP levels in patients whose LDL and other risk factors put them on the borderline for requiring statins—for example, a fifty-five-year-old man with an LDL of 120, high blood pressure, and a low HDL. Some doctors would prescribe a statin and some wouldn't. We use the CRP to help us decide. If his CRP is low (less than 1), we defer statin treatment and concentrate on lifestyle factors. If the CRP is clearly elevated (greater than 2), we prescribe a statin in addition to lifestyle modification.

this drug has generated considerable controversy. In clinical trials, ezetimibe reduces LDL levels but has not been shown to reduce the risk of heart attack or stroke and failed to slow the progression of plaques in the carotid arteries in the neck. This raised questions about the ultimate effectiveness of the drug in improving heart health.

Today we use Zetia as a first-line agent rarely and only in patients who cannot tolerate any of the statins due to side effects, and as a second-line drug in those who cannot reach their LDL goals despite maximum doses of a powerful statin. We never use the combination drug Vytorin: there is simply no rationale for combining a medium-potency statin such as simvastatin with Zetia when there are more potent statins available. Why take an expensive combination product of uncertain benefit when a well-studied generic drug is readily available?

HOW TO INCREASE YOUR HDL CHOLESTEROL

Although current national guidelines focus on reducing LDL cholesterol, we have known for decades that low levels of HDL or "good" cholesterol are strongly associated with an increased risk of coronary heart disease. We generally want men to have HDL levels over 40 or 45, while women should aim for levels above 45 or 50. National guidelines do not make strong recommendations for treating low levels of HDL, citing the lack of high-quality trials confirming a benefit to raising them and noting that we have few drugs to do so. Some physicians also point to recent controversial studies suggesting that if your LDL level is low enough, your HDL level may be less important. We disagree with the concept of ignoring HDL.

Lifestyle Changes to Increase Your HDL

So how can you increase your HDL? We begin with lifestyle, and exercise is a key feature. Aerobic exercise can raise HDL levels by 10 percent, with the degree of HDL increase proportional to the amount of exercise. If you are currently sedentary, walking thirty minutes a day will cause a measurable increase in your HDL levels; walking further or engaging in more strenuous exercise, such as running, biking, and swimming, will increase your HDL levels even more. Resistance exercise such as weight lifting does not help with HDL levels.

If you are overweight, weight loss can also boost HDL levels. A loss of just six or seven pounds can increase your HDL by 1 mg/dL. If you are extremely overweight and lose twenty to thirty pounds, you will see a substantial increase in your HDL level. However, don't be discouraged if the effects are not immediate; the HDL levels may actually decrease during active weight loss, but they will eventually increase when your weight restabilizes at a lower level.

As with LDL, your diet influences your HDL levels. Dietary fat increases HDL levels, but you need the right fats. This means focusing on polyunsaturated and monounsaturated fats from vegetable sources such as olive oil and canola oil. Avoid trans fats and limit the saturated fats found in meats and full-fat dairy products. If your HDL is low, you should also shy away from trendy ultra-low-fat, high-carbohydrate diets, as these will actually decrease your HDL levels.

Stop smoking! Quitting smoking can increase your HDL by up to 10

percent. But you *can* have a drink. Moderate alcohol consumption (one drink per day in women, one to two per day in men) has been shown to increase levels of HDL cholesterol. This means that if you already drink alcohol, you can continue (in moderation), but we don't write prescriptions for people to start drinking just to treat a low HDL level.

Medicines to Raise HDL?

When diet, exercise, and smoking cessation are not enough to raise a patient's HDL to acceptable levels, physicians often turn to drug therapy. The problems here are a relative lack of options and no proven medical benefit for the few HDL-raising drugs that are available. Unlike statins, which can dramatically increase LDL cholesterol, medicines designed to raise HDL have limited effectiveness, and some have important side effects. Statins can increase HDL by 5–10 percent. There are small but potentially important differences in the effects of various statins on HDL. For patients close to their HDL target, switching from one statin to another may help to reach that goal.

One of the B vitamins, niacin, raises HDL levels up to 25 percent (and also lowers triglyceride levels) when taken at high doses. Therefore, until recently, we often prescribed Niaspan, a prescription form of niacin, to increase HDL in patients with very low levels. A recent study raised questions about this practice. The National Institutes of Health released results of a randomized controlled clinical trial investigating the impact of niacin in patients with low HDL levels. More than 3,000 patients with cardiovascular disease, low HDL levels, and high triglyceride levels were randomly assigned to either niacin or a placebo in addition to the statin that they were already taking. Niacin *did* raise HDL, as expected, but it did not reduce heart attacks and may have even slightly increased the risk of stroke. Based upon this fi nding, the Data Safety and Monitoring Board stopped the study eighteen months ahead of schedule. We have reduced our practice of prescribing niacin supplements to increase HDL levels pending review of this study when it is published.

Occasionally physicians employ another class of drugs to raise HDL, the fibric acid derivatives (fi brates). These include gemfibrozil (Lopid), fenofibrate (Tricor and other brands), and fenofibric acid (Trilipix). Fibrates raise HDL by 10–15 percent, which sounds appealing. Unfortunately, clinical trials have failed to demonstrate compelling evidence for a reduction

in heart problems with fenofibrate, the most commonly used drug in the class. Gemfi brozil, another fi brate, has been associated with muscle damage when used with statins. For these reasons, we do not use fibrates to raise HDL in our patients.

NEW DRUGS TO INCREASE HDL CHOLESTEROL

We have progressed about as far as we can in lowering LDL cholesterol using statins. There are powerful new therapies under development to further lower LDL, but these will be useful only in the modest number of patients who can't get to goal using current drugs. However, many scientists remain cautiously enthusiastic about developing new medicines to raise levels of HDL cholesterol.

So far, the search for drugs to increase HDL has been an emotional and scientific roller coaster. After fifteen years of research costing more than $1 billion, Pfizer thought it had the answer with a medicine called torcetrapib. In animal experiments and preliminary studies, the drug worked as expected. It raised HDL 60 to 80 percent. But in a clinical trial involving 15,000 people, the medicine actually *increased* the risk of death and heart problems—a dead end for that medicine.

While Pfizer's experience left us disappointed and scratching our heads, we have not given up. Several drugs in the medical pipeline look promising. In a recent study of more than 1,600 patients, one of these new drugs more than doubled HDL levels while cutting LDL levels by 40 percent. If the next step, a larger study to assess the drug's impact on heart health, is successful, we may see the next blockbuster therapy for heart patients.

HIGH TRIGLYCERIDES AND METABOLIC SYNDROME

After LDL and HDL, the triglyceride level is the third most important value on your lipid blood test. What are triglycerides? Simply put, they are fat. Fat in your body and fat in food exist primarily as triglycerides. When we consume excess calories in any form, our body produces triglycerides, which are then stored in fat cells. Although we include blood triglyceride levels with every lipid panel, doctors argue about their relevance.

Of course, we have guidelines for normal values. We consider levels

Rx: CHOLESTEROL

Your liver makes most of the cholesterol in your body—only 20 percent comes from food

LDL cholesterol is the primary target

Know your target LDL:

- If you have coronary heart disease, your LDL cholesterol should be 70 mg/dL or less. If you have major risk factors for coronary heart disease, your LDL cholesterol should be 100 mg/dL or less
- LDL greater than 130 mg/dL is unhealthy even if you don't have heart disease

Lower your LDL level:

- Choose poly- or monounsaturated fats and high-fiber offerings
- Avoid saturated and trans fats
- Exercise
- Statins—make certain you get the right drug in the right dosage

HDL cholesterol: keep it up

Higher is better: over 40 mg/dL for men and over 45 mg/dL for women

Raise your HDL level:

- Exercise
- Weight loss
- Quit smoking

CHAPTER 4

YOUR WEIGHT AND YOUR HEART: HOW EXTRA POUNDS CREATE EXCESS RISK

THE BIG PICTURE

Right now, 55 percent of Americans are trying to lose weight. But in many cases they are dieting for the wrong reason. Their goal is to look better in that new bathing suit or fit into that favorite pair of jeans that seemed to be the perfect size last year but now feels just a little snug. These dieters are missing the point of weight loss efforts.

Being overweight threatens more than your appearance—it threatens your life. Today, two-thirds of Americans are overweight or obese (overweight is defined as a body mass index, or BMI, greater than 25, and obesity is defined as a BMI greater than 30). The heaviest among us will lose between five and fifteen years of life as a consequence of their extra pounds. The costs of the obesity epidemic ripple through society, from the impact on productivity to billions of dollars in health care expenditures, and there are no easy solutions.

Even those who are mildly obese face an increased risk of early death. The main health threat caused by obesity? You guessed it—heart disease. Obesity has become one of the most important reversible causes of heart

disease. When a person loses weight, his or her risk factors for heart disease nearly always improve. Obesity is both preventable and treatable, but few patients are successful over the long run.

JIM FINNEGAN: "BIG MAN"

When Jim Finnegan played college football, his teammates christened him "Big Man" because he made big plays as a wide receiver, even though at a paltry fi ve foot eleven and only 190 pounds he was dwarfed by the huge linemen.

A little too small for the NFL, Jim settled into a career as a stockbroker after his illustrious college experience. Jovial and well liked, he regaled clients with stories from his football days. His workweeks were filled with client meetings, often taking place over 18-ounce rib eye steaks or on the golf course, where Jim managed to play two or three times per week.

We met Jim when he was fifty-two years old, his football days three decades in the past. Jim came to us seeking cardiac clearance before undergoing knee replacement surgery. This type of examination is used to determine whether his heart would be able to tolerate the strain of the procedure.

Jim no longer looked like a college football star. He now carried 245 pounds on his fi ve-eleven frame, having gained two to three pounds a year since college graduation. The year-to-year change in Jim's physique was not that great, but over the years the cumulative impact was huge. And so was Jim's waistline.

Jim told us that he had not seen a doctor in ten years. We did a few basic tests and were not surprised by the results. Jim's blood pressure was 160 over 90—high blood pressure. His blood glucose was 130 mg/dL—diabetes. His LDL cholesterol was 160 mg/dL—high cholesterol. His wife reported that he had started to snore loudly, and a simple test confirmed that he had sleep apnea. When we presented Jim with his test results and new diagnoses, he responded, "I was healthy until I met you guys. Now I have all of these diseases. What happened?"

We explained that "what happened" was a 55-pound weight gain. His diabetes, hypertension, high cholesterol, and sleep apnea were all direct results of his bulging waistline. In addition, the extra weight probably stressed his joints and contributed to the arthritis in his knee. Because he also had some chest pain when walking briskly on the golf course, we performed