TEXTBOOK OF
Clinical cardiology

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Preface

Disorders of the heart and circulation, particularly cardiac ischemia (coronary artery disease), constitute the greatest cause of death in the United States and affect many of the patients who visit a physician's office or enter a hospital. Therefore it is important for medical students, members of the house staff, family practitioners, and internists, in addition to cardiologists, to understand the basic principles of diagnosing and treating the numerous cardiovascular disorders.

The basic examination of a patient with a suspected cardiovascular disorder still consists of taking a careful history, a physical examination, a routine 12-lead electrocardiogram, and a chest roentgenogram. However, this may not be adequate; newer, more advanced procedures may be needed. Remarkable strides have been made in the past few years in cardiac diagnosis and treatment. For example, in electrocardiography, we now have not only the conventional 12-lead electrocardiogram, but also exercise stress testing electrocardiography, continuous ambulatory (Holter) electrocardiographic monitoring, vectorcardiography, and His bundle recordings. In examination with x-ray techniques, we now have not only the conventional chest roentgenogram or fluoroscopic examination of the heart and lungs, but also cardiac catheterization and angiocardiography, coronary arteriography, and left ventriculography, as well as radioisotope examination of the heart and great arteries (radionuclide angiography). Echocardiography is now a very important *non*invasive diagnostic method for evaluation of pericardial disorders, left ventricular function, abnormalities of the heart valves, congenital malformations of the heart, and other heart conditions.

It is therefore most important for the *non*cardiologist to know the indications, limitations, and contraindications of these various test procedures, as well as to know their basic normal and the more common abnormal patterns or findings. This information appears in Part One of this book. Chapter 1 describes symptoms referable to the cardiovascular system to remind the reader not to be overwhelmed by the imposing array of cardiovascular tests that are now available. The reader is also cautioned to remember that the patient is at the end of the stethoscope and that the goal of a physical examination or of any other diagnostic procedure is the treatment of the patient. Chapters on physical examination with normal and abnormal cardiac findings follow, succeeded by chapters describing specialized diagnostic tests, including external pulse recording, phonocardiography, apexcardiography, measurement of systolic time intervals, electrocardiography including exercise stress testing, continuous ambulatory (Holter) monitoring, and His bundle recording, vectorcardiography, chest roentgenography, cardiac catheterization including selective angiocardiography, coronary arteriography, and left ventriculography, radionuclide angiocardiography, measurement of venous pressure, use of the Swan-Ganz double-balloon catheter, and laboratory blood tests.

Part Two is a systematic description of the

many disorders that can occur not only in the heart but also in the entire circulation. I prefer the term "syndromes" rather than "diseases" to describe these conditions, because "syndrome" implies a group of patients with similar functional (biochemical and pathophysiologic) and structural (pathologic, that is, physical, anatomic, and microscopic) changes whereas "disease" implies a definite, rigid entity of patients with similar anatomic and pathologic disorders. This distinction is important because if a physician treats a patient for a "disease" the treatment will tend to be rigid or stereotyped, whereas in treating a patient for a "syndrome," the physician will try to correct the abnormal biochemical, pathophysiologic, or structural changes that are present.

For example, consider a patient with congestive heart failure resulting from chronic rheumatic mitral stenosis. You can give the patient digoxin or a similar digitalis preparation to increase the force of cardiac contractions and the cardiac output. You can also institute a salt-poor or low-sodium diet to eliminate the excessive salt intake that has contributed to the congestive heart failure. Or you can give the patient a diuretic to eliminate the excessive sodium ions from the body; here again, there are many choices for the diuretic, depending on the potassium balance of the body and on other factors. Or you can use a combination of these three approaches. Finally, surgical treatment of the mitral valve may be indicated if the stenosis is severe and the mitral valve area, determined by cardiac catheterization, is abnormally small.

For these reasons, in Part Two I have stressed the pathophysiologic and structural changes that are present in the various cardiovascular disorders and have shown how these changes explain the clinical findings and diagnostic test abnormalities and, furthermore, how knowledge of these pathophysiologic and structural changes allows the doctor to recommend or to change therapy in a logical way. Biochemical changes are also important, particularly when diuretic therapy is prescribed.

The following cardiac syndromes are described in Part Two: syncope, sudden death and cardiac arrest, cardiogenic shock, congestive heart failure, coronary atherosclerosis and myocardial ischemia including stable and unstable angina, variant (Prinzmetal's) angina, and acute myocardial infarction, valvular cardiovascular syndromes, cor pulmonale (acute and chronic), emotional and behavioral factors in cardiovascular disorders, hypertensive cardiovascular syndromes, congestive cardiomyopathies and myocarditis, rheumatic fever, infectious endocarditis, acute pericarditis, cardiac tamponade, constrictive pericarditis and restrictive cardiomyopathy, and disorders of cardiac rhythm (bradyarrhythmias and tachyarrhythmias).

In describing each syndrome, I have used the following general plan: etiologic factors, biochemical changes, pathophysiologic changes, pathologic (anatomic, structural, and microscopic) changes, clinical findings, phonocardiograms or pulse tracings, electrocardiograms, echocardiograms, cardiac catheterization, laboratory tests, diagnosis, course and prognosis, and treatment. The above topics are discussed for any syndrome when they are relevant.

Many disorders that affect the heart and circulation cannot be included in the above list of cardiac syndromes because the pathophysiology, pathology, clinical findings, and diagnostic test findings are too varied. Therefore I have divided these disorders into two parts: Part Three describes congenital cardiac malformations. Part Four describes special conditions affecting the cardiovascular system, including cardiovascular syphilis, cardiac tumors, lesions of the thoracic aorta and superior and inferior vena cava, noncardiac disorders, drugs and other agents affecting the cardiovascular system, noncardiac surgery in cardiac patients, nonpenetrating cardiac trauma, pregnancy in cardiac patients, and environmental factors (air travel, high altitude, employment, and athletics) affecting the cardiovascular system.

Part Five describes cardiac pacing, defibrillation, and cardioversion.

Part Six describes cardiovascular pharmacology. After a general discussion of the electrophysiologic action of antiarrhythmic drugs, the various cardiovascular drugs are described in terms of their pharmacologic properties,

namely, antiarrhythmic drugs, anticoagulant and similar drugs, antihypertensive drugs, autonomic drugs, cardiotonic (positive inotropic) drugs, diuretics, and vasodilator drugs.

When a book has multiple authors, it is inevitable that there will be some duplication. However, this can be advantageous because in cardiology, as in all other medical specialties, there are differences of opinion.

I am indebted to my contributors, whose names and academic titles are listed elsewhere. The following persons also were helpful in other ways: Dr. Bernard Fish reviewed the chapters on congenital cardiac malformations, Dr. Ary L. Goldberger, my son, made helpful suggestions about many parts of the book, and Dr. Robert H. Lapin reviewed the chapter on infectious endocarditis.

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Emanuel Goldberger

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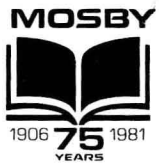

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PART ONE

Methods of examining a patient

1 Symptoms referable to the cardiovascular system

Taking a careful history is an important part of a cardiologic examination. You should question the patient carefully and systematically for symptoms related to all the organ systems, even though the patient may be concerned specifically about the heart. Try to get answers about symptoms as specific as possible. Use terms that the patient can understand, such as dizziness (not vertigo), difficulty in breathing (not dyspnea), and difficulty in swallowing (not dysphagia).

Severe symptoms, such as palpitation, difficulty in breathing, and pain in the chest, may be present in a patient who does not have a cardiac disorder. On the other hand, a serious cardiac disorder may be present without symptoms. In addition, symptoms caused by a heart ailment may occur at areas distant from the heart or chest; for example, atypical anginal pain may occur in the jaw or teeth, with minimal pain in the chest.

If a patient complains of pain in the chest, ask him or her to point to the painful area with the tip of the index finger or, if the pain is not localized, to place the hand over the painful area. If the complaint is difficulty in breathing on exertion, ask exactly how much exertion, walking, stair climbing, and so on, are needed to bring on the symptoms.

The following is a brief outline of information you should elicit.

WEIGHT CHANGES

Either gain in weight or loss of weight can be a cardiac symptom. A sudden gain in weight of 2 to 3 or more pounds in a few days may be a sign of fluid retention from congestive heart failure. A progressive loss of weight in a patient who is taking digitalis may be a sign of digitalis toxicity (digitalis cachexia). The digitalis produces anorexia, and as the patient loses weight, the effects of the digitalis become more pronounced.

FATIGUE

When a patient with a cardiovascular disorder complains of fatigue, this may be a sign of congestive heart failure. However, it may be a side effect of excessive diuretic therapy (with excessive lowering of the serum sodium and potassium concentrations). In hypertensive patients, it may also be attributable to excessive diuretic therapy, or to hypotension, or it may be a side effect of one of the many antihypertensive medications, such as methyldopa (Aldomet) and propranolol (Inderal).

FEVER AND CHILLS

See Chapter 2.

HEADACHE

Patients with systemic hypertension often complain of a dull, nagging, occipital or vertex headache. However, the headache is not related to the height of the blood pressure. The headache is probably caused by dilatation and distention of a branch or branches of the external carotid artery. The headache of hypertensive encephalopathy is caused by increased intracranial pressure and cerebral edema, and it may also be caused by petechial hemorrhages and small cerebral infarcts.

TINNITUS

Hypertensive patients also often complain of a continuous or intermittent buzzing or hissing noise in the ear or ears (tinnitus). It is probably caused by atherosclerosis of the internal carotid artery so that the patient hears the blood flowing through the artery in the carotid canal,

which lies just behind the eardrum. Tinnitus also occurs in patients who are not hypertensive.

VERTIGO, DIZZINESS, AND SYNCOPE

See Chapter 17.

INSOMNIA

Insomnia is usually not a symptom of a cardiovascular disorder. However, elderly patients with heart failure may complain of insomnia caused by cerebral hypoxia. If such a patient is given a hypnotic, symptoms may become more severe and the patient may become confused. Treatment consists in stopping the hypnotic and treating the heart failure.

CARDIOVASCULAR NOISES HEARD BY PATIENT

A grade 6 murmur (see Chapter 4) may be audible not only to the patient, but also to a member of the family. It may be caused by a ventricular septal defect, severe aortic regurgitation with retroversion of one of the aortic cusps, or an AV fistula. A patient may also hear the beating heart or even arterial sounds when the head rests on a pillow.

PAIN OR PRESSURE IN THE CHEST

When you question a patient about chest pain, remember that many patients with angina pectoris describe their symptoms as "pressure," not "pain."

Pain or pressure in the chest is a common cardiac symptom. However, a patient may have severe chest pain or pressure without a cardiac disorder. In addition, chest pain may be caused by both cardiac and noncardiac conditions; for example, a patient may have angina pectoris and also a severe osteoarthritis of the thoracic spine, or a left subdeltoid bursitis, which contributes to the pain, or a patient may have angina and a hiatus hernia of the stomach.

Conversely, many serious cardiac disorders, such as complete AV block, severe valvular lesions, severe myocardial ischemia, and even acute myocardial infarction, especially in the elderly, may be present without chest pain or pressure. One of the reasons for this is that patients differ in their reaction to pain. A very sensitive patient may become anxious because of awareness of (benign) premature beats. Another patient may disregard the symptoms of an acute myocardial infarction as "mild indigestion." Elderly patients may find it difficult to describe symptoms.

When a patient describes pain or pressure in the chest, it is important to obtain information about the site of the pain, type of the pain, spread of the pain to other regions, duration of the pain, and factors that may have precipitated the pain.

There are generally two types of patients with chest pain or pressure—patients with chronic pain or pressure, and patients with acute pain, or particularly acute pressure.

Patients with chronic pain or pressure may have angina pectoris and conditions such as anxiety, neurocirculatory asthenia, straight-back syndrome, mitral valve prolapse syndrome, costochondritis, left subdeltoid bursitis, thoracic-outlet syndrome (cervical rib, scalenus anticus syndrome), esophageal spasm (cardiospasm), hiatus hernia of the stomach, peptic ulcer, gallbladder disorders, and so on.

The pain of angina pectoris characteristically is dull (pressing, burning, squeezing, and so on, but *not* sticking. It is located substernally, *not* over the region of the left breast. (Many patients believe the heart is located beneath the left breast.) The anginal pressure is precipitated by cold, exercise, exertion, stress, eating, and so on. It lasts usually not more than 15 minutes. It may radiate down the inner side of the left arm, even to the fourth and fifth fingers. It may also radiate to the neck, jaw, teeth, throat, and other regions. Remember that typical anginal pain can occur at rest (Prinzmetal's variant angina, see Chapter 23).

Some patients may say that if chest pressure or pain occurs during walking they may be able to relieve the symptoms by continuing to walk (walk-through phenomenon). This is also a sign of angina pectoris.

The pain in noncardiac conditions simulating angina is usually sticking and is usually located over the left breast region. It may be aggravated by movement of the trunk and may last for hours. There may also be a feeling of "tight-

ness" in the chest caused by spasm of the muscles of the rib cage. The feeling of "tightness" may also last for hours.

When you ask a patient with angina pectoris to point to the location of the chest pain, he or she will often place a palm over the midsubsternal area or will make a fist and place it over this area, whereas a patient with chest pain caused by a noncardiac condition often will point to the area of pain with the index finger. The reason for these different gestures is that the anginal pain is diffuse, whereas noncardiac chest pain is often localized.

Detailed discussions of disorders that can simulate angina are found in Chapter 23.

Patients in whom pressure in the chest occurs acutely may have minimal symptoms, or the pressure may be severe and unremitting. Immediate hospitalization is indicated if you suspect that one of the following conditions is present: acute myocardial infarction, angina with symptoms of having become more severe or prolonged, acute pericarditis, pulmonary embolism, dissecting aneurysm of the aorta, perforation and rupture of the esophagus, and so on.

The symptoms of acute myocardial infarction are similar to those of angina but are usually more intense, last 20 minutes or longer, and are not relieved by nitroglycerin. The patient may also break out into a cold sweat, develop an ashen or cyanotic hue, or vomit. (Remember that occasionally a patient with acute myocardial infarction may have no symptoms or minimal symptoms that resemble angina pectoris.)

The differentiation of acute myocardial infarction from these other conditions is described in Chapter 24.

Chest pain in adolescents and young adults

Bernard Fish

Chest pain is a common complaint in adolescents and young adults. Usually the heart is not its origin; however there are a few types of chest discomfort that may be related to the heart. The first is typical ischemic pain related to stress or exercise. This is rare; however, it is of concern because it may be associated with severe aortic stenosis, IHSS, aortic regurgitation, congenital coronary artery abnormalities, acute or chronic myocarditis, or cardiomyopathy. Angina-like chest pain can also occur in patients with pulmonary hypertension, including primary pulmonary hypertension and Eisenmenger's syndrome, or pulmonary hypertension occurring in patients with severe mitral stenosis. Similar chest pain can occur in the tetralogy of Fallot, atrial septal defect, Ebstein's anomaly, and pulmonic stenosis.

Mitral valve prolapse may be associated with chest pain of an ischemic or nonspecific nature. Straightening of the thoracic spine can also cause nonspecific chest pain and murmurs (straight back syndrome). Acute pericarditis can also cause severe chest pain. This is typically worsened when the patient breathes or lies supine. Patients prefer to sit up, leaning forward in bed.

Tachyarrhythmias produce a feeling of fullness in the base of the neck and upper chest, sometimes with breathlessness and dizziness.

Chest pain in adolescents, when pronounced, can usually be traced to noncardiac causes such as costochondritis, pectoralis muscle spasm, trauma, herpes zoster, bronchitis, pneumonia, asthma, viral pleuritis or pleurodynia, tendinitis of the shoulder, brachial neuritis or cervical disk disease, pneumothorax, esophagitis, or hiatus hernia.

PALPITATION

Palpitation or awareness of the heart beating is also described by patients as skipping of heartbeats, heart flutter, or a jumping sensation in the chest. It is usually caused by premature atrial or ventricular beats or by paroxysmal tachycardia. However, a nervous patient with a normal cardiac rhythm may complain of palpitation, particularly if the heart rate reaches or exceeds 90 per minute. Palpitation may also occur in patients with AV block when the ventricular rate is irregular, and in pacemaker patients when pacemaker beats and spontaneous beats are both present.

DYSPNEA

Dyspnea, or breathlessness, is the uncomfortable awareness of difficult breathing. It may be caused by either a pulmonary or a cardiac

disorder. The explanation for dyspnea in an obstructive lung disorder is obvious. However, it is more difficult to explain the mechanism of dyspnea in a restrictive lung disorder (such as pulmonary fibrosis) or in patients with pulmonary congestion from left-sided congestive heart failure. In these patients, there is apparently increased lung stiffness, so that stretch receptors in the lungs are stimulated when the patient inhales. Dyspnea also occurs in patients with pulmonary hypertension, for reasons that are not clear.

It may be very difficult to differentiate cardiac from pulmonary dyspnea. One clinical way is to give the patient a therapeutic trial with diuretics. A patient with cardiac dyspnea will usually improve. If pulmonary dyspnea is present, this will not occur.

Pathophysiology. Dyspnea can occur in several ways:

1. Pulmonary congestion occurs as a result of left-sided congestive heart failure. The distended pulmonary vessels encroach on the alveolar spaces and decrease the breathing capacity of the lungs. In addition, pulmonary edema or pleural effusion may be present. This further decreases the effective lung volume.
2. Pulmonary congestion also interferes with the mechanical aspects of breathing because congested lungs are relatively inelastic and rigid. This hardness interferes with their expansibility and retractability and produces shallow ineffective respirations.

When a patient with left-sided congestive heart failure develops right-sided congestive heart failure, the severity of the dyspnea may decrease. The reason is that some of the blood that was formerly stagnant in the lungs is now pooled in the liver and lower extremities; therefore a decrease of dyspnea in a patient with left-sided congestive heart failure is not necessarily a sign of improvement.

Most patients with cardiac dyspnea complain of dyspnea *on exertion*. Some patients may also develop asthmatic wheezing in association with the dyspnea. This is known as *cardiac asthma*, in contrast to the usual bronchial asthma. (The differentiation is discussed in Chapter 21.)

Paroxysmal nocturnal dyspnea is almost always a symptom of acute left-sided congestive heart failure. The patient falls asleep normally but awakes in 1 to 2 hours with severe or extreme shortness of breath (see Chapter 21).

Sighing dyspnea. Sighing dyspnea is not true dyspnea. It occurs as a neurotic symptom in patients who are anxious, but who do not have a heart disorder. The patient takes a deep normal breath, when asked to breathe, and then exhales slowly, uttering a faint sigh.

Dyspnea should not be confused with *hyperpnea*, which is an exaggerated depth of respiration *without* symptoms of respiratory distress. Although hyperpnea can occur in association with dyspnea, it may also be present in nonpulmonary or noncardiac conditions, such as acidosis, severe anemia, or shock.

ORTHOPNEA

Orthopnea means difficulty in breathing, which disappears in an upright position. Thus a patient who develops dyspnea at rest will get relief by sitting or standing.

When orthopnea occurs during sleep, the patient may merely need an extra pillow at night. When orthopnea is pronounced, the patient may need to sleep in a sitting position. At times, the orthopnea is so severe that the patient grasps the side of the bed or a chair, to fix the shoulder girdle and obtain greater respiratory excursions.

Orthopnea occurs not only when there is pulmonary congestion, but also whenever the maximum breathing reserve is decreased. Therefore it can occur in acute or chronic pulmonary disease, mediastinal tumors, and even pronounced obesity, despite a normal heart.

Pathophysiology. The effectiveness of sitting or standing to relieve orthopnea is caused by several factors:

1. When one stands, the vital capacity increases.
2. When one stands, pulmonary congestion, the cardiac output, and therefore the work of the heart decrease. This occurs because a large amount of blood can be pooled in the veins of the lower extremities. This is the reason that some patients with severe orthopnea get relief not only

by sitting in bed, but also by dangling their feet from the side of the bed.

3. When a person lies down, the pulmonary veins drain upward into the left atrium, against gravity. However, in the standing position, the flow is downward. This helps to decrease pulmonary congestion.

Trepopnea is a form of orthopnea where the patient obtains respiratory relief by lying in one particular position, such as in the left lateral position, rather than in the right lateral or supine position, and so on. The variations in position may possibly influence pulmonary congestion.

COUGH

Pathophysiology. Coughing is usually caused by congestion of the bronchi. Since the bronchial veins empty into the pulmonary veins (which return blood to the left side of the heart) and also empty into the systemic veins and superior vena cava, which return blood to the right side of the heart), congestion of the bronchi and coughing may occur with either right-sided or left-sided congestive heart failure. However, coughing is more common with left-sided congestive heart failure.

Occasionally a patient may develop a cough because of mechanical pressure. For example, the left main bronchus may be displaced upward by a large left atrium. An aneurysm of the aorta may press on the bronchi, producing a rasping type of cough. A double aortic arch may compress the trachea and cause coughing.

The coughing may be dry, with little or no expectoration. Sometimes the patient has merely a tickling sensation in the back of the throat, resembling that of a postnasal drip. The cough may be worse at night, after the patient lies down to sleep. Occasionally it is paroxysmal, occurring particularly after exertion.

The sputum may be mucoid, or streaked with blood. Or the sputum may show a brownish discoloration from hemosiderin (a breakdown product of hemoglobin).

Coughing is important because it may be an early symptom of acute left-sided heart failure. This is particularly important when a patient with a paroxysmal tachycardia or acute myocardial infarction develops a cough.

HEMOPTYSIS

Hemoptysis, or coughing up blood, may be caused by a pulmonary or cardiac disorder. Occasionally a patient may expectorate blood-tinged saliva because of a nasopharyngitis, which may be mistaken as hemoptysis.

Hemoptysis may or may not occur in a cardiac patient even if severe pulmonary congestion is present. The bleeding may be slight or may consist of only streaks or pinpoints of blood. If pulmonary edema is present, a pink frothy sputum may appear. Occasionally, massive bleeding occurs, so that a severe acute anemia may result. This severe type of hemoptysis occurs particularly in the following three types of conditions.

Mitral stenosis. The hemoptysis may be precipitated by severe physical exercise, excitement, sexual intercourse, or pregnancy. It occurs because strong right ventricular beats flood the pulmonary circulation and the left atrium with blood that has difficulty in passing into the left ventricle because of an abnormally tight mitral valve opening. However, in such patients, acute left-sided congestive heart failure and acute pulmonary edema usually occur instead of hemoptysis. If such a patient develops right-sided congestive heart failure, the attacks of hemoptysis may cease because this pulmonary congestion is lessened. Dyspnea also decreases for the same reason.

Acute pulmonary embolism and infarction. The hemoptysis appears a few hours to a day after the infarction. However, it does not always occur.

Congenital cardiac malformation in which pulmonary hypertension has developed. See the discussion on Eisenmenger's syndrome in Chapter 41.

Hemoptysis may also occur during acute rheumatic fever, for reasons not understood. Other rarer causes of hemoptysis are rupture of an aneurysm of the aorta into the trachea or a bronchus, or rupture of a pulmonary arteriovenous fistula.

HOARSENESS

Hoarseness caused by paralysis of the left recurrent laryngeal nerve can occur in mitral ste-