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CARDIOMYOPATHY AND RELATED DISEASES

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Annotation: Cardiomyopathy, any cardiac disease process that results in heart failure due to a decrease in the pumping power of the heart or due to an impairment in the filling of the cardiac chambers. Persons with cardiomyopathy frequently retain excess fluid, resulting in congestion of the lungs, and have symptoms of weakness, fatigue, and shortness of breath. Sometimes they develop a potentially fatal arrhythmia, or abnormal heart rhythm.

Key words:cardiomyopathy, dilated, cobalt, drug, heard, blood, systolic dysfunction.

Most cardiomyopathies are idiopathic, or of unknown cause. Specific cases are usually categorized as dilated, hypertrophic, or restrictive, according to the observed abnormality. Dilated cardiomyopathy, the most common type of the disease, is characterized by an enlarged heart with stretching of the ventricle (lower chamber) and atrium (upper chamber). The left ventricle, which pumps oxygenated blood to the body tissues, shows weakness in contraction (systolic dysfunction) and stiffness in expansion and filling (diastolic dysfunction). These dysfunctions lead to fluid retention and eventually heart failure. Preceding heart attack is known to cause the condition, as is exposure to toxic substances such as alcohol, cobalt, and some anticancer drugs.

In hypertrophic cardiomyopathy, the ventricles are quite small owing to abnormal growth and arrangement of the cardiac muscle fibres. This form of the disease is often hereditary and has been associated with mutations in several different genes, each of which encodes a protein necessary for the formation of sarcomeres, the contractile units of muscle. However, mutations in two genes, *MYBPC3* (myosin-binding protein C, cardiac) and *MYH7* (myosin, heavy chain 7, cardiac muscle, beta), are responsible for roughly 80 percent of familial hypertrophic cardiomyopathy cases. The onset of symptoms in people affected by this form of the disease varies, ranging from infancy to late adulthood. Thickening, or hypertrophy, of the ventricular walls results in an extremely stiff heart, and subsequent impairment of the filling of the ventricles causes pressure to rise in the heart and lungs. The increased pressure in turn leads to shortness of breath and fluid retention. Hypertrophic cardiomyopathies are commonly associated with serious arrhythmias and sudden cardiac death.

Restrictive cardiomyopathy also is characterized by a stiff heart and impaired ventricular filling. In this case the abnormality is caused by the presence of fibrous (scar) tissue introduced by a disease such as amyloidosis. Patients display many of the symptoms of hypertrophic cardiomyopathy.

Treatment of cardiomyopathy is directed first toward identifying the underlying disease (e.g., hypothyroidism or hypertension). Patients are treated as any patient with heart failure; indeed, all patients with heart failure have some sort of cardiomyopathy. If general treatment measures fail, patients with cardiomyopathy can sometimes be helped with heart transplantation. Because some forms of cardiomyopathy are inherited, individuals from families with a history of the disease are encouraged to have physical examinations, electrocardiograms, and echocardiograms done periodically. In the future, genetic screening may make it possible to detect persons at risk for developing cardiomyopathy.

Heart disease, any disorder of the heart. Examples include coronary heart disease, congenital heart disease, and pulmonary heart disease, as well as rheumatic heart disease (*see* rheumatic

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fever), hypertension, inflammation of the heart muscle (myocarditis) or of its inner or outer membrane (endocarditis, pericarditis), and heart valve disease. Abnormalities of the heart's natural pacemaker or of the nerves that conduct its impulses cause arrhythmias. Some connective tissue diseases (notably systemic lupus erythematosus, rheumatoid arthritis, and scleroderma) can affect the heart. Heart failure may result from many of these disorders.

Heart failure, general condition in which the heart muscle does not contract and relax effectively, thereby reducing the performance of the heart as a pump and compromising blood circulation throughout the body.

Heart failure is a major public health concern in countries worldwide. Although reliable data on the prevalence of heart failure is lacking for some parts of the world, it is estimated that globally more than 25 million people are affected by it. There are multiple risk factors for heart failure, including older age (65 years or over), being male, having a family history of the condition, or having certain underlying conditions, particularly myocardial infarction (heart attack), cardiac valve insufficiency (leaking) or stenosis (narrowing), and diabetes. Certain lifestyle factors—such as tobacco smoking, alcohol consumption, physical inactivity, and a diet that predisposes individuals to high cholesterol and high blood pressure—also raise the risk of developing heart failure.

When heart failure occurs, the ability of the heart to contract is decreased (systolic dysfunction), or the heart becomes stiff and does not relax normally (diastolic dysfunction); in some cases both conditions exist together. With less blood ejected from the heart at each beat, the body attempts to compensate for the decreased circulation to peripheral organs. Perhaps the most important response is the retention of salt and water by the kidneys in an attempt to increase intracardiac pressures and improve circulatory volume. As a result of these reflex actions, patients with heart failure usually show signs of congestion, along with weight gain and swelling of the extremities and abdominal organs—a condition known as congestive heart failure. Patients with congestion in the lungs or chest cavity suffer from short-windedness, particularly with exertion or while trying to lie flat. The heart's response to the systemic effects of circulatory failure is to enlarge the chambers (cardiomegaly) and increase the muscle mass (hypertrophy). Treatment of heart failure is complex and multifaceted. Of prime importance is treatment of the specific underlying disease (such as hypertension, valvular heart disease, or coronary heart disease). Prescribed medications are usually aimed at blocking the adverse effects of the various neurologic, hormonal, and inflammatory systems activated by heart failure. These are generally drugs in the class of angiotensin-converting enzyme (ACE) inhibitors to lower blood pressure and decrease the heart's workload, beta-adrenergic blockers (beta-blockers) to stabilize the heartbeat, aldosterone antagonists to decrease salt retention, and vasodilators to relax the smooth-muscle lining of the veins and arteries. Diuretics are prescribed to remove excess fluid. Digoxin and digitoxin are commonly prescribed to increase the strength of heart contraction. (These latter drugs evolved from digitalis, which was introduced in the 18th century as one of the first effective remedies for congestive heart failure, known at the time as "dropsy.") Patients are also advised to limit their intake of salt and fluids, avoid alcohol and nicotine, optimize their body weight, and engage inaerobic exercise as much as possible. Much can be done to prevent and treat heart failure, but ultimately the prognosis depends on the underlying disease causing the difficulty as well as the severity of the condition at the time of presentation.

Arrhythmia, variation from the normal rate or regularity of the heartbeat, usually resulting from irregularities within the conduction system of the heart. Arrhythmias occur in both normal and

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diseased hearts and have no medical significance in and of themselves, although they may endanger heart function when coupled with other cardiac abnormalities.

Types of arrhythmias include tachycardia, which is a regular acceleration of the heart rate; bradycardia, a regular slowing of the heart rate; and premature atrial or ventricular beats, which are extra contractions within otherwise normal heart rhythm. While occasional irregularities are normal, prolonged or chronic arrhythmias associated with some forms of heart disease may reduce cardiac output, lowering blood pressure and affecting the perfusion of vital organs with blood, and can precipitate cardiac arrest and heart failure. Severe arrhythmias can trigger atrial fibrillation or ventricular fibrillation, in which the heart beats ineffectively at many times its normal rate.

Arrhythmias reflect the failure of the sinoatrial node, the normal cardiac pacemaker, to maintain a regular heartbeat, usually because of defects in the various pathways by which electrical impulses are carried to different areas of the heart. Anatomical defects or disease can slow down or speed up the propagation of electrical impulses, causing them to arrive out of the normal rhythm, or can turn the impulses back on their path, short-circuiting the pacemaker. Many arrhythmias can be corrected through physical methods, such as artificial pacemakers, defibrillators, and radiofrequency ablation (the application of radiofrequency energy to the area of the heart that is causing the arrhythmia), or by drugs such as beta-blockers and calcium channel blockers.

Heart attack, death of a section of the myocardium, the muscle of the heart, caused by an interruption of blood flow to the area. A heart attack results from obstruction of the coronary arteries. The most common cause is a blood clot (thrombus) that lodges in an area of a coronary artery thickened with cholesterol-containing plaque due to atherosclerosis.

Factors that contribute to the risk of atherosclerosis include high blood pressure (hypertension), diabetes mellitus, increased blood levels of low-density lipoprotein (LDL) cholesterol, smoking, and a family history of the disease. Particularly vulnerable to atherosclerosis are middle-aged men and individuals with the hereditary disease hypercholesterolemia. In the early 21st century, heart attacks were becoming increasingly common among women under age 55. Although the reason for this increase was unclear, increased rates of diabetes, hypertension, and obesity in women likely played a role.

Most heart attacks occur in the morning, a phenomenon that researchers have linked to circadian rhythm. In the morning hours, increasing circadian-driven secretion of certain hormones, particularly epinephrine, norepinephrine, and cortisol, triggers subsequent increases in oxygen demand and blood pressure. These factors in turn increase circulatory activity. In addition, the production of endothelial progenitor cells, which appear to play a crucial role in repairing the lining of blood vessels, also follows a circadian pattern, with fewer cells present in the circulation in the early morning. Decreased levels of these cells results in depressed endothelial maintenance, which scientists suspect may facilitate the onset of a heart attack upon waking. Typically, a person experiencing a heart attack has severe chest pain, described as crushing, squeezing, or heavy, that is unremitting for 30 to 60 minutes and sometimes is experienced for longer periods. It often radiates to the arms, neck, and back. The pain is similar to that of angina pectoris, but it is of longer duration. Other common symptoms include shortness of breath; sweating; nausea; rapid heartbeat, often complicated by one or more arrhythmias (irregular heartbeats); and reduced blood pressure. The intensity of the symptoms depends on the size of the area of muscle affected by the heart attack. A

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small percentage of individuals do not experience pain; in these cases heart attack may be diagnosed from a routine electrocardiogram (ECG).

The focus of treatment is to limit the size of the area of tissue lost from lack of blood (infarct) and to prevent and treat complications, such as arrhythmia. Thus, the sooner the heart rate can be monitored by an ECG and the more promptly the arrhythmia is reversed by defibrillation with either antiarrhythmic drugs or electrical shock, the greater the chance of survival. Pain is treated with analgesics such as morphine, and rest and sedation are required. Other drugs that may be administered include beta-adrenergic-blocking drugs (beta-blockers) to relax the heart muscle, anticoagulants (e.g., heparin) to prevent clotting, fibrinolytic drugs to dissolve existing clots, and nitroglycerin to improve blood flow to the heart. Coronary thrombolysis therapy is widely used; it involves the administration of drugs such as streptokinase or tissue plasminogen activator (tPA) to prevent further blood clots from forming. Angioplasty or coronary artery bypass surgery are additional measures for patients requiring further treatment.

The prognosis for patients who survive a heart attack depends largely on the degree of injury to the heart and the associated decline in heart function. Reduced heart function following an attack is caused by the formation of scar tissue that interferes with the normal electrical activity of the heart, leading to reduced heart muscle contractility, progressive weakening of the heart, and heart failure. To prevent such outcomes, scientists are investigating stem cell-based regenerative therapies, which aim to replace scar tissue with new heart muscle cells.

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