
INTRODUCTION

This “book within a book” is intended to provide a concise overview of common cardiovascular disorders and symptoms. It is set up in a consistent question-and-answer format to enable you to quickly find the information you seek. The various entries are cross-referenced to chapters that provide more detailed information.

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ANGINAL PECTORIS

WHAT IS IT?

Angina pectoris is chest pain caused by myocardial ischemia, a condition in which the amount of oxygen the heart muscle requires exceeds the amount it receives. It usually occurs on exertion and is relieved by rest. Angina generally is a symptom of coronary artery disease. In more severe cases, it may occur with minimal effort or at rest.

WHO GETS IT?

Angina affects both men and women, usually in middle age. Men are much more likely than women to experience it before age 60. It may develop weeks, months, or even years before a heart attack, or maybe experienced only after a heart attack has occurred.

WHAT ARE THE SYMPTOMS?

Angina itself is a symptom. The pain usually lasts only a few minutes and may be felt in a number of ways, although the characteristics are usually constant for any given person. It may be experienced as only a vague ache or mild discomfort, or it may be a burning, squeezing, steady pressure, or fullness. The pain or discomfort most commonly occurs in the center of the chest (under the breastbone) but may also radiate down one (particularly the left) or both arms, to the neck, shoulders, lower jaw, or back. In its classic form, angina occurs with exertion and is relieved by rest. It may be provoked as well by mental stress and anxiety, or the combination of a heavy meal and even mild exertion. In more advanced cases, angina may occur at rest or even wake the individual from sleep.

HOW IS IT DIAGNOSED?

The key to diagnosing angina is a careful history: the patient's report that pain occurs on physical exertion or

mental or emotional stress and subsides when the activity is stopped. A resting electrocardiogram may show changes during the period of pain. An exercise stress test may be used to establish a diagnosis and to determine the level of exertion that produces symptoms. In some cases a thallium or other nuclear scan maybe necessary to define how much of the heart is not receiving adequate oxygen when angina occurs.

HOW IS IT TREATED?

An episode of angina is treated by ceasing the activity that brought it on, and/or by taking medication, especially nitroglycerin.

WHAT ARE THE COMPLICATIONS?

In more severe cases, known as unstable angina, the episodes of pain may occur at rest and become quite frequent, more intense, and of longer duration. Untreated underlying coronary artery disease may lead to a heart attack or sudden death.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Angina maybe minimized by avoiding exertion or taking nitroglycerin before a stressful or strenuous activity known to produce pain. Several classes of drugs, sometimes used in combination with nitroglycerin, can reduce anginal episodes and increase the amount of activity that can be done before pain starts. Beta blockers reduce the heart rate, lower blood pressure, and diminish the force of the heart's contractions, thus reducing the heart's need for blood. Calcium channel blockers and long-acting nitrates lower blood pressure and help to dilate, or open up, the narrowed arteries. In more severe cases, coronary bypass, angioplasty, or a combination maybe required. In all cases, some life-style changes—moderate exercise, weight loss if appropriate, smoking cessation, dietary changes, and stress modification—should be implemented as soon as possible.

See Chapter 11.

AORTIC ANEURYSM

WHAT IS IT?

An aneurysm is an outward bulge in the wall of a blood vessel. Aortic aneurysms occur in the aorta, the body's major artery. The aorta branches, to distribute blood throughout the body. The main branch travels down the body through the chest (thoracic) area and the abdominal area—the two primary sites for aortic aneurysms. Aneurysms may bulge on only one side of the aorta or around the full diameter in ail directions. In a dissecting aneurysm (which also has a propensity for rupture), the inner and outer layers of the artery split apart and blood gets between the layers, causing swelling of the wall.

The blood supply to various organs (for example, the kidney) can be markedly reduced because the opening of the vessel has been narrowed.

WHO GETS IT?

Aneurysms develop in patients with atherosclerosis. An additional predisposing factor is hypertension. Patients with Marfan syndrome also develop dissecting aneurysms. Aneurysms may develop because of other congenital problems, but this is rare. Although aneurysms may develop in blood vessels in the limbs, this is rare and does not present the potentially life-threatening problems of those in the trunk.

WHAT ARE THE SYMPTOMS?

Aneurysms frequently produce no symptoms at all, and are detected only on physical examination. Rarely, thoracic aneurysms may cause chest pain. A dissecting aneurysm in the thoracic area can cause chest pain similar to that of a heart attack. In addition, a “tearing” sensation may be felt in the chest and back. Abdominal aneurysms often can be felt just beneath the skin as a small throbbing lump that is tender to the touch; when they begin to leak, they may cause pain that can radiate to the back and to the groin area. Dissecting aneurysms in the abdomen, which are rare, may cause severe pain and fainting.

HOW IS IT DIAGNOSED?

Aneurysms may be difficult to diagnose, because many produce no symptoms until they dissect or rupture. A routine chest X-ray may detect an aneurysm in that area! A physical examination and an X-ray of the abdomen may help to diagnose an aneurysm in the stomach. Echocardiography, CT scans, and MRI are also likely to be part of the diagnostic process for defining the size of an aneurysm. It is difficult to diagnose a blood vessel aneurysm in the brain except by special procedures.

HOW IS IT TREATED?

Drugs may be prescribed to lower blood pressure and reduce the risk of rupture. Abdominal aneurysms that are large or increasing in size should be treated surgically. Enlarging thoracic aneurysms should be considered for surgery. A dissecting or ruptured aneurysm requires emergency surgery.

WHAT ARE THE COMPLICATIONS?

Large aneurysms can apply pressure to and damage adjacent blood vessels or nerves. Aneurysms can also cause major disturbances in local blood flow and increase the risk of clot formation; if the clot breaks away, it can lodge elsewhere in the body and cause a stroke, or other organ damage. If an aneurysm bursts or ruptures, hemorrhage occurs and the supply of blood to

tissues beyond the site is cut off. A major aortic rupture can cause circulatory collapse and death if not treated immediately.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Congenital aneurysms cannot be prevented. A healthy life-style (a low-fat diet, regular exercise, and abstinence from smoking) can help prevent or slowdown the course of atherosclerosis, a predisposing factor in the development of other aneurysms. Hypertension should be carefully controlled to prevent aneurysm formation or extension.

See Chapter 17.

AORTIC VALVE DISEASE

WHAT IS IT?

The aortic valve is one of four valves that control the flow of blood into and out of the heart. In particular, the aortic valve controls the flow of oxygenated blood pumped out of the heart from the left ventricle into the aorta, the main artery leading to the rest of the body. If the valve is abnormally narrow (stenosis), the heart must work harder for a sufficient amount of blood to be pumped out with each beat. On the other hand, if the valve does not close properly, it is called insufficient because some of the blood being pumped out into the aorta regurgitates, or leaks backward, into the left ventricle with each beat. In either case, the work of the ventricle increases. As a result, its muscular wall thickens (a condition known as hypertrophy) and the left ventricle may become larger (dilate).

WHO GETS IT?

A congenitally deformed valve and rheumatic fever are leading causes of aortic valve disease. Aortic regurgitation is occasionally but rarely associated with other types of rheumatoid (joint) disease, such as ankylosing spondylitis, Reiter's syndrome, rheumatoid or psoriatic arthritis, and systemic lupus erythematosus. Severe hypertension in the presence of other structural abnormalities of the valve also may cause aortic regurgitation.

WHAT ARE THE SYMPTOMS?

An aortic valve disorder usually does not cause any symptoms in its early stages. As the problem progresses, it may produce shortness of breath, angina, light-headedness, dizziness, and even fainting, especially upon exertion. Many elderly people with aortic stenosis remain free of symptoms.

HOW IS IT DIAGNOSED?

Most cases of aortic valve disease can be diagnosed by a physical examination, during which such signs as a

characteristic heart murmur can be detected. A chest X-ray, an electrocardiogram (to determine whether the heart is enlarged), and an echocardiogram may also be done. If enough significant symptoms are present to warrant possible surgery, cardiac catheterization may be necessary.

HOW IS IT TREATED?

Blood pressure and weight should be kept as normal as possible. Limitations on strenuous activity (especially lifting heavy objects) are recommended, particularly for those with stenosis. If symptoms are present with severe stenosis or regurgitation, surgery to replace the defective valve, either with a plastic or metal prosthetic device or with a pig valve, may be recommended. In general, individuals with aortic stenosis who are free of symptoms do not need surgery. Vasodilating drugs, used in treating hypertension, may be useful in aortic regurgitation but not in aortic stenosis.

WHAT ARE THE COMPLICATIONS?

In general, individuals with aortic valve disease are at risk for left-sided heart failure and for heart valve infections (infective endocarditis). Aortic stenosis also carries a risk of sudden death, but usually there are plenty of warning symptoms prior to a serious event.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Avoidance of rheumatic fever, particularly by prompt treatment of a strep throat, is a major preventive measure. If aortic valve disease is present, the prophylactic use of antibiotics before any dental extractions or surgery is necessary to prevent infective endocarditis.

See Chapter 13.

ATHEROSCLEROSIS (ARTERIOSCLEROSIS)

WHAT IS IT?

Arteriosclerosis is a general term used to describe conditions in which the walls of arteries thicken and develop a buildup of fatty material on the inner surface and lose elasticity; its common name is "hardening of the arteries." Some hardening is a natural part of aging. More often, it is caused by atherosclerosis, a buildup of fatty deposits, fibrous tissue, and calcium, called plaque, on the interior of the artery walls. Lipids (fats), including cholesterol, are a major component of plaque. Arteriosclerosis can affect any artery. It is of major concern when it affects the arteries of the heart (coronaries), the neck and brain (carotid), and the legs and kidneys, as well as the aorta itself.

WHO GETS IT?

Although hereditary factors predispose people to arteriosclerosis, life-style plays a critical role in its development. Some degree of atherosclerosis is almost always present in middle-aged and elderly people in countries where the typical diet is high in saturated fat. Smokers and individuals who are obese or have hypertension, hyperlipidemia, or diabetes are at greater risk. It is also more common in men than in women, except after the age of 60, when the differences become less.

WHAT ARE THE SYMPTOMS?

Arteriosclerosis may cause no symptoms for many years. When an artery becomes significantly narrowed by plaque deposits, symptoms may occur and will vary according to the vessel involved. For example, when the coronary arteries are affected, symptoms of angina (chest pain) or a heart attack may occur. Narrowing of the carotid arteries may cause symptoms of stroke, such as weakness of an arm or leg, blurred vision, or slurred speech. Narrowing of the blood vessels in the legs may cause calf or thigh pain on walking (intermittent claudication).

HOW IS IT DIAGNOSED?

A certain degree of atherosclerosis maybe presumed in all middle-aged and older adults. Feeling the carotid pulse in the neck or pulses in the feet may provide clues for vessel narrowing. Typical symptoms strongly suggest the diagnosis. The degree and location of narrowing can be diagnosed by angiography (studying the blood vessels after the injection of a dye) or, in the case of the carotid arteries, with certain types of echograms. A stress test may provide indirect evidence of atherosclerosis in the coronary blood vessels.

HOW IS IT TREATED?

There are limited, but encouraging, data to suggest that a major decrease in serum cholesterol level, accomplished through drugs and diet and other life-style changes, can slow and, in a few cases, reverse plaque buildup in the coronary arteries. Reducing elevated blood pressure and smoking cessation are also helpful. Depending on the degree of arterial narrowing and symptoms, medications may be prescribed to dilate blood vessels and help prevent the formation of blood clots. In severe cases, angioplasty may be advised to dilate the narrowed vessels with a balloon or surgery to bypass them with a section of vein or artery taken from elsewhere in the body.

WHAT ARE THE COMPLICATIONS?

Severe arteriosclerosis markedly narrows the artery, impeding the normal flow of blood, which can lead to pain in the legs or chest (angina), heart attack, or stroke, depending on the blood vessels involved. The disorder also increases the risk of thrombosis (the formation of blood clots); such clots may completely obstruct blood flow in the artery involved.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Lifelong heart-healthy habits, such as eating a low-cholesterol, low-fat diet, exercising regularly, and avoiding smoking and obesity, may help prevent arteriosclerosis. If diabetes or hypertension is present, it should be carefully controlled through life-style changes and medical treatment.

See Chapters 2, 11, and 17.

ATHLETE'S HEART

WHAT IS IT?

Athlete's heart is a general term describing a series of changes often seen in the function and structure of the hearts of those who regularly participate in strenuous physical exercise and whose bodies are highly conditioned. These variations, which could suggest illness when seen in nonathletes, are considered normal physiological adaptations when seen in athletes. They enable the heart to deliver a higher than normal level of blood and oxygen to peripheral tissues in the arms and legs in order to sustain athletic performance.

WHO GETS IT?

Highly trained professional athletes, as well as recreational athletes who pursue extended exercise regimens, such as those who train for marathons, are most likely to develop athlete's heart.

WHAT ARE THE SYMPTOMS?

The hallmarks of athlete's heart are bradycardia (a slower than normal heartbeat-usually around 45 to 60 beats per minute), cardiomegaly (overall enlarged heart), and cardiac hypertrophy (thickening of the muscular wall of the heart, usually of the left ventricle). These changes usually occur in the absence of symptoms (such as shortness of breath, excessive fatigue, or chest pain) that would suggest heart disease. The physiological stress of dynamic exercise conditioning causes the heart to enlarge to meet the physical challenges encountered. Because the heart becomes more effective in its pumping ability, it does not need to beat as often to meet these challenges.

HOW IS IT DIAGNOSED?

Simply measuring the pulse indicates the presence of bradycardia. The enlarged heart sometimes maybe observed by physical examination, but is more likely to be detected by X-ray or an ECG. Listening to the heart with a stethoscope may reveal a quiet murmur, indicative of the larger volume of blood being pumped with each beat. An echocardiogram may help to rule out any additional structural heart disease.

HOW IS IT TREATED?

Abnormalities in heart rate, size, and function that derive solely from exercise conditioning normally need not be treated, because they do not represent disease.

WHAT ARE THE COMPLICATIONS?

When such presumed abnormalities are detected in an athlete, it is important to assure that the abnormalities are indeed due solely to exercise conditioning and not to some concurrent cardiac disorder. In particular, special attention must be given to differentiate athlete's heart from various conditions that might cause a slow heartbeat (heart block) or heart enlargement secondary to high blood pressure or a heart valve defect. Because these cardiac disorders may occur in athletes as well as in nonathletes, care must be taken to avoid overlooking them. The greatest complication is misdiagnosis.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Because athlete's heart is not pathological, no steps to prevent or minimize its development are necessary. Indeed, the changes in the heart appear to indicate that it may be more efficient than the heart of the nonathlete.

See Chapter 2.

ATRIAL FIBRILLATION

WHAT IS IT?

Atrial fibrillation is a form of tachycardia or rapid heartbeat. The heart normally beats at a rate of about 60 to 80 beats per minute at rest. In atrial fibrillation, the atria (the upper chambers of the heart) beat very rapidly and totally irregularly at more than 300 beats per minute. Blood is not pumped efficiently to the ventricles. The ventricles (the lower chambers of the heart) usually respond irregularly at rates that range from 100 to 200 beats per minute. As a result, the actions of the two chambers of the heart are completely uncoordinated. The ventricles do not get enough time to fill properly and the atria don't push out enough blood with each beat. The disorder may initially be intermittent, with periods of atrial fibrillation lasting a few minutes, hours, or days alternating with even longer periods of normal heart rhythm. However, atrial fibrillation may also become chronic.

WHO GETS IT?

Atrial fibrillation may occur in the absence of underlying anatomic heart disease. More commonly, however, it occurs in individuals with some form of heart disease, especially in older people with atherosclerosis or hypertension, or in those with valvular heart disease. Less commonly, it may occur in those who have chronic obstructive lung disease, overactive thyroid function, or certain congenital heart defects.

WHAT ARE THE SYMPTOMS?

The most common symptom is awareness of a rapid irregular heartbeat. It may be described as palpitations, or a fluttering sensation in the chest. If the heart rate is very rapid, the individual may feel weak, light-headed, or nauseous, have shortness of breath, or, in unusual cases, even lose consciousness.

HOW IS IT DIAGNOSED?

The presence of a rapid irregular heartbeat can be diagnosed by taking the pulse, listening to the heart with a stethoscope, and testing with an electrocardiogram. Because atrial fibrillation may come and go, there may be no signs of it when the patient visits the physician's office. In this case a portable ECG (Helter monitor), which provides a continuous recording over a 24- or 48-hour period, may be used. This test will obviously miss cases that do not recur during the monitoring period.

HOW IS IT TREATED?

In most people, treatment with medication such as digitalis and/or a beta blocker improves the efficiency of the ventricular contractions by slowing the heart rate and may restore the rhythm to normal. If therapy does not restore normal heart rhythm, supplementary drugs such as quinidine sulfate or procainamide may be prescribed. In some cases, persistent atrial fibrillation may be treated by electrical cardioversion—the administration of an electric shock to the heart while the individual is sedated or anesthetized. Once normal heart rhythm is reestablished, further medication may be prescribed to prevent recurrences, especially if the underlying cause of the disorder is a chronic one that cannot be effectively treated. Anticoagulant medication is frequently employed to decrease the risk of blood clot formation. Correcting an overactive thyroid may prevent further episodes.

WHAT ARE THE COMPLICATIONS?

Those who suffer with atrial fibrillation are at increased risk of blood clots, a serious complication, and therefore may be given anticoagulant medication or chronic aspirin therapy. In those with severe underlying heart disease, the rhythm disturbance may lead to decreased heart function and increasing heart failure.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Measures to prevent valvular and coronary heart disease will decrease the chances of atrial fibrillation. In many cases, however, little can be done to prevent it, but potential triggers should be avoided. These vary from patient to patient, but include cigarettes, caffeine, and alcohol. Medication may be the only way to prevent recurrence if one episode has occurred. Individuals with atrial fibrillation should be under a physician's care in order to receive optimal, regulated medication.

See Chapter 16.

BRADYCARDIA

WHAT IS IT?

The adult heart at rest normally beats at a rate of about 60 to 80 beats per minute. A rate below 55-60 beats per minute is considered slow and is called bradycardia. Infants have a much higher normal rate (110 to 130 beats per minute), and so bradycardia in infants is a rate below 100 beats per minute.

WHO GETS IT?

Slower than average heart rates are normal in people who are physically fit, and are probably normal in all individuals during sleep. Many athletes who train regularly have resting heart rates of 45-60 beats per minute. Bradycardia also may occur secondary to certain illnesses (such as decreased thyroid function, certain gastrointestinal disorders, and jaundice) or to abuse of certain drugs. People with known heart disease (including hypertension) who are being treated with medications that slow the heart, such as beta blockers and certain calcium channel blockers, may experience bradycardia. It may also be a temporary consequence of certain types of heart attack. Bradycardia is common in elderly people, whether or not they suffer from arteriosclerosis, and in infants with certain types of congenital heart disease.

WHAT ARE THE SYMPTOMS?

Bradycardia usually does not cause symptoms unless the heart rate is below 40-45. The condition becomes a concern only if it results in an inadequate output of blood from the heart, producing such symptoms as fatigue, shortness of breath, light-headedness, and fainting. Such symptoms are most likely to occur upon exertion, when the body's need for oxygenated blood increases, but can also occur at rest.

HOW IS IT DIAGNOSED?

A slower than normal heartbeat can be detected simply by taking the pulse. An electrocardiogram will help to define the type of bradycardia and determine whether heart block or another condition is present.

HOW IS IT TREATED?

If the bradycardia does not cause symptoms, no treatment is necessary. If there are symptoms, medications can be given to increase the rate of the heartbeat. If fainting or serious symptoms persist despite medication, a permanent pacemaker may need to be implanted. In specific instances, certain medications may have to be withdrawn because of their slowing effect.

WHAT ARE THE COMPLICATIONS?

Severe bradycardia (fewer than 30 beats per minute) can be an emergency situation, leading to brain oxygen deprivation and convulsions. Death may result unless immediate medical measures are taken to increase the heart rate.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Dosage instructions for heart disease and hypertension medications should be followed carefully. Since the more serious form of bradycardia is due to heart block or damage to heart muscle from a heart attack, measures to slow the process of atherosclerosis (cessation of smoking, control of blood pressure and blood cholesterol, and regular exercise) can be helpful.

See Chapter 16.

CARDIAC ARREST (SUDDEN DEATH)

WHAT IS IT?

Cardiac arrest is the failure of the heart muscle to pump blood because of a severe rhythm disturbance or cessation of all beating activity. If the heart rhythm is not restored within a short time, the condition is fatal.

WHO GETS IT?

Cardiac arrest most commonly occurs in those who have heart disease and develop a severely abnormal heart rhythm known as ventricular fibrillation; this arrhythmia may occur independently or during a heart attack. Others who have problems with lesser arrhythmias, particularly those arising from the ventricles, also may be at risk. In addition, cardiac arrest is frequently the final event of death from many causes. People with non-heart-related problems may go into cardiac arrest following severe sudden blood loss, major burns, severe allergic reactions (anaphylaxis), hypothermia, drug overdose, drowning, or electric shock. Cardiac arrest may also be the result of a complete heart block, in which impulses fail to get through to the pumping chambers of the heart and the muscle fails to contract.

WHAT ARE THE SYMPTOMS?

The person suffering cardiac arrest immediately collapses, loses consciousness, and has no pulse. If breathing continues briefly, it is shallow.

HOW IS IT DIAGNOSED?

The symptoms of cardiac arrest are dramatic and easily observed, even by a layperson. No special diagnostic techniques are required. If pulse is unobtainable and breathing has stopped, the diagnosis is confirmed.

HOW IS IT TREATED?

Immediate resuscitation efforts are necessary to prevent death. If the arrest occurs outside the hospital, a trained layperson can initiate cardiopulmonary resuscitation (CPR) to restore circulation until medical personnel arrive. This procedure involves opening the airway, breathing into the victim's mouth, and compressing the chest at regular intervals. If the arrest occurs in a hospital, resuscitation will be supplemented by advanced life support techniques. Defibrillation with an electric current may be successful in restarting the heart. Intravenous medications may be given. When the victim has stabilized, further care will include diagnosis and treatment of the disorder that caused the cardiac arrest.

WHAT ARE THE COMPLICATIONS?

Even if an individual is resuscitated after cardiac arrest, complications may occur. If resuscitation did not begin promptly (within approximately five minutes), permanent brain damage may result from brain oxygen deprivation during the absence of circulation.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Every effort should be made to prevent or treat any cardiovascular or respiratory disease. If chest pain or tightness suggestive of a possible heart attack develops, immediate care should be obtained in a hospital emergency room. In addition, those at risk for cardiac arrhythmias should be identified and their condition treated. Some individuals with severe heart disease and a history of severe arrhythmias may require an implantable defibrillator (an AICD).

See Chapter 27.

CARDIAC TUMOR

WHAT IS IT?

Abnormal growths known as tumors rarely arise in the heart. Nevertheless, they can develop in the myocardium (the heart muscle itself), the endocardium (its inner lining), or the epicardium (the outer covering of the heart). Primary malignant cardiac tumors are particularly rare and occur mostly in children; most are the type of tumor known as sarcomas. More than 75 percent of primary cardiac tumors, however, are benign. The most common type is a myxoma, a tumor composed of mucouslike tissue, which accounts for half of all primary benign cardiac tumors. Most myxomas arise in the left atrium, although they can occur in any heart chamber. Other benign tumors, made up of muscle or fatty tissue, and cysts of the pericardium (outer heart lining) are also found, but those are rare. When tumors occur in the heart, they are more apt to be secondary tumors—malignancies that originally developed elsewhere and metastasized to the heart.

WHO GETS IT?

Although myxomas can arise at any age, they tend to occur in individuals between the ages of 30 and 60, are more common in women than in men, and may run in families. Secondary cardiac tumors are most likely to be metastasis in people who have lung or breast cancer, lymphoma, or malignant melanoma, although other types of cancer can also spread to the heart.

WHAT ARE THE SYMPTOMS?

Depending on the location and extent of the tumor in the heart, symptoms may include pain, palpitations, fever, and weight loss; symptoms of heart failure (shortness of breath, fatigue, and swollen ankles) may occasionally be noted. Some cardiac tumors may cause no symptoms.

HOW IS IT DIAGNOSED?

Accurate diagnosis is often difficult. Some signs of cardiac tumor, such as a heart murmur (particularly with a myxoma), may be detectable on physical examination. Other tumors that cause no symptoms maybe suspected after a routine X-ray shows heart enlargement. An electrocardiogram and an echocardiogram will often help make the diagnosis. Sometimes cardiac catheterization may be necessary.

HOW IS IT TREATED?

Surgical removal can usually completely cure myxomas. Tumors of the pericardium also may require surgery. Surgical success is less likely, but often possible, for muscle tumors. Only palliative therapy is available for tumors of the heart that metastasize from elsewhere. This therapy may include radiation or chemotherapy to slow the growth of the tumor or reduce its size.

WHAT ARE THE COMPLICATIONS?

Tumors such as myxomas can obstruct normal blood flow in the heart (particularly at the mitral valve) and increase the risk of blood clot formation, leading to a stroke or fainting. Primary malignant tumors often lead to the development of congestive heart failure, fluid collection between the heart muscle and the membrane that surrounds it (pericardial effusion), heartbeat irregularities, or heart block. The prognosis for patients with malignant cardiac tumors is poor.

HOW CAN IT BE PREVENTED OR MINIMIZED?

There are no known measures to prevent the occurrence of cardiac tumors. Early detection through surveillance and routine examination may allow treatment of cancers before they metastasize to the heart.

CARDIOMYOPATHY

WHAT IS IT?

Cardiomyopathy is a general term describing disease of the heart muscle. Primary cardiomyopathy involves changes in the muscle's structure or function from unknown causes. Examples include *congestive cardiomyopathy*, in which the heart enlarges, weakens, and no longer pumps effectively, increasing the risk of heart failure and blood clots; hypertrophic *cardiomyopathy*, in which the heart muscle overgrows and thickens, possibly impeding the flow of blood through the heart; and *restrictive cardiomyopathy*, in which the heart muscle wall stiffens. *Secondary cardiomyopathy* may result from some other systemic disease, metabolic disorders, or infection. Examples include diffuse coronary disease with multiple heart attacks; *alcoholic cardiomyopathy*, in which the muscle is believed to be damaged directly by alcohol and secondary nutritional deficiencies; and viral *cardiomyopathy*, which is caused by a viral infection of heart muscle.

WHO GETS IT?

Although some types of cardiomyopathy are attributable to specific causes in groups of people, such as alcoholics, a common underlying problem appears to be diffuse coronary artery disease. In some cases, no cause can be diagnosed. Hypertrophic cardiomyopathy is rare, but tends to occur more often in young adults and more often in men than in women.

WHAT ARE THE SYMPTOMS?

Except for cardiomyopathy associated with an infection, these disorders—and thus their symptoms—usually develop slowly. The most common symptoms are those of congestive heart failure, such as fatigue, swelling, and shortness of breath. They may be chronic or acute. In some instances, irregularities of heart rhythm may be a prominent symptom.

HOW IS IT DIAGNOSED?

Physical examination may reveal an enlarged heart, a characteristic murmur, or changes in heart sounds. Symptoms may suggest the diagnosis. This plus an ECG, chest X-ray, and possibly an echocardiogram or radionuclide studies will usually provide the information needed for a diagnosis. In some cases, cardiac catheterization and, rarely, a biopsy may be necessary.

HOW IS IT TREATED?

If a treatable underlying cause, such as alcoholism, can be identified, it should be treated. Depending on the type of cardiomyopathy, certain drugs may be prescribed to decrease the heart's workload, regulate the heartbeat,

help prevent blood clot formation, and help prevent fluid accumulation in the body; these drugs include vasodilators, digitalis, ACE inhibitors, anticoagulants, and diuretics. Congestive and dilated cardiomyopathies often respond well, at least initially, to medical therapy. Treatment of some cardiomyopathies that result from viral infections may not be too effective. Therapy for those with restrictive cardiomyopathy may be particularly limited. If end-stage heart failure develops, heart transplantation may be an option.

WHAT ARE THE COMPLICATIONS?

Unless a treatable cause is identified and therapy provided, the outlook for some patients with cardiomyopathy may be bleak. Outlook is, at least in part, dependent upon the degree of cardiac dysfunction. Congestive heart failure commonly occurs. Arrhythmias or a heart block may develop. Heart block may require implantation of a pacemaker. In severe cases of congestive cardiomyopathy, blood clots form in the heart and may travel to other parts of the body and cause a stroke. Sudden death can occur.

HOW CAN IT BE PREVENTED OR MINIMIZED?

It is important to seek medical care early and to embark on an appropriate medical regimen, which should include therapy for any treatable primary causes of cardiomyopathy. Potential causes such as alcohol should be avoided. Hypertrophic cardiomyopathy is most frequently a congenital defect and cannot be prevented.

See Chapters 14 and 15.

CONGENITAL HEART DISEASE-CYANOTIC

WHAT IS IT?

Any defect of the heart or the major blood vessels that is present at birth is called congenital heart disease. In the more severe types of disorders (which, fortunately, are not common), a major symptom is blueness of the infant's skin at birth. Called cyanosis, this blueness indicates that the supply of oxygenated blood in the baby's body is inadequate. The most common of such heart disorders include *tetralogy of Fallot* (four abnormalities in heart structure that impair the normal flow of blood); *transposition of the great vessels* (transposition of the aorta and pulmonary arteries in their attachment to the heart, so that oxygenated blood goes back to the lungs instead of out to the body through the aorta); *tricuspid atresia* (lack of a valve to allow blood flow between the right heart chambers); and *total anomalous venous return* (inability of oxygenated blood from the lungs to

reach the left atrium directly). In each instance, the fundamental problem is an inability to oxygenate blood because of altered cardiac anatomy.

WHO GETS IT?

About one in every 120 babies has some congenital heart defect. The majority of defects, however, are minor and do not cause cyanosis. A defect may occur with an associated genetic abnormality (such as Down syndrome). In some cases an illness (such as rubella) afflicted the mother during fetal heart development, and in some cases medication taken by the mother during pregnancy may have caused the defect. There may be no identifiable cause.

WHAT ARE THE SYMPTOMS?

The primary symptom of cyanotic congenital heart disease is bluish skin. Children with tetralogy of Fallot and transposition of the great vessels also are born with a drumsticklike swelling of the ends of the fingers and toes (termed "clubbed") and will be underdeveloped. If heart failure is present, the baby may have difficulty eating because he or she lacks energy for vigorous sucking. Such an infant tends to cry less than normal and also may experience shortness of breath.

HOW IS IT DIAGNOSED?

Complete evaluation of a cyanotic infant or child will include a physical examination, chest X-ray, electrocardiogram, and echocardiogram. Cardiac catheterization is often required to define the anatomic problem.

HOW IS IT TREATED?

Surgery to partially correct transposition of the aorta and pulmonary blood vessels, so that oxygenated blood can flow into the general circulatory system from the heart, is usually done before the baby is 2 to 3 months old. Further surgery will probably be necessary before the child enters school to create artificial blood vessels and establish normal circulation. Tetralogy of Fallot also requires surgical correction before age 4 or 5, although earlier emergency surgery may be necessary. Supplemental oxygen and medication may be necessary to help tide the baby over until the surgery.

WHAT ARE THE COMPLICATIONS?

Cyanotic congenital heart disease is a severe condition. In the days before effective surgery was available, it resulted in failure to thrive, severe heart failure, or sudden death. Fortunately, today the prognosis with surgery is good, although long-term status into adulthood has not yet been defined after most types of surgery.

HOW CAN IT BE PREVENTED OR MINIMIZED?

If there is any family history of congenital heart disease, genetic counseling before pregnancy should be considered. Women who have not been vaccinated against ru-

bella (German measles) should be vaccinated before becoming pregnant. Unnecessary drugs should be avoided during pregnancy.

See Chapter 20.

CONGENTIAL HEART DISEASE NONCYANOTIC

WHAT IS IT?

Any defect of the heart or the major blood vessels that is present at birth is called congenital heart disease. In the more severe types of disorders, a major sign is blueness of the infant's skin at birth. Called cyanosis, this blueness indicates that the supply of oxygenated blood in the baby's body is inadequate. Congenital heart disease that does not cause bluish skin is called noncyanotic. It is more common and less serious than cyanotic disorders. Noncyanotic disorders include *congenital aortic or pulmonic stenosis* (a narrowing of one or the other of these heart valves), *ventricular or atrial septal defects* (small holes between the heart's lower or upper chambers, yielding an excess of blood circulation in the lungs), *coarctation of the aorta* (an abnormal narrowing that impairs blood flow to the lower part of the body), and *patent ductus arteriosus* (failure of an extra blood vessel between the aorta and pulmonary artery to close down as it should after birth, causing excessive blood flow to the lungs).

WHO GETS IT?

Congenital heart defects occur in about one of every 120 babies. Many defects are minor. They may be caused by an associated genetic abnormality (such as Down's syndrome), by an illness (such as rubella or diabetes) that afflicted the mother during fetal heart development, by some medication taken by the mother during pregnancy, or frequently by some unknown factor.

WHAT ARE THE SYMPTOMS?

Many cases of noncyanotic congenital heart disease do not exhibit symptoms. If mild heart failure is present, the baby may have difficulty eating because of lack of energy for vigorous sucking. Such an infant may not gain weight normally and tends to cry less than normal. With more severe problems, the baby's breathing may be rapid and distressed. If the heart problem is not diagnosed in infancy, symptoms may first arise in young children who probably are growing at a below-normal rate. They may become short of breath upon exertion and, eventually, even at rest.

HOW IS IT DIAGNOSED?

Noncyanotic congenital heart problems can usually be detected by the presence of a heart murmur heard with

a stethoscope. Most heart murmurs heard in childhood are benign and need not cause worry; but other heart murmurs signal the existence of particular types of congenital heart disorders. Further evaluations are likely to include a chest X-ray, electrocardiogram, and echocardiogram. When surgery is contemplated, cardiac catheterization may be necessary.

HOW IS IT TREATED?

Treatment depends upon the type of defect. Initial treatment usually is not needed for congenital aortic or pulmonary stenosis, and surgical correction maybe delayed until late childhood or early adulthood. A severe condition, however, may necessitate immediate surgery. Small holes in the heart (atrial or ventricular septal defect) may not require treatment, or they can be closed in the catheterization laboratory using new techniques; surgical correction of larger holes maybe delayed until after age 4. In general, coarctation of the aorta necessitates surgical correction, usually between the ages of 4 and 8. Sometimes patent ductus arteriosus can be corrected with medication; if not, surgical correction is performed before the child starts school. The use of prophylactic antibiotics before dental work or surgery is recommended for most cases. Children with these defects need not limit their physical activity unless exercise results in excessive fatigue or shortness of breath.

WHAT ARE THE COMPLICATIONS?

Ventricular and atrial septal defects pose a risk of pulmonary hypertension and heart failure if the hole is large and is not repaired until adulthood. Stenosed valves and coarctation of the aorta also may increase the work of the heart and, over time, cause heart failure.

HOW CAN IT BE PREVENTED OR MINIMIZED?

If there is any family history of congenital heart disease, genetic counseling before pregnancy should be considered. Women who have not been vaccinated against rubella (German measles) should be vaccinated before becoming pregnant. Avoidance of illness and drugs during pregnancy is the only known preventive measure. Prior to surgical repair of the defect, symptoms can be minimized by the use of diuretics or digitalis.

See Chapter 20.

CONGESTIVE HEART FAILURE

WHAT IS IT?

In contrast to cardiac arrest, when the heart stops pumping completely, heart failure is a condition in which the heart keeps pumping, but inefficiently, generally because of inadequate heart muscle contraction. Because the heart does not pump a normal amount of blood forward,

pressure may build up in the venous system. This may cause congestion in various tissues in the body. The most common sites of such congestion are the lungs, liver, and ankles, which become swollen.

WHO GETS IT?

The underlying cause of heart failure is damage to heart muscle. The damage may be the result of a variety of factors, including atherosclerosis, a heart attack, valvular heart disease, hypertension, rheumatic fever, elevated blood pressure in the lungs because of lung disease, and, in unusual cases, a congenital heart defect.

WHAT ARE THE SYMPTOMS?

Congestive heart failure often develops slowly, and its most common symptoms are shortness of breath, swollen ankles (edema), and weight gain. Initially, respiratory symptoms may occur only when the individual is exercising or lying flat in bed. However, as heart failure becomes more severe, these symptoms tend to occur even at rest in any position. Some irregularities in heart rhythm may also occur, which can result in palpitations and, less commonly, dizziness or syncope (fainting). Acute pulmonary edema is a form of heart failure that develops suddenly, causing extreme shortness of breath and severe anxiety. Wheezing may develop and be accompanied by a cough that produces frothy, pink phlegm.

HOW IS IT DIAGNOSED?

Diagnosis of congestive heart failure or acute pulmonary edema is largely based on the history of characteristic symptoms and a physical examination. Extra heart sounds and crackling sounds in the lungs (rales) maybe heard on examination. A chest X-ray may reveal evidence of congestion in the lungs and heart enlargement. Other diagnostic tests may include an electrocardiogram or an echocardiogram.

HOW IS IT TREATED?

Therapy involves rest, medications such as diuretics and vasodilators to decrease the heart's workload, and a low-sodium diet to help rid the body of excess fluid. Digitalis is the preferred medication to increase the force of the heart's pumping action. If blood pressure is very high, specific blood-pressure-lowering drugs will also be used. In some cases if a specific treatable cause of the heart failure is identified (e.g., extreme narrowing of a heart valve), surgery may be indicated. In rare cases, if the heart is irreversibly damaged and does not respond to therapy, heart transplantation also maybe an option.

WHAT ARE THE COMPLICATIONS?

If acute heart failure is not treated, the patient may experience respiratory failure, literally drowning in bodily fluid. Fortunately, this is rare. Serious rhythm irregularities may also occur, but can be treated. In some instances, heart failure becomes chronic and does not

respond to therapy. About 34,000 deaths from congestive heart failure occur annually. Many of these, however, occur during the course of a heart attack.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Some causes of congestive heart failure are unavoidable. Other cases may be prevented by early treatment of hypertension and life-style modifications to reduce the risk factors for atherosclerosis. Most cases of congestive heart failure will respond, at least initially, to medical therapy. Rigorous medical care is required.

See Chapter 14.

COR PULMONALE

WHAT IS IT?

Cor pulmonale is a form of secondary heart disease that is the result of abnormally high blood pressure in the pulmonary or lung arteries, known as pulmonary hypertension. The process begins when severe lung disease prevents the individual from getting enough oxygen. In response to the lack of oxygen, the pulmonary arteries—which carry blood from the heart to the lungs—constrict, adding to the pressure. Ultimately they become thickened, further impairing the flow of blood. The heart must work harder to compensate for this poor circulation, and its right side becomes enlarged and thickened. The additional workload can eventually cause right-sided heart failure.

WHO GETS IT?

Cor pulmonale occurs most often in adults who have severe lung disease. These individuals are usually smokers. It can also develop in people with lung disease such as cystic fibrosis, which is not caused by smoking. Risk also may increase in those who are very obese or who live at high altitudes. The pulmonary hypertension that leads to cor pulmonale may be caused by any disorder that impairs the flow of blood through the lungs. More than half of all cases are caused by chronic bronchitis or emphysema or both. Other possible causes include congenital heart disease, pulmonary embolism, primary pulmonary hypertension, certain vascular diseases, and chronic infections, as well as extensive loss of lung tissue because of surgery or trauma.

WHAT ARE THE SYMPTOMS?

Almost all patients have shortness of breath because of the underlying lung disease. Swollen ankles are also common. Other symptoms may be vague or similar to those causing the underlying lung disorder—a chronic cough, various types of chest pain, and drowsiness. The first specific signs indicating a failure of the right side of the heart may not occur until the cor pulmonale is considerably advanced.

HOW IS IT DIAGNOSED?

A complete physical exam may provide important clues. An electrocardiogram and chest X-ray will also be very helpful. Pulmonary function tests and, in some cases, an echocardiogram, a nuclear scan of the heart or lungs or both, and right-sided cardiac catheterization may be necessary to pinpoint the diagnosis. Even under optimal circumstances, however, the diagnosis may be difficult to make.

HOW IS IT TREATED?

Therapy for cor pulmonale is likely to include modified bed rest, supplemental oxygen, and diuretics to help rid the body of excess fluid. If right ventricular failure occurs, digitalis and vasodilating medications may also help. Even after successful treatment, cor pulmonale may cause recurrent problems unless the underlying cause of the pulmonary hypertension is amenable to treatment, which is often not the case. Daily home treatment with oxygen may be necessary on a long-term basis. Ultimately, in very rare and carefully selected cases, combination lung and heart transplantation may be recommended.

WHAT ARE THE COMPLICATIONS?

Although the main risks are associated with the underlying lung disorder, cor pulmonale can lead to heart failure and chronic invalidism.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Medical attention is important for any lung or heart disorder or for any new symptoms in the course of existing lung disorders. Eliminating cigarettes and exposure to smoke and other sources of air pollution is imperative. Some cases of cor pulmonale that result from congenital heart disease may be helped by surgery.

CORONARY ARTERY DISEASE (ISCHEMIC HEART DISEASE)

WHAT IS IT?

Coronary artery disease, coronary heart disease, and ischemic heart disease are various names given to a condition in which the coronary arteries—those that feed the heart muscle itself—are narrowed. As a result, the blood supply to the heart muscle is decreased. The narrowing is almost invariably due to atherosclerosis, the buildup of fatty plaques on the inner walls of the arteries.

WHO GETS IT?

Coronary artery disease affects more than 6 million Americans and is the leading cause of death in the United States. It occurs more frequently in individuals with a

family history of premature (below age 60) heart disease, as well as in those who smoke or have high blood pressure, high blood cholesterol, or diabetes mellitus (each of which is also somewhat influenced by heredity). Obesity, physical inactivity, and stress also play a role in the development of atherosclerosis. Risk rises with age, and men are at greater risk than women, although the female risk rises dramatically within five to ten years after menopause. Life-style changes, medication, or both can modify these risk factors, with the exception of heredity, age, and gender.

WHAT ARE THE SYMPTOMS?

Coronary artery disease may exist for many years without causing any symptoms. The most common symptom is chest pain (angina). In most instances, symptoms are not noticed until artery narrowing has progressed. These may not be symptoms, however, until one of the complications of coronary artery disease, a heart attack, occurs.

HOW IS IT DIAGNOSED?

In the absence of symptoms, coronary artery disease may be diagnosed as a result of positive findings during an exercise stress test (possibly including a nuclear imaging study), or it may be documented by a coronary angiogram. When chest pain on exertion is present, coronary artery disease should usually be considered; tests will be used to confirm the diagnosis or to determine its extent.

HOW IS IT TREATED?

Treatment is complex and must be individualized. It can include the following (ranging from simplest to most complex): life-style modifications medications, including daily aspirin, cholesterol- or blood-pressure-lowering agents, beta blockers, nitroglycerin derivatives, and calcium channel blockers; coronary angioplasty and bypass surgery.

WHAT ARE THE COMPLICATIONS?

The most common and serious complications of coronary artery disease are myocardial infarction (heart attack) and sudden cardiac death. These events are most often precipitated by the formation of a blood clot that obstructs a coronary artery already narrowed by atherosclerosis. Other complications may include various heart rhythm disturbances and heart failure.

HOW CAN IT BE PREVENTED OR MINIMIZED?

The same life-style measures applied for treatment are pivotal in helping prevent or minimize the impact of coronary artery disease. Maintaining ideal weight, exercising regularly, keeping blood pressure within a normal range, eating a low-fat, low-cholesterol diet, and avoiding cigarette smoking are the key elements of coronary artery disease prevention.

See Chapters 2 and 11.

ENDOCARDITIS

WHAT IS IT?

Endocarditis is an inflammation or infection of the endocardium, which is the inner lining of the heart muscle and, most commonly, the heart valves. It is usually caused by a bacterial infection. The bacteria cluster on and around the heart valves; this may impair their ability to function properly. The acute form of endocarditis may cause more severe symptoms, while symptoms of the chronic form may be milder, making it more difficult to diagnose.

WHO GETS IT?

Although bacterial endocarditis may occur in anyone at any time, it is unusual in persons who do not have valvular heart disease. Valves deformed by a previous attack of rheumatic fever were once a major predisposing factor, but this is less so today since rheumatic fever has become much less common. Other predisposing factors include artificial heart valves, some congenital heart disorders, and, infrequently, mitral valve prolapse. People with such risk factors are more likely to develop endocarditis when exposed to an infection from any source. Dental surgery, urologic or gynecologic surgery, colonoscopy, and skin infections increase the risk of endocarditis. Intravenous drug users are at particular risk for development of endocarditis, even if there is no preexisting anatomic valve deformity.

WHAT ARE THE SYMPTOMS?

The symptoms of bacterial endocarditis include a low-grade fever, fatigue, loss of appetite, night sweats, chills, headaches, joint discomfort, and tiny pinpoint-sized hemorrhages on the chest and back, fingers, or toes. Upon examination, the physician also may detect a new heart murmur and small hemorrhages in the mucous membranes of the eyes.

HOW IS IT DIAGNOSED?

Diagnosis is usually suspected based on the patient's history, symptoms, and findings such as a new murmur. It is confirmed by a blood test ("culture") to identify an infecting organism. An echocardiogram may occasionally be helpful in identifying a clump of bacteria on the heart valve.

HOW IS IT TREATED?

Bacterial endocarditis almost always requires hospitalization for antibiotic therapy, generally given intravenously, at least at the outset. Occasionally, therapy with oral antibiotics at home will be successful. Antibiotic therapy usually must continue for at least a month. In unusual cases, surgery may be necessary to eliminate areas of infection or to repair or replace a damaged heart valve.

WHAT ARE THE COMPLICATIONS?

If bacterial endocarditis is not adequately treated, it may be fatal. This is dependent upon the infecting organism. Even when treated, further damage to a heart valve may lead to heart failure. In addition, blood clots may form and travel through the bloodstream to the brain or lungs.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Those who have any predisposing factors for bacterial endocarditis should be given antibiotics before any medical or dental surgery and whenever any significant skin infection occurs. Such prophylactic therapy will help prevent the spread of bacteria to the bloodstream. Those with a prior history of endocarditis must be monitored for at least a year because of the possibility of a relapse or reinfection of a heart valve.

See Chapter 13.

HEART BLOCK

WHAT IS IT?

The heart's electrical system normally sends impulses from the two atria (the upper chambers) to the two ventricles (the lower chambers) in a pattern that causes the coordinated contraction called the heartbeat. If these electrical messages are slowed or interrupted along their normal paths, the heart rate or rhythm can be impaired. There are various degrees of heart block. First-degree heart block is generally of no consequence to the individual and is detected only by an electrocardiogram. In second-degree heart block, occasional beats are not conducted and the pulse becomes a bit slower and somewhat irregular. In third-degree (or complete) heart block, the electrical messages don't get through to the ventricles at all and the atria and ventricles beat independently. Heart rate is usually slow and irregular. Complete heart block can produce symptoms such as syncope (fainting).

WHO GETS IT?

Heart block is most likely to occur in the elderly and those with atherosclerosis or primary myocardial disease [cardiomyopathy]. It can also be seen in individuals with an enlarged heart as a result of untreated hypertension or rheumatic heart disease. An uncommon form is seen in infants (congenital heart block). Alternatively, it can develop as a side effect of certain cardiac drugs that can impair the normal electrical patterns. In some cases of heart block, the cause is never found.

WHAT ARE THE SYMPTOMS?

First- and second-degree heart block generally do not cause symptoms, because the heart rate and rhythm may remain quite normal. In third-degree heart block, there

may be fatigue, light-headedness, fainting, or symptoms of heart failure. Very severe cases may result in sudden death.

HOW IS IT DIAGNOSED?

A slower than normal or irregular heartbeat can be detected simply by feeling the pulse. However, an electrocardiogram will show electrical patterns characteristic of the different degrees of heart block. (Many cases of slow or irregular heartbeat, however, are not a result of a heart block.) If there are symptoms but the heart block is intermittent and not detected on physical examination, the physician may recommend continuous monitoring of the heartbeat with a Helder monitor, a portable device that the patient wears while going about his or her usual daily activities.

HOW IS IT TREATED?

Most cases of first-degree and even second-degree heart block require no treatment, especially if there are no symptoms. If cardiac medications are being used for other purposes, reducing or changing them may occasionally eliminate or reduce the heart block. Chronic complete heart block with symptoms requires implantation of an artificial pacemaker to take over the job of providing regular electrical heart stimulation through the power of a very small, long-lasting battery. Depending on the type of heart block and the type of pacemaker used, it may simply send one regular signal, or may respond only when the heart's own pacemaker fails to function properly. In some cases, it may be programmed to vary the heart rate according to different needs—such as faster for exercise and slower for sleep. Transient third-degree heart block that occurs during a heart attack may require a temporary pacemaker, which can be removed when spontaneous heart rhythm returns to normal.

WHAT ARE THE COMPLICATIONS?

Most people with first- and second-degree heart block can go about their lives without difficulty. In some cases of second-degree heart block and in most cases of complete heart block, there is a danger of fainting, possible convulsions, and, in some cases, death. The risk for persons with first- and second-degree heart block is related more to the underlying disorder—coronary or hypertensive heart disease, etc.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Prevention of atherosclerosis and early treatment of hypertension may help to prevent heart block. Routine evaluation, particularly of the elderly, may uncover heart block before it is symptomatic. If the individual is taking cardiac medication, it maybe altered, or, if appropriate, a pacemaker may be considered. Since many cases of heart block are related to narrowing of the coronary arteries, a healthy life-style that includes a low-fat diet,

regular exercise, and avoidance of smoking may help lower the risk.

See Chapters 16 and 26.

HYPERLIPIDEMIA

WHAT IS IT?

Hyperlipidemia is an excess of fatty substances called lipids, largely cholesterol and triglycerides, in the blood. It is also called hyperlipoproteinemia, because these fatty substances travel in the blood attached to proteins; the fat-protein complexes are called lipoproteins. The best-known lipoproteins are low-density lipoprotein (LDL) and high-density lipoprotein (HDL); another is very-low-density lipoprotein (VLDL). LDL, which carries most of the body's cholesterol in the blood, is known as the "bad" lipoprotein, because it tends to carry serum cholesterol from the liver to the arteries, where it forms plaques on arterial walls. HDL is known as the "good lipid, because it tends to carry cholesterol away from arterial walls and back to the liver. A subcategory of hyperlipidemia is hypercholesterolemia, in which there is a high level of total cholesterol. (VLDL largely contains triglycerides).

WHO GETS IT?

Some types of hyperlipidemia, such as familial hypercholesterolemia, are hereditary. Others may occur secondary to diseases such as diabetes, nephrosis, hypothyroidism, and alcoholism. The most common type of hypercholesterolemia is believed to be due largely to an interaction between genetic factors and excess cholesterol and fat, especially saturated fat in the diet.

WHAT ARE THE SYMPTOMS?

Hyperlipidemia usually has no overt symptoms and tends to be discovered during routine examination or evaluation for atherosclerotic cardiovascular disease. Pinkish-yellow deposits of fat (known as xanthomas) may develop under the skin (especially around the eyes) in individuals with familial forms of the disorder or in those with very high levels of cholesterol in the blood.

HOW IS IT DIAGNOSED?

Diagnosis is made by evaluating laboratory analyses of blood samples, which provide information on total blood fat levels, as well as its fractions. Cholesterol and triglyceride levels tend to rise with age. A total serum cholesterol level under 200 mg/dl, with LDL below 130 mg/dl and HDL above 35–40 mg/dl, is considered normal for adults. If HDL levels are particularly high, a higher total serum cholesterol level may be acceptable. This is more common among women, who may have HDL levels of 60–70 mg/dl. While their total cholesterol level may

be above 220–240 mg/dl, their risk for heart disease is not high. Triglyceride levels above 250 mg/dl are considered abnormal,

HOW IS IT TREATED?

A low-cholesterol, low-fat diet is the first line of therapy for hypercholesterolemia. A low-carbohydrate diet, eliminating alcohol, is recommended for hypertriglyceridemia. Maintaining ideal weight and increasing exercise may help both conditions. If life-style measures do not provide sufficient benefit, drug therapy may be necessary. A number of classes of cholesterol-lowering drugs are available.

WHAT ARE THE COMPLICATIONS?

Hyperlipidemia predisposes the individual to coronary heart disease and other vascular diseases. The risk tends to rise in direct correlation with increased levels of blood lipids and becomes substantially greater if other risk factors, such as cigarette smoking and high blood pressure, are present. Those with familial types of severe hypercholesterolemia are at risk of coronary heart disease early in life if the disorder is not diagnosed and treated.

HOW CAN IT BE PREVENTED OR MINIMIZED?

A healthy life-style, including a low-fat, low-cholesterol diet, regular exercise, and maintenance of desirable weight, can often prevent or minimize hyperlipidemias. Treatment of diabetes, if present, may also help to lower certain of the fats in the blood.

See Chapters 3,4, and 5.

HYPERTENSION (HIGH BLOOD PRESSURE)

WHAT IS IT?

As blood circulates in the body, pressure is exerted against the inner walls of arteries. The level of that pressure is reported in two numbers: The systolic (the higher number) is the pressure of the blood on the artery walls when the heart beats (the pumping pressure); the diastolic (the lower number) is the pressure in the arteries between heartbeats (the resting pressure). A normal blood pressure in adults is between 110/70 and 140/90 mm Hg. Although blood pressure tends to fluctuate within this normal range, a persistent elevation of blood pressure above 140/90, regardless of age, is considered hypertension.

WHO GETS IT?

Essential, or primary, hypertension (for which the cause remains unknown) is more common in those over 40. It

accounts for over 90 percent of cases. It tends to run in families and afflicts men and women equally, but it is more common in blacks than in whites. The far less common secondary hypertension is more likely to occur in younger people. When the underlying disorder is treated, the hypertension usually disappears. The use of oral contraceptives may result in high blood pressure, but this is rare. Elevated blood pressure occurs in about 5–10 percent of pregnancies, but it usually disappears afterward.

WHAT ARE THE SYMPTOMS?

Hypertension usually causes no symptoms unless it is severe. Contrary to popular belief, headaches are not common, although an early-morning headache in the back of the head may signal an elevated pressure. Dizziness may also be noted.

HOW IS IT DIAGNOSED?

Blood pressure is easily measured with an inflatable cuff attached to a sphygmomanometer. Because a variety of everyday circumstances (including anxiety in the doctor's office) can temporarily affect blood pressure, the diagnosis of hypertension should be made only after repeated readings. The exception is a very high pressure (150/105 to 170/110 mm Hg or higher). In this case, a diagnosis can be made on the basis of the first one or two recordings.

HOW IS IT TREATED?

Mild or borderline hypertension (140/90 to 160/100 mm Hg) usually is treated first with life-style modification, including reducing weight to ideal levels, stopping smoking, reducing salt and fat in the diet, exercising regularly, avoiding excessive alcohol and caffeine, and learning relaxation and stress reduction techniques. If these measures prove ineffective in reducing pressure to a normal level, drug therapy is usually recommended. Moderate to severe pressure (above 160/100) is more likely to be treated early on with medication in addition to life-style modification. Drugs that maybe used alone or in combination include beta blockers, diuretics, angiotensin-converting enzyme (ACE) inhibitors, and calcium channel blockers.

WHAT ARE THE COMPLICATIONS?

The complications of prolonged untreated hypertension include stroke, heart attack, heart failure, retinal hemorrhages, and kidney failure. Untreated hypertension can dramatically decrease life expectancy.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Even individuals with a family history of hypertension may be able to prevent it by maintaining ideal weight and eating a low-salt diet. Blood pressure should be checked in adolescence and early adulthood. If the reading is normal, testing should be repeated every three years. If there is a family history of hypertension, initial

testing should begin earlier, and, in adulthood, should be repeated annually. Women who are pregnant or who use oral contraceptives should have more frequent checks. Life-style modification and drug therapy have greatly reduced the risks of complications associated with hypertension.

See Chapter 12.

KAWASAKI DISEASE

WHAT IS IT?

Also known as mucocutaneous lymph node syndrome, Kawasaki disease is a very rare disease of children that can affect the skin, mucous membranes, lymph glands, joints, heart, and coronary arteries. In the coronary arteries, the condition can cause dilation of the blood vessels and coronary artery aneurysm. The initial illness usually lasts only 2 to 12 weeks, but relapses may occur and damage to coronary arteries may be permanent.

WHO GETS IT?

Kawasaki disease occurs primarily in children under age 10, with 80 percent of those stricken under age 5. It is more common in boys than in girls and much more common in Asians (especially Japanese) than other races. Although the cause is unknown, Kawasaki disease does not appear to be hereditary or contagious. A virus is the likely cause.

WHAT ARE THE SYMPTOMS?

Kawasaki disease begins with a high fever, irritability, lethargy, swollen lymph glands in the neck, and, in some cases, colicky abdominal pain. Within a day or two a red rash appears on the trunk. In the next few days, the lips, tongue, and other mucous membranes take on a reddish color. Hands and feet swell, and skin on the palms and soles becomes red and then scaly, and then peels. Some patients also develop muscle or joint pain, diarrhea, pneumonia, or meningitis. When heart-related problems occur, they usually develop around the tenth day of illness when other symptoms are disappearing. The most common cardiac problem is inflammation of the coronary arteries, which occurs in about 20 percent of all cases; others include inflammation of heart muscle (myocarditis) or the lining around the heart (pericarditis), arrhythmias, and heart valve dysfunction. Most heart problems disappear within six weeks, but permanent coronary artery damage can result.

HOW IS IT DIAGNOSED?

Because there is no one test to reveal Kawasaki disease, the diagnosis is based on the presence of the critical symptoms and the exclusion of other possible disorders. Cardiac problems are diagnosed using an electrocardiogram and an echocardiogram.

HOW IS IT TREATED?

There is no specific treatment for the disease. Aspirin, given to reduce fever and pain and to help prevent blood clots, also may help reduce the risk of damage to coronary arteries.

WHAT ARE THE COMPLICATIONS?

As a result of inflammation, aneurysms (bulges or blisters) can form in the coronary arteries; if a blood clot forms in that area, myocardial infarction (heart attack) may occur. Less commonly, the aneurysm may burst. In about 1 percent of cases, death occurs, usually related to heart complications.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Since the cause is unknown, Kawasaki disease cannot be prevented. Some research has suggested that intravenous gamma globulin, if administered early in the course of illness, maybe effective in preventing coronary artery problems. Those who have developed heart-related problems because of Kawasaki disease should have regular medical check-ups. Aspirin therapy should be continued as long as a coronary aneurysm is present.

See Chapter 20.

LEFT VENTRICULAR ANEURYSM

WHAT IS IT?

The term aneurysm generally refers to an outward bulge in the wall of a blood vessel. Although aneurysms are more common in arteries, they sometimes arise in the left ventricle, or lower (pumping) chamber, of the heart. The portion of the ventricle bulges outward, deforming the shape of the heart, as well as not contracting well when the heart normally squeezes blood out to the body.

WHO GETS IT?

Left ventricular aneurysms usually arise as a result of a severe heart attack. In about 10-20 percent of heart attacks in which a substantial amount of the heart wall muscle dies, an aneurysm may form in the ventricle within a few days. It is often not detected until later on, when complications might occur.

WHAT ARE THE SYMPTOMS?

Ventricular aneurysms usually do not cause pain or specific symptoms. They set the stage, however, for the development of ventricular arrhythmia (tachycardia), heart failure, and the formation of thrombi (blood clots) in the heart, which may break loose and travel to other parts

of the body. The symptoms of heart failure, especially shortness of breath, or an arrhythmia may indicate that a ventricular aneurysm is present after a heart attack.

HOW IS IT DIAGNOSED?

Aneurysms may be difficult to diagnose, because many produce no symptoms. A chest X-ray, echocardiogram, and radionuclide scan maybe used, in addition to physical examination, to diagnose the aneurysm and define its size. Cardiac catheterization and angiography is most helpful and definitive in making a diagnosis.

HOW IS IT TREATED?

Treatment is generally focused on specific problems associated with aneurysm, such as heart failure, cardiac arrhythmia, and blood clots. Ventricular aneurysms may require surgical removal if heart failure or arrhythmias cannot be effectively treated with drugs. In the case of arrhythmias an implantable defibrillator (AICD) maybe placed at the time of heart surgery.

WHAT ARE THE COMPLICATIONS?

Unlike other aneurysms, left ventricular aneurysms are not usually at risk of rupture. Because of the thinned and damaged heart muscle, however, arrhythmias and congestive heart failure are potential complications. A blood clot may form in the aneurysm. This presents a risk of embolization to other parts of the body and can cause additional complications such as stroke. Many people may live for years with a ventricular aneurysm.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Measures to reduce the risk of a heart attack—low-fat diet, regular exercise, smoking avoidance, and careful control of high blood pressure and diabetes, if present—can help lower the risk of a ventricular aneurysm. Effective, early treatment of a heart attack may also be helpful.

See Chapter 11.

MARFAN SYNDROM

WHAT IS IT?

The Marfan syndrome is an inherited disorder of connective tissue that affects the heart, blood vessels, lungs, eyes, bones, and ligaments. When the heart is affected, the heart valves may be oversized and may function improperly, permitting a partial backward flow of blood (aortic, mitral, or tricuspid regurgitation) and resulting in a heart murmur. When the aorta (the body's main artery, which carries all blood exiting the heart) is af-

ected, it may enlarge and/or split in one or more places, leaking blood into the chest or abdomen. This is known as a dissecting aortic aneurysm.

WHO GETS IT?

The Marfan syndrome is caused by an abnormal gene, inherited from one parent, that is believed to produce a defect in one of the proteins that make up connective tissue. Although the Marfan syndrome usually runs in families, the abnormal gene can result from a mutation. A rare disorder, the Marfan syndrome affects only about 25,000 Americans.

WHAT ARE THE SYMPTOMS?

Symptoms may be present at birth, may not appear until later in life, even in adulthood, or may never be experienced. All the possible signs of the Marfan syndrome (which may range from mild to severe) are rarely present in one person, nor are they limited to those with this syndrome. However, people with the Marfan syndrome usually are tall and slender, with long, thin arms and legs, loose joints, and long, thin fingers and toes. Other skeletal manifestations may include flat feet, a spinal curvature, a deformed breastbone, and a highly arched palate. Eye symptoms may include nearsightedness and an off-center lens. Cardiovascular symptoms depend upon the cardiovascular abnormalities involved; they may not be present or may include breathlessness, fatigue, palpitations, and fainting. If aortic dissection occurs, there may be a sudden onset of severe chest pain or cardiac collapse.

HOW IS IT DIAGNOSED?

No single test can diagnose the Marfan syndrome, but often the individual's appearance is quite typical. A complete examination will search for all possible signs of the disorder. Because eye lens dislocation rarely occurs in other disorders, even a subtle dislocation is an important diagnostic feature. It is detectable only by dilating the pupils for ophthalmologic examination. An electrocardiogram or other tests to detect cardiovascular abnormalities may also help confirm the diagnosis.

HOW IS IT TREATED?

The Marfan syndrome cannot be cured, but its symptoms can be treated. Treatment depends upon how the individual is affected. For cardiovascular problems, beta blockers or other drugs may be prescribed to regulate blood pressure and heart rhythms. In some cases a heart valve or a part of the aorta may be replaced surgically.

WHAT ARE THE COMPLICATIONS?

The greatest threat is the possibility of a sudden split (dissection) of the aorta, which can cause death if not identified and treated immediately with surgery.

HOW CAN IT BE PREVENTED OR MINIMIZED?

No test is yet available to determine if an unborn child has the Marfan gene. Genetic counseling can help affected families understand their risks. Regular medical checkups, at least yearly, are advised to monitor progression of the disorder so that appropriate treatment can be initiated. Antibiotics must be taken before dental or medical surgery to reduce the risk of endocarditis. Daily activities should be tailored to reduce heart strain; heavy exercise, contact sports, and lifting heavy objects should be avoided. Individuals who show early signs of the Marfan syndrome involving the first part of the aorta should be evaluated regularly by X-ray and echocardiography.

See Chapters 13 and 17.

MITRAL VALVE DISEASE P R O L A P S E

WHAT IS IT?

The mitral valve is one of four valves that control the flow of blood into and out of the heart. In particular, the mitral valve controls the flow of freshly oxygenated blood from the left atrium (upper heart chamber) into the left ventricle (lower heart chamber), from where it is pumped out into the body. If the valve is deformed, one or both of the leaflets—the flaps that open and close to form the valve—may bulge (prolapse) into the atrium during each heartbeat. In addition, a small amount of the blood that is supposed to enter the ventricle may regurgitate, or leak backward into the atrium. A characteristic clicking sound and/or murmur can be heard when listening to the heart with a stethoscope. Depending on the extent of the regurgitation, the heart may have to work harder to assure that an adequate amount of blood is circulated to all the body tissues.

WHO GETS IT?

Mitral valve prolapse is almost invariably congenital and present at birth but not usually detected until later. This common disorder occurs much more frequently in women and is particularly common in those who have a narrow, concave chest cavity and other skeletal abnormalities. The syndrome frequently is detected in teenage girls or women in their 20s and 30s, and it has been estimated that as many as 10 to 15 percent of the young female population may have this condition.

WHAT ARE THE SYMPTOMS?

Most people with mitral valve prolapse have no symptoms. When symptoms do occur, they are most likely to include palpitations, shortness of breath, and atypical,

sticking chest pains that may occur at rest. In cases of significant regurgitation, heart failure may develop, but is rare.

HOW IS IT DIAGNOSED?

Some signs of mitral valve prolapse, such as the characteristic clicking sound and a heart murmur heard with a stethoscope, are detectable during a physical examination. An evaluation is likely to include a chest X-ray, an electrocardiogram, and an echocardiogram. Most of the time, however, the diagnosis can be made without specific tests.

HOW IS IT TREATED?

Most often, treatment for mitral valve prolapse is not necessary. If symptoms develop and interfere with the enjoyment of life, beta-blocking drugs may be helpful in relieving palpitations or chest discomfort. Individuals with signs of severe mitral valve prolapse may be advised to avoid strenuous competitive sports. Prophylactic antibiotics may be recommended prior to dental or surgical procedures to prevent endocarditis.

WHAT ARE THE COMPLICATIONS?

Serious complications are rare among those with mitral valve prolapse, but may include a greater risk of blood clot formation, and, very rarely, sudden death. Individuals with mitral valve prolapse also are at greater risk of infective endocarditis (inflammation of the lining of the heart) and problems associated with mitral regurgitation, including heart failure.

HOW CAN IT BE PREVENTED OR MINIMIZED?

There are no known methods of preventing mitral valve prolapse. It is a fairly widespread but benign condition, except in extremely unusual circumstances.

See Chapters 2 and 13.

MITRAL VALVE DISEASE STENOSIS AND REGURGITATION

WHAT IS IT?

The mitral valve controls the flow of freshly oxygenated blood from the left atrium (an upper heart chamber) into the left ventricle (a major lower heart chamber), from where it is pumped out into the body. If the valve is stenosed (narrowed), the amount of blood that is pushed into the left ventricle is diminished. On the other hand, if the valve does not close properly, it is called incompetent (regurgitant or insufficient), because some of the blood that is pushed through the valve into the left ventricle regurgitates, or leaks backward, into the atrium

with each beat. In either case, the heart must work harder to try to pump an adequate amount of blood to the brain, kidneys, and other parts of the body. In response to regurgitation, the left ventricle and the chamber dilate. This can result in elevated pressure in the heart and, ultimately, heart failure. In mitral stenosis, pressure builds within the left atrium and is passed back through the pulmonary veins, leading to congestion in the lungs and, in severe cases, pulmonary edema.

WHO GETS IT?

Mitral stenosis is almost invariably caused by rheumatic fever, although a very small number of cases are congenital. Although mitral regurgitation also is frequently due to rheumatic fever, it also may be associated with various heart muscle disorders, as well as conditions such as mitral prolapse. Mitral regurgitation may follow a heart attack if the part of the heart muscle to which the valve structures are attached is damaged by the attack.

WHAT ARE THE SYMPTOMS?

Mitral valve disorders may not cause symptoms for many years. In the meantime, however, the burden on various chambers of the heart may result in a diminution of heart function. Lung congestion may result in shortness of breath, especially after exercise or when lying flat in bed. Pressure on the bronchial tree by the enlarged atrium may cause chronic coughing. Both stenosis and regurgitation can cause swollen ankles and marked fatigue. Sometimes easy fatigue is the only symptom suggesting that the valve disorder is resulting in poor heart function.

HOW IS IT DIAGNOSED?

Mitral valve disease may be diagnosed during a physical examination when signs such as specific types of heart murmur are detected. A chest X-ray, an electrocardiogram, and an echocardiogram will help confirm the diagnosis, delineate heart size, and help define the exact extent of the valve abnormality. For advanced cases, cardiac catheterization is usually indicated.

HOW IS IT TREATED?

No treatment is necessary for patients who remain free of symptoms. Prophylactic use of antibiotics prior to dental work or surgery is necessary to prevent endocarditis (infection of the lining of the heart valves). If an irregular heart rhythm like atrial fibrillation is also present, anticoagulant drugs may be prescribed to help prevent blood clot formation. Beta-blocking drugs and digitalis or quinidine may be used to slow the heart rate or to restore a normal rhythm in those subjects with mitral stenosis who develop atrial fibrillation. Breathlessness may be treated with diuretics to decrease fluid buildup. Drugs called "afterload reducers" that decrease the heart's work may be effective in alleviating some of the symptoms that may be noted with mitral regurgitation. If symptoms persist in cases of mitral stenosis, a

surgical procedure called valvulotomy maybe used to widen the valve, or surgery to replace the valve may be advised.

WHAT ARE THE COMPLICATIONS?

Individuals with mitral valve disease are at risk for heart failure and endocarditis. The long-term complications of both stenosis and regurgitation include atrial fibrillation, a rhythm disturbance that may be associated with the formation of blood clots within the atria. This arrhythmia increases the risk of a stroke, because a blood clot may break loose and travel through the bloodstream to lodge in various arteries.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Avoidance of rheumatic fever by prompt treatment of a strep throat is the major preventive measure. If mitral valve disease is present, the prophylactic use of antibiotics before dental extractions or surgery can help prevent infective endocarditis.

See Chapters 2 and 13.

MYOCARDIAL INFARCTION

WHAT IS IT?

Myocardial infarction is the medical term for a heart attack. An infarct (an area of dead or dying tissue) occurs in the myocardium (heart muscle) when there is a marked decrease in the oxygen supply to an area of the muscle. In more than 90 percent of cases, this decrease is caused by an obstruction or closure of one of the coronary arteries, caused by a blood clot blocking an artery narrowed by atherosclerosis. Less commonly, the obstruction may be caused by an arterial spasm, which also closes off the blood flow.

WHO GETS IT?

The highest incidence of myocardial infarction is in middle-aged and elderly men. The incidence in women rises about five to ten years after menopause. About 45 percent of all individuals who experience a heart attack are under age 65; 5 percent are under 40. Heart attacks are more common in those who smoke, are obese, or have high blood cholesterol levels, high blood pressure, diabetes, or a family history of arteriosclerosis at an early age (before age 65). A small number of heart attacks occur in people who have none of these risk factors.

WHAT ARE THE SYMPTOMS?

The most common symptoms of a heart attack are a feeling of pressure, tightness, squeezing, or pain in the center of the chest, lasting at least 5-15 minutes and less commonly for more than an hour. The discomfort may

spread to the shoulders, neck, jaw, or arms, particularly radiating down the left arm. Pain may or may not be accompanied by sweating, nausea, light-headedness, or shortness of breath. In many people, the symptoms of heart attack are mistaken for indigestion. Further, in about 20 percent of heart attacks, there are no noticeable symptoms ("silent" heart attacks).

HOW IS IT DIAGNOSED?

Heart attack should be suspected and medical attention sought whenever an adult experiences unexplained chest pain or pressure. Heart attacks are usually diagnosed based on the patient's symptoms and an evaluation of heart function by examination with a stethoscope, as well as measurements of blood pressure and pulse. An electrocardiogram and blood tests (cardiac enzymes) will usually, but not invariably, confirm the diagnosis. Initial therapy is usually based on these evaluations. Follow-up examination to assess the extent of the heart attack and heart damage may include echocardiography, a stress test, nuclear scans, and coronary angiography.

HOW IS IT TREATED?

All heart attacks require urgent medical treatment. Many can be aborted and heart muscle damage minimized if the individual immediately seeks emergency care and is treated with drugs (such as tissue plasminogen activator, streptokinase, APSAC, or urokinase) that may dissolve the blood clot. To be maximally effective, these "thrombolytic" drugs must be started within two to four hours from onset of symptoms. Other treatment may include medications to alleviate pain, to stabilize abnormal heart rhythms, to dilate blood vessels, to lower the heart's workload, and to decrease the risk of further blood clot development. Follow-up care may include angioplasty to open narrowed vessels or cardiac surgery to provide adequate blood supply. In addition, life-style changes (cessation of smoking, regular exercise, and diet modifications) may be recommended as appropriate.

WHAT ARE THE COMPLICATIONS?

Severe arrhythmias, heart failure, shock, and cardiac arrest are potentially life-threatening complications of a heart attack. With improved early treatment, these complications are becoming much less frequent. Rarely, the heart muscle may rupture, requiring immediate surgery.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Appropriate life-style changes that reduce the risk of atherosclerosis may help prevent a heart attack. These include stopping smoking, eating a low-fat diet, losing excess weight, and controlling blood pressure and diabetes. For individuals who have had a heart attack, routine use of aspirin is generally advised. The need to recognize symptoms of a heart attack and seek immediate treatment cannot be overemphasized. It may be lifesaving.

See Chapter 11.

MYOCARDITIS

WHAT IS IT?

Myocarditis is an inflammation of heart muscle—the muscle that contracts to pump blood out of the heart and relaxes to allow its return. This inflammation can seriously impair both the pumping action and the electrical activity of the heart. Consequently, myocarditis can result in congestive heart failure and arrhythmias.

WHO GETS IT?

Myocarditis is uncommon, but can occur in virtually anyone. The inflammation is a complication of a variety of infectious diseases, most commonly the Coxsackie Type B virus. It also can arise as a result of infection with other viruses, bacteria, parasites, or fungi. Less commonly, myocarditis develops after exposure to certain drugs, arsenic, or other toxic chemicals, or as a complication of some metabolic, granulomatous, or connective tissue disorders.

WHAT ARE THE SYMPTOMS?

Symptoms of myocarditis vary widely. In adults, they can sometimes mimic those of a heart attack—mild to severe pain in the center of the chest, which may radiate to the neck, shoulders, and upper arms. In severe cases, symptoms include breathlessness, rapid pulse, and heart arrhythmias. In infants, symptoms also may include bluish skin, heart murmurs, and a poor appetite.

HOW IS IT DIAGNOSED?

Myocarditis maybe suspected whenever chest pain or arrhythmia symptoms suggestive of congestive heart failure occur during the course of an infectious illness, especially a viral one. It should also be suspected when such symptoms occur in the absence of an obvious diagnosis. Diagnosis may require blood tests, a chest X-ray, electrocardiogram, echocardiogram or radio-nuclide angiocardigram, and, in rare cases, biopsy of a tissue sample from the heart muscle.

HOW IS IT TREATED?

Mild, viral-related myocarditis in adults often cures itself with little or no direct treatment. Similarly, mild cases caused by other types of infection often require only taking antibiotics or other drugs to treat the underlying disease. More severe myocarditis may cause marked heart arrhythmias and heart failure if inflammation sufficiently damages the heart muscle or myocardium. In such cases, medications to stabilize heart function may be necessary. These may include vasodilators, digitalis, diuretics, ACE inhibitors, and other drugs. In certain severe types of myocarditis, steroids maybe prescribed. Sometimes even after myocarditis is resolved, the heart muscle remains permanently damaged. If a heart block

or marked slowing of the heart rate occurs, a pacemaker may be required. In advanced, severe cases, cardiac transplantation may be the only alternative.

WHAT ARE THE COMPLICATIONS?

In severe cases, myocarditis can lead to heart failure and even death.

HOW CAN IT BE PREVENTED OR MINIMIZED?

There are only a few known measures to reduce the occurrence of this rare disease. Avoiding exposure to infectious diseases and having any such illness treated promptly may help. Should myocarditis occur, bed rest is usually required until the inflammation subsides. During this time, alcohol, salt, and any other substances that may increase the heart's work or irritate it further should be avoided.

See Chapter 15.

PERICARDITIS

WHAT IS IT?

The heart is wrapped in a cellophane-like bag or membrane called the pericardium. Pericarditis is an inflammation of this membrane. There are two major types: acute and chronic constrictive pericarditis.

WHO GETS IT?

Acute pericarditis comes on suddenly and may be caused by a bacterial, viral, or fungal infection, or it may occur in association with certain diseases, such as rheumatic fever, rheumatoid arthritis, systemic lupus erythematosus, scleroderma, chronic kidney failure, and tumors. It may also be precipitated by a heart attack or a serious chest injury. A form of pericarditis may also be noted within several weeks after heart surgery. Chronic pericarditis, which is uncommon, develops slowly and may be caused by a chronic infection, such as tuberculosis.

WHAT ARE THE SYMPTOMS?

Acute pericarditis usually causes pain in the center of the chest, which may radiate to the neck or left shoulder. Unlike angina or heart attack, this pain maybe “sticking” in nature and worsens with deep breathing, coughing, or twisting of the upper body. Nevertheless, the pain at times may mimic that of a heart attack. When acute pericarditis is triggered by an infection, fever, chills, and weakness also tend to occur. Chronic pericarditis may not cause any symptoms until the long-term inflammation of the pericardium causes it to thicken and contract to the point where it interferes with normal heart filling. (This condition is known as constrictive pericarditis.) Pain may not be a prominent symptom, but symptoms

that mimic heart failure may develop, including shortness of breath and edema (accumulation of fluid in the legs and abdomen), swelling in the abdomen because of fluid (ascites), and swelling of the liver.

HOW IS IT DIAGNOSED?

The patient's history may be sufficient to make a diagnosis. Characteristic sounds heard through the stethoscope (a rubbing sound), an electrocardiogram, a chest X-ray, and an echocardiogram may be necessary to confirm the diagnosis of acute pericarditis. Additional tests to identify the cause of the pericarditis may include blood cultures, skin tests, and, depending on the individual case, sampling of the fluid in the sac surrounding the heart, or (rarely) a biopsy of the pericardium itself. Diagnosis of chronic obstructive pericarditis generally requires cardiac catheterization.

HOW IS IT TREATED?

Analgesics, ranging from aspirin to morphine, as well as anti-inflammatory drugs may be given to ease the pain or reduce the inflammatory reaction of acute pericarditis. No further treatment may be necessary for pericarditis caused by a viral infection, which tends to clear by itself within a few weeks. If an underlying treatable cause for the pericarditis can be identified, further treatment will be directed toward its alleviation. Antibiotics may be given for a bacterial infection, while steroids such as cortisone may be given in other cases, and non-steroidal anti-inflammatory agents such as indomethacin in still other cases. Steroid drugs may also be prescribed to reduce the inflammation in pericarditis resulting from a heart attack. Diuretics and a salt-restricted diet are also recommended for constrictive pericarditis. In severe cases, surgery may be necessary to remove the thickened pericardium.

WHAT ARE THE COMPLICATIONS?

The major complication of acute pericarditis is pericardial effusion, in which fluid collects in the sack between the pericardium and the heart. If a large amount of fluid collects, the result may be cardiac tamponade, in which the return of blood to the heart from the veins is severely impaired, resulting in a fall in blood pressure. In such cases, the pericardial fluid must be removed by needle aspiration. This is usually a relatively easy and safe procedure. The major problems with chronic pericarditis include congestive heart failure with symptoms that mimic liver or kidney failure. Such complications may require surgical intervention to remove the pericardium.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Prompt treatment of any infection or other condition affecting the lining of the heart or other organs may help prevent this disorder. In many cases it cannot be prevented; on the other hand, the majority of cases are uncomplicated and of short duration. Prompt diagnosis and therapy with anti-inflammatory agents can help minimize the symptoms of pericarditis.

PERIPHERAL VASCULAR DISEASE

WHAT IS IT?

Peripheral vascular disease is a disorder in which the blood supply to the legs or arms is impaired; when normal blood flow is limited, pain may occur. This pain, called intermittent claudication, occurs most often with walking or similar exercise of the legs and is akin to angina, the chest pain that occurs when the heart muscles do not receive enough blood. Peripheral vascular disease is caused by the build-up of fatty deposits, known as atherosclerotic plaque, on the interior surface of the large arteries of the extremities (especially the legs, thus narrowing the channel through which blood can circulate. It is also known as arteriosclerotic obliterans, peripheral atherosclerotic disease, and angina of the leg.

WHO GETS IT?

Peripheral vascular disease primarily occurs in those who are middle-aged or elderly. At greater risk are individuals who already have atherosclerosis elsewhere, or who are at high risk of developing it those who smoke, have diabetes, high blood cholesterol, hypertension, and a family history of cardiovascular disease and are overweight.

WHAT ARE THE SYMPTOMS?

Pain that occurs upon exercise and ceases with rest is the classic symptom. The discomfort can range from mild aching to cramps to severe pain. Pain is usually centered in the calf, but also can arise in the thigh, hip, or buttocks. In severe cases, pain occurs with minimal exercise or at rest, and there may be ulceration of the skin. In some circumstances, impotence may occur in males, and the legs may feel cool to the touch. In unusual cases, peripheral vascular disease can cause pain in the arms during exercise.

HOW IS IT DIAGNOSED?

Physicians often diagnose the condition solely on the description of symptoms and by finding a reduced or absent pulse on examination of the leg arteries. Ultrasound can also be valuable in making the diagnosis. If surgery is being considered, arteriography may be recommended to confirm the precise location and severity of the arterial narrowing.

HOW IS IT TREATED?

Various medications may be prescribed to dilate blood vessels and help prevent blood clots. Unfortunately, drug treatment is ineffective in many cases. The best treatment is walking, which helps develop additional (collateral) blood vessels, allowing blood flow to bypass the affected arteries. If in some cases symptoms are disabling, several procedures are available to widen the nar-

rowed artery balloon angioplasty, to compress the plaque against the inner arterial walls; surgical endarterectomy, to remove plaque from the walls; or surgery to bypass the blocked artery, using a vein taken from elsewhere in the leg or a synthetic artery.

WHAT ARE THE COMPLICATIONS?

The foot on the affected leg may become cold and numb, with dry skin and limited nail growth. Skin ulcers may develop, even after only slight injury. A blood clot may form in a narrowed artery, cutting off circulation to the lower leg or foot and causing acute pain. In severe cases, which are not too frequent, impaired blood flow can be disabling and may increase the risk of gangrene. Urgent surgery may be required to save the limb in these cases.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Avoiding smoking and adopting a low-fat, low-cholesterol diet and a regular exercise regimen can help prevent peripheral vascular disease. Diabetes and hypertension should be treated at an early stage. If peripheral vascular disease does occur, weight reduction can reduce the burden on the legs and daily exercise can assist the body in its efforts to increase the size and distribution of the smaller blood vessels in the area (known as collateral circulation). Scrupulous foot care should include keeping the feet warm and dry and avoiding constricting garters and tight shoes or socks as well as prompt professional treatment for calluses, corns, ulcers, or foot injuries.

See Chapter 17.

PREMATURE BEATS-ATRIAL AND VENTRICULAR

WHAT IS IT?

The heart's beating rhythm is controlled by a natural pacemaker in an area called the sinoatrial node, located toward the top of the heart. It sends out electrical stimuli in rhythmic waves that normally follow prescribed pathways from the atria (the upper chambers) to the ventricles (the lower chambers), producing sequential and coordinated contractions. If erratic electrical stimuli originate elsewhere in the heart muscle, the normal rhythm is disturbed and becomes irregular. The result is called extra or premature beats. The stimulus for such premature beats may arise in the atria, in the ventricles, or (less commonly) in the AV node—the area that separates the upper and lower parts of the heart. Although the heart chamber where the stimulus arises beats prematurely, the following beat usually occurs after a “compensatory” pause and is generally a stronger contraction. Nevertheless, the following beat arises in a normal fashion.

WHO GETS IT?

The occasional occurrence of premature beats is quite common and often is noted in people with a completely normal heart. Atrial or ventricular premature beats may be triggered by the use of tobacco, alcohol, caffeine, and certain drugs or can be provoked by factors such as anxiety, which causes excess release of adrenalinelike substances. Ventricular premature beats (VPBs) are also frequently seen in those who have heart disease. Even in these patients, however, an occasional atrial premature beat (APB) or ventricular premature beat is not of great significance. Premature beats may be noticed in subjects with rheumatic or atherosclerotic heart disease, mitral prolapse, myocarditis, cardiomyopathy, and heart failure and during attacks of angina. VPBs occur to some extent in more than 90 percent of individuals who have a heart attack.

WHAT ARE THE SYMPTOMS?

Premature beats often may not even be noticed, or may be experienced as a sensation of a skipped or extra heart-beat, palpitation, or heart flutter. If these occur in runs of more than five to ten, there maybe some light-headedness or a feeling of weakness.

HOW IS IT DIAGNOSED?

Premature beats are identifiable by listening to the heart with a stethoscope or taking the pulse at the wrist. Premature beats usually are easily recognizable on an electrocardiogram. Once the premature beats have been identified, further diagnostic procedures are usually not necessary. If the beats are very frequent, arise from different parts of the heart, or cause symptoms, further studies may be necessary to determine whether they are harbingers of a more serious rhythm disturbance or whether they indicate underlying heart disease. These tests may include an exercise stress test, nuclear imaging studies of the heart, Helter monitoring, and an echocardiogram.

HOW IS IT TREATED?

Occasional premature beats that cause no symptoms in a healthy person are of no concern and need not be treated. Eliminating the use of caffeine or nicotine and reducing alcohol intake (if appropriate) may control both APBs and VPBs. If premature beats occur as part of heart failure or following a heart attack, correcting or treating the basic problem may eliminate the extra beats. *Medications should be used only if symptoms are annoying or runs of extra beats occur.* More harm than good may often be done by treating people with premature beats. Reassurances that the “extra beats” are not life-threatening are an important element of treatment.

WHAT ARE THE COMPLICATIONS?

Premature beats may be the forerunners of more severe heart arrhythmias, such as ventricular tachycardia or fibrillation, especially following a heart attack. Usually they are not.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Excessive use of caffeine should be avoided. Smoking cessation and limiting the intake of alcohol can help reduce the occurrence of this type of heart rhythm abnormality.

See Chapter 16.

PULMONARY EDEMA

WHAT IS IT?

Pulmonary edema is a condition that is usually secondary to heart disease, most commonly to heart failure. When the left side of the heart is not pumping effectively, pressure builds up in the heart. Blood in the pulmonary veins (the pathway from the lungs to the heart) gets backed up. Accumulation of excess fluid raises pressure in the pulmonary veins and eventually in the lung tissue. As a result of the backup and increased pressure, fluid passes out of the blood vessels into the little sacs of the lungs (alveoli) that are the normal sites of oxygen and carbon dioxide exchange. As fluid builds up, the lung tissue becomes waterlogged; this condition is called pulmonary edema. Acute pulmonary edema is a potentially life-threatening event.

WHO GETS- IT?

Pulmonary edema is a severe symptom of heart failure and may have such diverse causes as heart attack, heart valve disorders, cardiomyopathy, cardiac arrhythmias, and severe hypertension. It may be the first sign of a heart problem that has gone undiagnosed and untreated for an extended period. It also may occur in people who suffer from “mountain sickness” at very high altitudes.

WHAT ARE THE SYMPTOMS?

Shortness of breath develops and worsens over the course of minutes to several hours. Sometimes it is so acute that the individual gasps for breath and has a sense of suffocation. It may be accompanied by a cough, which is at first dry but eventually produces blood-tinged sputum. Sometimes wheezing occurs. A severe attack also can produce pale skin, sweating, anxiety, and low blood pressure. Symptoms may develop slowly or rapidly, presenting an acute crisis. Pulmonary edema may begin with an acute attack at night, when blood pools in the lungs as the individual lies in bed. Those with chronic pulmonary edema will experience fatigue and shortness of breath, especially after exertion.

HOW IS IT DIAGNOSED?

The symptoms listed above and characteristic signs, such as the sound of rales (a sound like the wrinkling of paper) heard in the chest with a stethoscope, indicate

pulmonary edema. A physical examination, blood tests (particularly to evaluate gases in the blood), and a chest X-ray may confirm the diagnosis.

HOW IS IT TREATED?

Acute pulmonary edema is an emergency that usually requires hospitalization and oxygen support. If an acute attack occurs, emergency medical personnel should be called immediately. Until help arrives, the individual should sit upright. If nitroglycerin tablets are available, one should be placed under the tongue to dilate blood vessels and help distribute blood away from the lungs. Once the patient is stabilized, various medications will be given, such as a diuretic to help drain excess fluid from the lungs, digitalis to improve heart function, and morphine to slow and deepen breathing. Sometimes a phlebotomy—the removal of a certain volume of blood from the body—may be necessary. Additional therapy will depend upon the underlying cause of the heart failure. Chronic pulmonary edema is a form of chronic heart failure and is treated accordingly.

WHAT ARE THE COMPLICATIONS?

If not treated promptly, acute pulmonary edema can be fatal. Effective treatment usually enables restoration of heart function. With proper therapy, individuals who have survived an episode of acute pulmonary edema can lead a reasonably normal life, although they may have to restrict some activity.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Proper treatment of cardiac problems can help prevent pulmonary edema. Patients with heart failure must strictly adhere to a low-salt diet. A significant amount of sodium ingested over a short period can precipitate pulmonary edema.

See Chapters 14 and 27.

PULMONARY HYPERTENSION

WHAT IS IT?

Pulmonary hypertension is a condition in which the pressure in the vessels that carry blood from the heart to the lungs (the pulmonary blood vessels) is abnormally high. Primary pulmonary hypertension is a rare disorder of unknown cause in which the small and medium pulmonary arteries become narrowed and the pressure elevated. Secondary pulmonary hypertension is a more common disorder that occurs as a result of some other lung or heart disease. Both tend to be chronic conditions, although the primary condition is more serious.

WHO GETS IT?

Primary pulmonary hypertension is very uncommon but is three times more common in women than in men, and the average age at diagnosis is 35. Secondary pulmonary hypertension may be caused by almost any chronic lung disorder, but its association with chronic bronchitis and emphysema is especially common. It is also associated with certain types of congenital heart disease in which there is increased flow to the lungs, as well as with scleroderma, a disorder characterized by excessive buildup of fibrous connective tissue, and some neuromuscular diseases that affect the respiratory muscles.

WHAT ARE THE SYMPTOMS?

Pulmonary hypertension may result in shortness of breath, chest pain, and occasional dizziness upon exertion. In addition, there also may be wheezing, coughing, and swollen ankles because of water retention. In secondary pulmonary hypertension, the symptoms may be primarily those of the underlying condition.

HOW IS IT DIAGNOSED?

Often pulmonary hypertension will be suspected on the basis of the patient's report of symptoms and the findings of characteristic heart or breathing sounds during a complete physical examination. Tests to confirm the diagnosis may include a chest X-ray, electrocardiogram, echocardiogram, and tests to monitor the level of oxygen in the blood.

HOW IS IT TREATED?

Primary pulmonary hypertension is largely untreatable medically, although some patients may be helped by drugs that dilate the blood vessels. The effect of such drugs must first be evaluated during cardiac catheterization, because in some patients they may cause serious problems. In secondary pulmonary hypertension, treatment of the underlying condition (such as heart failure) may help alleviate the lung problem. If it is the result of congenital heart disease, surgery may be advised. Medications such as diuretics to relieve fluid retention or digitalis to improve heart muscle contraction may be prescribed, as well as a period of bed rest, perhaps with supplemental oxygen. If all other treatments fail, a lung or heart/lung transplant may be recommended.

WHAT ARE THE COMPLICATIONS?

Primary pulmonary hypertension can be fatal within two to five years after the initial diagnosis, although many people survive for years with the condition and without any specific treatment. Secondary pulmonary hypertension usually can be treated, but it also can have grave complications. The outcome for the patient depends on the condition causing it. If the condition is chronic heart failure or severe emphysema, the prognosis is not good. If pulmonary hypertension causes the right ventricle of the heart to enlarge, the condition is then called cor pulmonale.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Primary pulmonary hypertension cannot be prevented. Prompt treatment of conditions that maybe associated with secondary pulmonary hypertension may prevent or minimize it. Since chronic lung disease—most commonly caused by smoking—is a major cause of secondary pulmonary hypertension, stopping smoking is an important preventive measure. Prompt treatment for infection and regular treatment for any heart or lung disorder or scleroderma are also important. If secondary pulmonary hypertension becomes chronic, prophylactic antibiotics and annual influenza immunization may be recommended to help protect against respiratory infections.

See Chapter 25.

PULMONIC VALVE DISEASE

WHAT IS IT?

The pulmonic valve controls the flow of blood from the right ventricle (lower chamber) of the heart into the pulmonary artery, through which it travels to the lungs for oxygenation. If the valve is stenosed (narrowed), its opening is abnormally small and the heart must work harder to overcome the resulting resistance in order to pump a sufficient quantity of blood. In severe cases, it cannot accomplish this and an insufficient amount of blood moves out of the ventricle with each heartbeat. On the other hand, a pulmonary valve that does not close properly is called regurgitant (incompetent or insufficient), because with each beat, some of the blood that should be pumped from the heart into the pulmonary artery regurgitates, or leaks backward, into the ventricle. In either case, the heart must work harder to pump adequate amounts of blood. The right ventricle may compensate for this either by enlargement (dilation) or an increase in muscle thickness (hypertrophy).

WHO GETS IT?

Pulmonic valve disease is extremely rare and is almost invariably congenital. In severe cases, it can cause a life-threatening emergency in infants. Rarely, the condition may go unrecognized until adulthood.

WHAT ARE THE SYMPTOMS?

In newborns, pulmonic valve stenosis often occurs in conjunction with other heart abnormalities, and symptoms may arise from the combination of anomalies. The main signs and symptoms are shortness of breath and cyanosis (bluish skin), indicating that the baby's blood is not being sufficiently oxygenated. In older children and adults, the only symptoms may be pale skin, shortness of breath on exertion, and easy fatigability. Frequently there will be no symptoms.

HOW IS IT DIAGNOSED?

Some signs of pulmonic valve disease, such as a heart murmur, are detectable during a physical examination. A chest X-ray, an electrocardiogram, and an echocardiogram will usually confirm the diagnosis. Cardiac catheterization is necessary if surgery is planned to correct the valve abnormality.

HOW IS IT TREATED?

Mild pulmonic valve disorders may not require treatment. However, severe stenosis in a newborn requires immediate surgery to establish more normal blood flow. In older children and adults who develop symptoms that impair quality of life, surgery to replace the defective valve may be warranted.

WHAT ARE THE COMPLICATIONS?

As in other types of valve disease, pulmonary valve deformities increase the risk of infective endocarditis, an infection on the valve surface. In severe pulmonic valve disease, right-sided congestive heart failure may develop.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Congenital heart disease cannot be avoided, but fortunately it is rare. Inpatients with pulmonary valve disease, prophylactic use of antibiotics before dental extractions and surgery can help prevent the development of infective endocarditis.

See Chapters 13 and 20.

RHEUMATIC HEART DISEASE

WHAT IS IT?

The term rheumatic heart disease does not refer to a single disorder, but rather to the various types of acute and chronic heart disorders that may occur as a result of rheumatic fever. Every part of the heart, including the pericardium (the outer covering) and the endocardium (the inner lining), may be damaged by inflammation caused by rheumatic fever. However, the most common form of rheumatic heart disease relates to the heart valves, particularly the mitral valve. If the heart has been involved in an attack of acute rheumatic fever, it may take several years for valve damage to develop.

WHO GETS IT?

Rheumatic fever is no longer common in the United States. When it does occur, it usually affects children between the ages of 5 and 15, following a sore throat caused by streptococcal bacteria (strep throat). If the sore throat is not treated promptly with antibiotics, the infection may affect other parts of the body, including the heart.

WHAT ARE THE SYMPTOMS?

Symptoms vary depending upon the type of heart damage caused by the rheumatic fever. In milder cases, there are usually no symptoms. In cases of advanced valve abnormalities, breathlessness, palpitations, heart arrhythmias, fever, swollen feet, dizziness, and chest pain may be experienced.

HOW IS IT DIAGNOSED?

In some cases, a heart murmur (which can be heard with a stethoscope) develops during or after a bout of rheumatic fever, signaling the development of minor to major heart valve changes. In others, more severe problems become immediately apparent. In the majority of cases, symptoms of heart disease develop slowly after an initial attack of rheumatic fever and do not appear until young adulthood or middle age. Diagnosis of heart involvement usually requires a chest X-ray, electrocardiogram, or echocardiogram.

HOW IS IT TREATED?

If heart damage from rheumatic fever is identified in childhood or young adulthood, prophylactic antibiotics may be recommended daily until about the age of 25–30 to prevent recurrence of rheumatic fever and to help avoid the development of endocarditis. Further therapy depends on the type of heart damage present. Medications may be prescribed to help slow a rapid heartbeat, while anticoagulant drugs may be recommended to help prevent the development of blood clots. In advanced cases, surgery may be needed to replace the damaged heart valves.

WHAT ARE THE COMPLICATIONS?

The most common long-term heart problems involve an abnormal flow of blood in the heart because of damaged heart valves. Generally the mitral or aortic valve is involved and does not open fully (stenosis) or close properly (insufficiency). Individuals with rheumatic heart disease also have a greater risk of developing bacterial endocarditis.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Rheumatic fever and subsequent heart disease have become fairly rare in the United States since the development of antibiotics. Any child with a persistent sore throat should have a throat culture to check for strep. Penicillin or another antibiotic will usually prevent the development of rheumatic fever from such an infection. About 60 percent of those afflicted with rheumatic fever develop some degree of subsequent heart disease. Individuals who have had rheumatic fever should receive prophylactic antibiotics before any medical or dental surgery to help prevent infection and subsequent bacterial endocarditis.

See Chapters 2, 13, and 20.

SHOCK

WHAT IS IT

Shock occurs when blood pressure falls to a severely low level (about 50 to 60 mm Hg for the upper reading, or systolic pressure) for a period of time, causing the flow of blood to the body to become inadequate. Because the flow of oxygenated blood to vital tissues and organs is impaired, they may cease to function adequately. If this lasts for a short period of time, the effects will be transient. If shock becomes prolonged, it will result in permanent impairment of certain organ systems and can ultimately lead to death. Major types of shock are cardiogenic (from a cardiac source such as a heart attack), hypovolemic (after severe loss of blood or fluids), *anaphylactic* (from an allergic reaction), and *septic* (as a result of overwhelming infection).

WHO GETS IT?

Shock occurs in a wide variety of circumstances and affects various individuals, as defined by the type of shock. Cardiogenic shock occurs when the heart fails to pump adequately; it may result from a heart attack, a severe, sustained arrhythmia, cardiomyopathy, or pulmonary embolism. Hypovolemic shock is caused by acute blood or fluid loss, which might result from external bleeding because of severe injury or internal bleeding from a peptic ulcer, ruptured ectopic pregnancy, or other disorder, or it may arise from fluid loss caused by prolonged severe diarrhea or vomiting, heat exhaustion, or severe burns. Anaphylactic shock is the result of an intense allergic reaction that causes blood vessels to dilate dramatically, leading to a relative shortage of blood volume. Septic shock occurs in the course of a severe infection and is also associated with profound blood vessel dilation.

WHAT ARE THE SYMPTOMS?

Fatigue, faintness, nausea, and a feeling of panic are often the major symptoms of shock. Other symptoms may include chills, cold hands and feet, pale and clammy skin, palpitations, sweating, and thirst. Breathing is rapid but shallow, and the pulse is rapid but weak. In septic or anaphylactic shock there may also be fever. If the condition is not treated promptly, lethargy, drowsiness, confusion, and loss of consciousness may occur.

HOW IS IT DIAGNOSED?

Low blood pressure alone does not constitute shock. Someone can faint from low blood pressure and not be in shock! If the skin is warm and dry and few symptoms other than low blood pressure are present, the patient has hypotension but not shock. Shock is diagnosed based on the overt symptoms and appearance of the individual, the presence of one of the diseases that may cause it, a very low blood pressure, and a weak pulse

that is usually greater than 100 beats per minute. While laboratory tests and other diagnostic measures such as an electrocardiogram can aid in reaching a precise diagnosis, such measures should only be considered after appropriate emergency treatment has been employed.

HOW IS IT TREATED?

Emergency treatment is essential for survival. The patient is placed flat on his or her back with legs raised to provide maximum blood flow to the heart and brain. An exception might be the patient who is breathing rapidly with gurgling sounds in the chest. This suggests congestion in the lungs secondary to cardiogenic shock. In this case the patient should be kept in a semi-sitting position. If blood or fluid loss is believed to be the cause of the shock, intravenous fluids should be given, or if there has been blood loss, blood transfusions. Drugs may be injected to strengthen the heartbeat, slow a runaway heartbeat, and raise blood pressure. Oxygen support may be provided. After blood pressure has stabilized at a level sufficient to relieve symptoms and return more normal function to organs such as the kidneys, therapy for the underlying cause of the shock can be instituted.

WHAT ARE THE COMPLICATIONS?

If not treated promptly, shock maybe fatal. If the brain and kidneys are deprived of adequate blood and oxygen, severe damage may occur. Kidney failure may result if shock is not reversed within a few hours.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Immediate first aid should be provided until emergency medical personnel arrive. This includes keeping the victim warm and lying down with legs slightly raised (about a foot)—except in certain circumstances (see above). As noted, if breathing worsens in this position, heart failure may be part of the shock syndrome and the person should be kept in a sitting position. If breathing or heartbeat stops, cardiopulmonary resuscitation should be undertaken. In the case of trauma, if bleeding is observed, the flow should be stemmed by applying direct pressure to the site of the bleeding or using a tourniquet above the bleeding site (if possible). In the case of an anaphylactic shock, the immediate injection of an antihistamine or adrenaline may be lifesaving. Many people with a history of severe allergic reactions carry these medications (in injectable form) with them. Most important, get the person to a hospital emergency room at once.

See Chapter 27.

STROKE AND TIA

WHAT IS IT?

A stroke, sometimes called a cerebrovascular accident, is a form of cardiovascular disease affecting the blood

supply to the brain. The most common types (cerebral thrombosis and cerebral embolism) are caused by blood clots that interfere with the delivery of oxygen to various parts of the brain. Cerebral thrombosis is similar to a heart attack, in that a blood clot forms in an artery (already narrowed by atherosclerosis) in the brain or one in the neck leading to the brain. In a transient ischemic attack (TIA or ministroke), the interruption of blood flow, and thus the occurrence of symptoms, is only temporary or intermittent. In a cerebral embolism, a blood clot formed elsewhere (usually in the heart) travels in the bloodstream to block blood flow in or to the brain. Less common, but usually more serious, are hemorrhagic strokes (cerebral hemorrhage and subarachnoid hemorrhage), which occur when a blood vessel in the brain bursts, interrupting the normal flow of oxygen to the brain.

WHO GETS IT?

Cerebral thrombosis most commonly occurs in older people with long-established atherosclerosis and untreated high blood pressure. Cerebral emboli are more frequent in those who have irregular heart rhythms such as atrial fibrillation, heart attacks, or heart failure. Hemorrhagic strokes are more common in those with uncontrolled high blood pressure. They may also arise as a result of a head injury or a burst congenital aneurysm (weakened arterial wall in the brain).

WHAT ARE THE SYMPTOMS?

The symptoms of a stroke may include sudden weakness or numbness of the face, arm, and leg on one side of the body; difficulty in speaking or understanding others; dimness or impaired vision in one eye; unexplained dizziness or unsteadiness; and sudden falls. TIA symptoms are similar but milder, such as temporary weakness, visual disturbances, or loss of feeling on one side of the body, or other stroke symptoms that last only a few minutes. TIAs themselves are a significant warning sign of a future stroke.

HOW IS IT DIAGNOSED?

A complete history and physical and neurological examination form the basis for diagnosing most strokes and TIAs. Observation of the patient's symptoms can reveal much about the stroke's location to a neurologist. ACT scan of the brain will usually pinpoint the area of the stroke. An evaluation of brain blood flow, such as by Doppler ultrasound scan of the carotid arteries (arteries in the neck that bring oxygenated blood to the brain), may be performed to help determine prognosis and design optimal therapy.

HOW IS IT TREATED?

Blood clots cause at least 70 percent of all strokes; if such strokes are treated within a few hours of onset with anticoagulants (blood-thinning drugs), some stroke damage may be avoided. Such drugs can even be haz-

ardous, however, with hemorrhagic strokes. After the acute phase, treatment focuses on rehabilitation and therapy to prevent a stroke recurrence. The therapy includes treatment or modification of risk factors (high blood pressure, exposure to cigarette smoke, high cholesterol, etc.). It may involve antiplatelet drugs, including aspirin, and anticoagulants. For some patients, an endarterectomy—surgery to remove atherosclerotic plaque in a neck artery—may be advised.

WHAT ARE THE COMPLICATIONS?

When areas of the brain are deprived of oxygen, nerve cells in the area die in a matter of minutes. As a result, near and distant body parts controlled by these brain centers can no longer function properly. Depending on the extent of the stroke, impairment of movement, speech, memory, vision, behavior, or other functions may occur. In some cases the impairment may be permanent; in others, recovery may range from partial to complete. Hemorrhagic strokes generally are more life-threatening, because extensive bleeding can cause pressure within the brain and damage to areas around the bleeding site.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Stroke prevention is aimed at controlling or eliminating key risk factors, such as high blood pressure, heart disease, diabetes, high serum cholesterol levels, cigarette smoking, obesity, and physical inactivity.

See Chapters 2 and 18.

SYNCOPE (FAINTING)

WHAT IS IT?

Syncope, or fainting, is simply a loss of consciousness.

WHO GETS IT?

Fainting is usually the result of decreased blood flow to the brain. The most common cause is vasovagal syncope, a nerve response in which the heartbeat slows and blood vessels in the abdomen and lower limbs dilate. The blood then pools in these areas, and less is available to the brain. This type of fainting occurs following an emotional upset, such as viewing an accident or having blood drawn. A similar mechanism operates when someone faints after a few drinks or a large dinner, or after standing still for a long time on a hot day. Fainting also may be caused by a very slow heartbeat (below 40–45 beats a minute) or a very rapid one (more than 140–150 beats a minute). It can occur as a result of heart failure, a heart attack, or severe stenosis (narrowing) of the aortic valve. Some antihypertensive medications may briefly lower blood pressure too much when the patient stands up (orthostatic hypotension). Other causes of syncope in-

clude severe low blood sugar, heat exhaustion, hyperventilation (rapid breathing), severe anemia, and stroke.

WHAT ARE THE SYMPTOMS?

The onset of fainting may be heralded by a feeling of weakness, unsteadiness, light-headedness, or, in some cases, palpitations or a feeling of emptiness in the chest. Numbness, tingling, loss of movement on one side of the body, blurred vision, confusion, or difficulty speaking may infrequently follow the fainting episode.

HOW IS IT DIAGNOSED?

A medical history and physical examination most often will reveal a simple explanation for syncope—e.g., a vagal episode, orthostatic hypotension after extended bed rest and getting up suddenly, or an emotional upset that causes hyperventilation. In some cases, a complete diagnostic evaluation to identify the underlying cause of the syncope may require blood tests, an electrocardiogram, Helter monitoring, or other, more complicated studies.

HOW IS IT TREATED?

Someone who faints should be kept in a reclining position with feet slightly raised in as cool an environment as possible. An upright position should not be resumed until the person regains consciousness, and then only slowly. The choice of further treatment is totally dependent upon the cause of the syncope. Occasional benign syncope usually warrants none. Frequently a simple fainting episode is overtreated. Drug-related fainting may require a change in medication. Sometimes lifestyle modification, such as dietary measures for low blood sugar (more frequent meals), may be indicated. In unusual cases, medical or surgical therapy may be needed to control or correct the underlying disorder. For example, severe changes in heart rates or rhythms may warrant drug therapy or implantation of a pacemaker; an aortic valve stenosis may warrant surgery to replace the valve.

WHAT ARE THE COMPLICATIONS?

A single fainting episode in an otherwise healthy person may not present a problem, assuming there was no fall resulting in injury. However, some medical evaluation should be undertaken to exclude a specific medical cause of syncope. If the fainting was caused by a serious cardiac disorder or stroke, specific treatment is obviously necessary.

HOW CAN IT BE PREVENTED OR MINIMIZED?

People who have a tendency to faint when blood is drawn, or in emotional situations, should sit down or lie down immediately if they begin to feel light-headed. If dizziness occurs after taking medication, the doctor should be notified. Recurring rapid or slow heart rate or heart valve disease requires specific medical treatment. Older men who might have a tendency to feel dizzy

or actually faint after urinating at night should probably sit rather than stand. (Fainting can occur after the sudden emptying of the bladder.)

See Chapter 2.

TACHYCARDIA

WHAT IS IT?

The heart normally beats at a rate of about 60 to 80 beats per minute at rest. A rate faster than 100 beats a minute in an adult is called a tachycardia. Most people experience transient rapid heartbeats, called sinus tachycardia, as a normal response to excitement, anxiety, stress, or exercise. If tachycardias occur at rest or without a logical cause, however, they are considered abnormal. The two main types of tachycardias are: abnormal *supraventricular tachycardias* (which originate in the upper chambers of the heart, the atria) and *ventricular tachycardias* (which originate in the lower chambers of the heart, the ventricles). The most common forms of tachycardia are *paroxysmal supraventricular tachycardia*, which generally has a rate of 140 to 200 beats per minute, develops spontaneously, and stops and starts suddenly, but may recur; *atrial flutter*, in which the atria beat at 240 to 300 beats per minute, although the actual pulse rate is much slower, because not all of these impulses are translated into contractions of the ventricles; *ventricular tachycardia*, a very serious arrhythmia initiated in the ventricles, in which the heart rate is usually between 150 and 250; and *atrial fibrillation* (*see* separate entry).

WHO GETS IT?

Sinus tachycardias are most likely to occur in those who are easily excitable, suffer anxiety, or drink a lot of caffeine-containing beverages. They may also be seen in people with thyroid disease with fevers or with certain drugs (especially asthma or allergy medications and those containing adrenaline). The occurrence of tachycardia under any of these circumstances does not necessarily imply underlying heart disease. More severe types of tachycardia tend to occur in those who have underlying heart disease. They may be caused by an electrical disturbance within the heart without an anatomic deformity, or by congenital defects, coronary artery disease, chronic disease of the heart valves, or chronic lung disease. Tachycardias may also occur in the course of a heart attack.

WHAT ARE THE SYMPTOMS?

The main symptom is awareness of a rapid heartbeat, commonly called “palpitations.” Depending on the cause and extent of the tachycardia, other symptoms may include shortness of breath, dizziness, actual syncope (fainting), chest pain, and severe anxiety.

HOW IS IT DIAGNOSED?

The type of tachycardia usually can be diagnosed by measuring the pulse and taking an electrocardiogram. In unusual instances, more complex electrophysiologic evaluation may be necessary.

HOW IS IT TREATED?

Medical treatment depends on the cause and type of the tachycardia. Sinus tachycardias usually do not require treatment other than therapy for the underlying cause, if any. A supraventricular paroxysmal tachycardia may respond to certain simple maneuvers. This may involve holding one's breath for a minute, bathing the face in cold water, or massaging the carotid artery in the neck. In other cases, medication may be prescribed to slow the heartbeat on a continual basis. If tachycardia is severe, or arises from the ventricle, immediate injectable medication or electric shock (electroconversion) maybe required to stimulate the heart to return to a normal rate. In rare severe and resistant cases of ventricular tachycardias, a defibrillation device (AICD) something like a pacemaker may be implanted surgically to help maintain a normal heart rhythm. In elderly people or those with underlying heart disease, it is important to stop even the less severe types of tachycardias within a few hours, if at all possible, because a prolonged rapid rate may result in decreased heart function.

WHAT ARE THE COMPLICATIONS?

In persistent cases of a ventricular tachycardia, the rapid rate continues, the heart cannot pump blood effectively, and ventricular fibrillation, in which normal heart muscle contraction fails and the heart quivers, may occur. If fibrillation is not stopped with an electrical shock and normal rhythm restored within a few minutes, it will be fatal.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Complete medical evaluation is mandatory in order to identify any serious arrhythmias. Most cases of palpitations will be benign. In certain instances, medication must be taken regularly. Environmental factors such as caffeine and smoking should be eliminated.

See Chapters 16 and 26.

TRICUSPID VALVE DISEASE

WHAT IS IT?

The tricuspid valve is one of four valves that control the flow and direction of blood in and out of the heart. Blood enters the right atrium (upper heart chamber) and passes through the tricuspid valve into the right ventricle (lower pumping chamber), from where it is pumped out through the pulmonary artery to the lungs. If the valve

is narrowed (stenosed), it becomes difficult for a sufficient amount of blood to move through the right heart chambers with each beat. If the valve does not close properly, some blood flowing into the ventricle leaks back into the atrium with each beat. This condition is known as regurgitation or insufficiency. In both cases, the heart must work harder to pump an adequate amount of blood. In stenosis, the right atrium becomes enlarged, while the right ventricle does not fill completely and remains small. In regurgitation, both right chambers enlarge substantially.

WHO GETS IT?

Tricuspid valve disorders, which *are rare*, often occur in conjunction with other heart valve problems, particularly with mitral valve disorders. Tricuspid valve stenosis is usually caused by rheumatic heart disease, although it is occasionally due to a congenital condition. Tricuspid valve regurgitation is often secondary to high pressure within the heart's chambers, usually caused by pulmonary hypertension. Rheumatic heart disease can also cause it. Isolated tricuspid regurgitation may be the result of endocarditis, particularly in intravenous drug abusers.

WHAT ARE THE SYMPTOMS?

Tricuspid regurgitation and stenosis may be present for years without symptoms. When symptoms do occur, they may include an uncomfortable fluttering sensation in the neck or chest because of heart rhythm irregularities. Both conditions can produce the symptoms of right-sided heart failure, including discomfort in the upper abdomen because of an enlarged liver, fatigue, and swelling.

HOW IS IT DIAGNOSED?

Signs of tricuspid valve disease, such as a heart murmur and an abnormal pulse in the jugular vein in the neck, may be detectable during a physical examination. A chest X-ray, an electrocardiogram, and an echocardiogram are helpful in reaching the diagnosis. Cardiac catheterization may be performed if surgery is being considered.

HOW IS IT TREATED?

Tricuspid valve disorders usually require no treatment in and of themselves, although related heart valve problems may require specific treatment. If atrial fibrillation is present, it can be treated with oral antiarrhythmic drugs. In the case of severe stenosis or regurgitation, surgery to replace or repair the defective valve may be recommended.

WHAT ARE THE COMPLICATIONS?

Individuals with tricuspid valve disease are at risk for heart failure and for atrial fibrillation (which in turn increases the risk of blood clot formation). As in other types of valve disease, tricuspid disorders also increase the risk of infective endocarditis.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Limiting the risk of rheumatic fever, particularly by prompt treatment of strep throat, is the major preventive measure for tricuspid disease. Prophylactic use of antibiotics before dental extractions and surgery can help prevent the development of infective endocarditis.

See Chapter 13.

VENOUS DISEASE

WHAT IS IT?

Veins are vessels that carry blood from arms, legs, the head, etc., back to the right side of the heart. The most common venous diseases are phlebitis and varicose veins. Phlebitis is an inflammation of a vein that may be caused by an injury, infection, or chemical irritation. Often a clot is formed in the inflamed vein; this condition is called thrombophlebitis. There are two types of phlebitis. Superficial phlebitis, which is not serious, is the inflammation of a surface vein that may occur after an intravenous infusion or a bruise. The other type is deep thrombophlebitis, which refers to a clot forming in an inflamed vein below the skin surface. In deep thrombophlebitis, there may be some danger of a clot being thrown off to another part of the body. Repeated phlebitis can lead to poor venous drainage or venous insufficiency with chronic swelling. Varicose or swollen and twisted veins may result from poor function of the internal valves that normally help to push blood upward from the legs to the heart.

WHO GETS IT?

Women develop these venous diseases more often than men, and a hereditary predisposition may underlie them. Obesity, hypertension, pregnancy, a family history with genetic predisposition, and the use of garters around the thighs may contribute to the development of varicose veins. Those at greater risk of phlebitis include smokers, people who have varicose veins or other evidence of venous insufficiency, those who are bedridden for long periods of time, especially after surgery or a fracture, and those having intravenous therapy. People who are overweight or who have heart disease are also susceptible. The risk of thrombus (clot) formation is greater in those who are aged, inactive, have heart disease, or use oral contraceptives or after long airplane or car trips.

WHAT ARE THE SYMPTOMS?

Swollen veins just under the skin surface are likely to be varicose. The legs also may be achy, painful, warm to the touch, and easily fatigued. In phlebitis, the leg may be swollen, tender, and red and may feel achy and heavy. As phlebitis progresses, the skin may become painful and bluish. The only symptom of superficial phlebitis may be slight tenderness.

HOW IS IT DIAGNOSED?

Venous diseases usually are easily diagnosed by simple observation. The major symptom of superficial phlebitis may be a hard, clothesline-like area in the armor leg. In some cases of suspected thrombophlebitis, it may be necessary to inject a dye into the vein to visualize its interior surface (venography).

HOW IS IT TREATED?

Therapy for an acute, deep phlebitis may include bed rest and elevation of the leg, a nonsteroidal anti-inflammatory drug to reduce inflammation, warm soaks, bed rest, and an anticoagulant to help prevent blood clots. If an embolism is identified, a fibrinolytic drug to dissolve the clot may be prescribed; in rare cases, surgical removal of the clot may be needed. Superficial phlebitis may merely require warm wet soaks and aspirin. The use of specially fitted elastic stockings will help ease the symptoms of varicosities (varicose veins). If varicosities cause skin ulcers or pain or are particularly unsightly, injections of drugs to obliterate the vein or surgery to remove it may be advised.

WHAT ARE THE COMPLICATIONS?

Severe varicosities may predispose the patient to skin ulcers and thrombophlebitis. The major complication of phlebitis is dislodging of a blood clot, which may travel to the lungs (pulmonary embolism). This can have serious consequences and may be fatal.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Avoidance of overweight and of garter usage can help prevent varicose veins. If they occur, the use of support hose and the avoidance of long periods of standing can help alleviate symptoms. Elastic stockings and, in some, medical therapy can help prevent phlebitis in those at risk after surgery or during periods of mandatory bed rest. The chances of repeated attacks of phlebitis may be decreased by avoiding inactivity. Physical therapy with an active exercise plan after surgery can decrease risk of phlebitis. For certain individuals, anticoagulants may be necessary.

See Chapter 17.

WOLFF-PARKINSON-WHITE SYNDROME

WHAT IS IT?

Wolff-Parkinson-White (WPW) syndrome represents a congenital abnormality involving the heart's electrical function. Although many people with this abnormality exhibit no symptoms, the syndrome can result in episodes of rapid heartbeat called paroxysmal supraventricular tachycardia (PSVT). In contrast to a normal 60

to 80 beats per minute, the rate rises, generally to 180 to 240 per minute. WPW is caused by abnormal conduction of electrical signals in the heart. Electrical signals arrive at the ventricles prematurely, because they travel through a shortcut (bypass tract) between the atria and the ventricles. This condition makes the heart susceptible to rhythm abnormalities.

WHO GETS IT?

Although the congenital anomaly that causes Wolff-Parkinson-White syndrome is present at birth and symptoms may arise in infancy or childhood, tachycardias are more likely to develop later in life. WPW may be associated with other congenital malformations, but generally it occurs alone.

WHAT ARE THE SYMPTOMS?

People who have WPW may have no symptoms at all or may experience palpitations and, possibly, chest pain, shortness of breath, and fainting. Fainting indicates that the heart is beating so rapidly that it is unable to pump adequate amounts of blood to the brain. The palpitations may be described as skips, thumps, butterflies, fluttering, or racing of the heart.

HOW IS IT DIAGNOSED?

In the presence of WPW, an electrocardiogram shows characteristic changes indicating the existence of an abnormal pathway from the atria to the ventricles. If attacks of tachycardia are frequent, special studies of the electrical activity of the heart (electrophysiologic tests) may be done to determine the location of the shortcut pathway and its response to different drugs.

HOW IS IT TREATED?

In the absence of tachycardias, often no treatment is necessary. When it is, it is best individualized and de-

pendent upon the extent and frequency of the tachycardia and based upon electrophysiologic studies. Sometimes, simple avoidance of stress and dietary sources of caffeine maybe helpful in preventing episodes of tachycardia. In other cases, the physician will prescribe medication to stabilize heart rhythm. This medication may be taken only at the time of an attack or, more likely, on a continuing basis to prevent the development of supraventricular tachycardia. If an attack cannot be controlled by medication, treatment with a brief electrical shock may be necessary to restore normal rhythm. If medication is insufficient to control the repeated episodes of rapid rhythm, open-heart surgery may be necessary to eliminate the abnormal pathway. A new technique uses radiofrequency current, delivered via a catheter, to eliminate the abnormal pathway without surgery.

WHAT ARE THE COMPLICATIONS?

The paroxysmal supraventricular tachycardias of WPW can be disconcerting but, by themselves, are not usually life-threatening. In some people, however, the tachycardia may be so rapid that it causes fainting. Certain individuals with this disorder are at greater risk of ventricular tachycardia or fibrillation, a much more serious irregular rhythm that can be fatal.

HOW CAN IT BE PREVENTED OR MINIMIZED?

Beyond taking medication, the patient should learn nonmedical techniques to decrease the risk of tachycardia (such as avoidance of caffeine and of excessive alcohol) and mechanical methods to help slow down the heart or terminate an episode of tachycardia. One such method is straining by closing the nose and mouth and trying to exhale.

See Chapters 16 and 26.