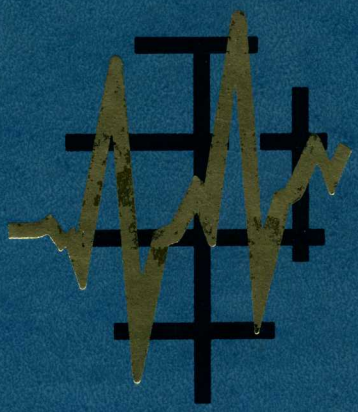
Essentials of CLINICAL CARDIOLOGY



Emanuel Goldberger

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CLINICAL CARDIOLOGY

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To
BLANCHE
with love

Preface

Essentials of Clinical Cardiology is a cardiology text for noncardiologists. It presents an overview of current clinical cardiology.

This book is a revision of my book, *Textbook of Clinical Cardiology,* which was published in 1982. It differs from that book in many ways. It is half the size, and I have eliminated much of the technical material. I have written the entire book myself in order to present a unified point of view throughout the book.

I have rewritten a large part of the book emphasizing clinical features of heart disease. I have added a chapter on geriatric cardiology and an appendix on the use of neck vein pulsations in diagnosing cardiac arrhythmias. Some tests are very expensive, and the physician should be aware of the cost-benefit relationship of such tests.

Part One describes the symptoms and physical signs of cardiovascular disease. It reminds 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 the goal of a physical examination or of any other diagnostic procedure is the treatment of the patient. Part Two describes cardiac syndromes. Part Three describes special conditions affecting the cardiovascular system. Part Four describes cardiac pacing, defibrillation, and cardioversion.

I have not included a discussion of congenital heart disease. My reason for this is that most cases are diagnosed by pediatricians who follow the patients through corrective surgery and into adulthood.

I appreciate the contribution of Dr. Michael V. Cohen, who reviewed the material on Doppler echocardiography; of Valerie Mencher, who took some of the Doppler echocardiograms; and of Alie Cahill, who took some of the electrocardiograms. I am indebted to Dr. Robert H. Lapin for writing the section on treatment of infectious endocarditis. I am grateful to the many contributors to the *Textbook of Clinical Cardiology* for allowing me to use some of the illustrations from their chapters and for using their chapters as a basis for the revision. I want to thank Dr. Jack J. Kleid who supplied most of the echocardiograms for the text.

Finally I am indebted to my son, Dr. Ary L. Goldberger, for his help and excellent suggestions.

Emanuel Goldberger, MD, FACP

Contents

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 sites 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 major symptoms of heart disease are chest pain (angina), dyspnea (exertional and paroxysmal nocturnal), orthopnea, and palpitation. The following is a brief outline of information you should elicit.

WEIGHT CHANGES

Either weight gain or weight loss 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 progres-

sive 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 due to overaggressive diuretic therapy or to hypotension, or it may be a side effect of one of the many antihypertensive medications, such as methyldopa (Aldomet) or propranolol (Inderal).

FEVER AND CHILLS

See Chapter 3.

HEADACHE

Patients with systemic hypertension often complain of a dull, nagging, occipital or vertex headache; however, the headache is not related to the blood pressure. It 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.

VERTIGO, DIZZINESS, AND SYNCOPE

See Chapter 13.

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 atrioventricular (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.

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 16).

Some patients may say that when 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 "tightness" 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 often places a palm over the midsubsternal area or makes a fist and places 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 16.

Patients whose 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, lasting 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. (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 19.

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 sinus rhythm may complain of palpitation, particularly if the heart rate reaches or exceeds 90 beats per minute. Palpitation may also occur in patients with AV block when the ventricular rate is irregular and in pacemaker patients when both pacemaker beats and spontaneous beats are 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.

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 decreased 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 16.

Occasionally, patients with coronary heart disease develop dyspnea instead of chest pressure.

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 16).

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 is difficulty in breathing that disappears when the patient assumes 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 whenever the maximum breathing reserve is decreased. It can accompany acute or chronic pulmonary disease, mediastinal tumors, and even pronounced obesity, despite a normal heart.

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 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 aneursym 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.

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 for 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, 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 three types of conditions: mitral stenosis, acute pulmonary embolism and infarction, and congenital cardiac malformation in which pulmonary hypertension has developed.

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

HOARSENESS

Hoarseness caused by paralysis of the left recurrent laryngeal nerve can occur in mitral stenosis, atrial septal defects, or aneurysm of the aortic arch. It is usually caused by compression of the nerve between the dilated main pulmonary artery (or the left pulmonary artery) and the inferior surface of the arch of the aorta, where the nerve loops around the aorta on its way to the neck. The nerve can also be compressed and injured by pericardial or mediastinal adhesions or by enlarged mediastinal lymph nodes. Rarely, an aneu-

rysm of the arch of the aorta causes hoarseness by compressing the right recurrent laryngeal nerve. Remember that hoarseness can also occur in noncardiac conditions, such as mediastinal tumors and pulmonary tuberculosis.

Improvement in the patient's cardiac condition may cause hoarseness to disappear, but if the nerve has been severely injured, the hoarseness may be permanent.

GASTROINTESTINAL SYMPTOMS

Anorexia, bloating, and nausea may be signs of right-sided congestive heart failure or may be side effects of therapy with digitalis, potassium salts, diuretics, and so on. If the symptoms are caused by digitalis excess, they worsen if the patient's weight decreases (because of loss of appetite) and the digitalis dosage is not decreased. Indigestion and heartburn may be atypical symptoms of angina.

Vomiting may occur in acute right-sided congestive heart failure because of stretching of the capsule of an enlarged liver. Vomiting may also occur in acute myocardial infarction as part of the clinical picture of acute myocardial infarction, or it may be produced by injections of morphine or a similar drug.

Dysphagia, or difficultly in swallowing, caused by a cardiovascular disorder is rarely seen today. It is caused by pressure on the esophagus. It may occur in mitral regurgitation and stenosis when a giant left atrium presses on the esophagus, or in massive pericardial effusion, or in double aortic arch, aneurysm of the aorta, or dissecting aneurysm of the aorta.

Cardiac disorders may be associated with severe abdominal pain. In acute right-sided heart failure, intense right upper abdominal pain, caused by stretching of the distended liver capsule, may simulate gallbladder colic or acute cholecystitis. In acute myocardial infarction, there may be intense epigastric pain with vomiting, which may simulate a ruptured peptic ulcer, gallbladder colic, or acute cholecystitis. In acute rheumatic fever, vague abdominal pain, or right lower quadrant abdominal pain associated with vomiting and leukocytosis, may simulate acute appendicitis.

NOCTURIA

Nocturia, or excessive urination at night, is a common complaint of patients with congestive heart failure. When the nocturia is pronounced, the total urine volume at night may be greater than that passed during the day. Nocturia occurs because the metabolic needs of the body decrease during rest; therefore the cardiac output at night improves, kidney blood flow improves, and more urine is formed.

Nocturia also commonly occurs in elderly men with prostatism and may be a symptom of renal insufficiency.

FUNCTIONAL CLASSIFICATION OF PATIENTS WITH CARDIOVASCULAR DISORDERS

It is often important to describe the general condition of a cardiac patient concisely. The following classification of the New York Heart Association, based on both the patient's symptoms and the physical findings, is helpful:

Functional class	*Definition*
I	*Uncompromised.* Patients with a cardiac disorder without limitation of physical activity. Ordinary physical activity causes no discomfort.
II	*Slightly compromised.* Patients with a cardiac disorder with slight to moderate limitation of physical activity. Ordinary physical activity causes discomfort.
III	*Moderately compromised.* Patients with a cardiac disorder with moderate to marked limitation of physical activity. Less than ordinary physical activity causes discomfort.
IV	*Severly compromised.* Patients with a cardiac disorder who are unable to carry out any physical activity without discomfort.

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