GUIDE TO CLINICAL REASONING

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To my wife for her illuminating presence

PREFACE

Many medical students react to a body of information in continuous expansion by retaining the most salient features of diseases. In their process of learning, they often neglect atypical manifestations. Rather, they commit to memory, and expect to see, so-called textbook patients with a characteristic clinical picture and an uneventful response to therapy. However, patients rarely conform to the ideal pattern conceived by students. Incipient features of diseases are often subtle. Aging, concurrent disorders, and iatrogenic reactions may affect the course of the primary disease or its treatment. Textbook patients do not even exist in the classical textbooks, which fully describe all the facets of diseases. Unfortunately, most students rarely or reluctantly resort to these weighty books, replete with what they consider to be minutiae, unlikely to occur and unworthy of attention. More popular concise, pocket-sized manuals reinforce the students' tendency to cling to simplified versions of potentially complicated problems. As a result of their data acquisition process, students are easily puzzled by findings that depart from the typical picture they have in mind, and they often fail to detect, or to interpret correctly, inconspicuous, though important, diagnostic hints.

This book is designed as an aid to the early detection of clues and pitfalls in diseases, clinical syndromes, and laboratory problems frequently encountered in daily medical practice. Emphasis has not been put on classical, generally well known features, or on clinical "pearls." Instead, elusive diagnostic pointers and misleading manifestations have been stressed in order to sensitize the medical observer to their possible significance. Whenever needed, the altered clinical picture in the elderly patient has been described. Special attention has been given to danger signals requiring hospitalization of the patient and/or

aggressive therapeutic measures, as well as to common side effects of therapy and detrimental drug interactions. Various factors capable of influencing the results of technical examinations have been mentioned to incite the reader to critical judgment in the ordering and evaluation of laboratory tests. Mimicking conditions and features excluding a contemplated diagnosis have also been detailed. A brief checklist sums up the essential questions to be answered in the diagnostic workup of a clinical problem. The bibliography refers to recent reviews and monographs providing more extensive clinical information.

In order not to produce a lengthy volume (obviously a self-defeating result), the author has strived to be succinct and to avoid repetitions. Some typographical devices contribute to brevity. The abbreviation Cfr. introduces additional clinical manifestations or investigational procedures confirming the discussed entities. The sign ⟶ indicates the possible meaning(s) of various features within a specific setting. Considerations of space have not deterred the author from correlating, as much as possible, clinical and laboratory findings with their pathophysiological mechanisms, which are occasionally preceded by the capital letter *M*.

Although this book is aimed primarily at medical students, residents in medicine and general physicians may find it useful in quickly reviewing the diagnostic possibilities of an atypical problem and the pros and cons of a working hypothesis or of a planned treatment. It is the author's hope that this volume, by emphasizing easily overlooked aspects of diseases, will help the reader reach the correct diagnosis as early as possible, without awaiting that mythical "textbook patient."

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CONTENTS

CARDIOVASCULAR DISEASES

CONGESTIVE HEART FAILURE

DEFINITION AND PATHOPHYSIOLOGY

CONGESTIVE HEART FAILURE (CHF): Inability of the heart to maintain an adequate output, leading to diminished blood flow to the tissues, and congestion in the pulmonary and/or the systemic circulation.

Heart failure may be due to reduced contractility (e.g., ischemic myocardial disease), diastolic mechanical inhibition of cardiac performance (e.g., mitral stenosis, pericardial tamponade), and systolic mechanical ventricular overloading with excessive pressure loading (aortic stenosis, essential hypertension) or increased volume loading (mitral or aortic regurgitation). Less effective myocardial contraction results in inadequate ventricular emptying during systole, decrease in stroke volume, and increased residual volume of the ventricle. In *left ventricle failure,* the left ventricular stroke volume becomes smaller than the right ventricular stroke volume. More blood is being pumped into the pulmonary vascular bed by the right ventricle than is removed from this bed by the left ventricle. The result is an increase in pulmonary venous and left atrial pressures, and left ventricular diastolic pressure. When the heart begins to fail, compensatory mechanisms come into play: increased left ventricular stroke volume related to an increased length of resting ventricular muscle fibers (Frank-Starling law), increased heart rate, and sympathetic stimulation. The pulmonary congestion with increased stiffness of the lungs necessitates greater work in breathing and produces dyspnea. When the pulmonary capillary pressure exceeds the oncotic pressure of the plasma, pulmonary edema occurs. The same sequence can affect the failing right

ventricle. The most frequent cause of *right ventricular failure* is left ventricular failure usually associated with some degree of pulmonary hypertension. Accumulation of blood in the right atrium and the great veins results in increased venous pressure, liver engorgement, and edema. Decreased cardiac output, renal vasoconstriction, and increased aldosterone levels result in increased proximal tubular reabsorption of sodium and water. This increases the intravascular volume and also elevates cardiac output by the Frank-Starling mechanism. Arteriolar constriction maintains blood pressure in the face of a reduced cardiac output.

ETIOLOGY

Reduced contractility: Coronary artery disease; myocarditis; cardiomyopathy; acidosis; drugs.

Pressure overload: Hypertension; aortic or pulmonary valve stenosis; idiopathic hypertrophic subaortic stenosis (IHSS); coarctation of the aorta; pulmonary artery hypertension (chronic pulmonary disease, pulmonary emboli, left ventricular failure).

Volume overload: Aortic regurgitation; mitral regurgitation; pulmonary regurgitation; tricuspid regurgitation; left-to-right shunts.

Obstruction to atrial emptying: Mitral stenosis; tricuspid stenosis; atrial myxoma; atrial thrombus.

Reduced diastolic relaxation: Restrictive cardiomyopathy; ischemic heart disease; constrictive pericarditis; pericardial tamponade.

PRECIPITATING AND POTENTIALLY TREATABLE FACTORS

Systemic hypertension; myocardial infarction (may be silent); ectopic cardiac arrhythmias; pulmonary embolism; pulmonary infection; thyrotoxicosis; anemia; renal disease; acute rheumatic fever; infective endocarditis; rupture of chordae tendinae; pregnancy; prostatic obstruction in the elderly male; liver disease; increased work load; emotional stress; excessive salt intake; sodium-containing drugs (antacids); digitalis toxicity; electrolyte disturbances induced by excessive diuretic therapy; surgically correctable disorders (mitral stenosis, constrictive pericarditis).

Iatrogenic factors Excessive IV fluids; amantadine; carbamazepine; doxorubicin; corticosteroids; emetine; estrogens; propranolol; quinidine; sodium-containing antacids; discontinuation of digitalis, antihypertensives.

THE CHARACTERISTIC DIAGNOSTIC PATTERN

• *Left ventricular failure:* Exertional dyspnea; orthopnea; paroxysmal nocturnal dyspnea; S_3 or S_4 gallop; moist rales in the lungs; enlarged left ventricle.

• *Right ventricular failure:* Systemic venous congestion; hepatomegaly; peripheral edema; cardiomegaly; gallop.

• Chest x-ray: Cardiomegaly; pulmonary congestion.

VARIANT, ATYPICAL, AND MISLEADING MANIFESTATIONS

• *Dyspnea:* May be related to low cardiac output, in the absence of pulmonary congestion (e.g., acute pericardial tamponade, primary pulmonary hypertension).

• *Dyspnea on effort:* May not be acknowledged by: sedentary, chronically ill, or bedridden patients; patients who decrease their physical activities or gradually adjust to less physical activity; obtunded patients.

• *Orthopnea:* Increased venous return in recumbency and elevation of the diaphragm. May eventually diminish with time because of: progressive impairment of right ventricle function, development of relative tricuspid regurgitation or pulmonary arteriosclerosis and increased pulmonary arteriolar resistance which tend to "protect" the pulmonary capillaries.

NOTE: Patients may complain of dyspnea when they turn on their left side (*left lateral decubitus dyspnea*).

• *Paroxysmal nocturnal dyspnea:* Increase in blood volume occurring with resorption of the dependent edema accumulated during the day. Physical examination may be normal when the patient is examined some hours after the episode.

• *Nocturnal, dry, hacking cough:* May be present with or without rales; due to pulmonary congestion; clears in the sitting position (cfr., chest x-ray reveals congestion).

• *Bronchial wheezing:* May be caused by bronchial edema ("cardiac asthma"). Wheezing may occur with or without rales. Cardiac asthma may occur only on exertion, paroxysmally at night, or as the earliest manifestation of pulmonary edema. (Cfr., Palpable gallop; x-rays.)

• *Unilateral peripheral edema:* Suggests local venous insufficiency; it may also occur in the patient who spends long periods of time with one side of the body recumbent or one limb dependent. Edema in the bedridden patient may be localized to the presacral area.

• *A presystolic or S_4 gallop:* Indicates reduced compliance of the left

ventricle (ischemic heart disease, systemic hypertension) but does not indicate a failing left ventricle per se.

• *Right upper quadrant pain and tenderness:* May be due to relatively rapid hepatic engorgement with distention of the liver capsule.

• *Jaundice:* May result from pulmonary infarction or centrolobular necrosis of the liver.

• *Protein-losing enteropathy:* May occur in severe systemic venous congestion secondary to tricuspid stenosis or constrictive pericarditis.

• *Nausea, diarrhea, and malabsorption:* Due to splanchnic congestion (or to digitalis).

• *Splenomegaly:* Usually in patients who have hepatomegaly; may be associated with cardiac cirrhosis.

NOTE: Congestive heart failure is a rare cause of cirrhosis.

• *Exophthalmos:* May result from an increase in venous pressure.

• *Fatigue and weakness:* Related to low cardiac output; often seen in conjunction with either left- or right-sided CHF.

• *Weight loss:* Related to low cardiac output; may become so marked that carcinoma of the bowel may be suspected.

• *Associated conditions confusing the clinical picture:* (1) Chronic lung disease and interstitial fibrosis: cardiac enlargement may be difficult to diagnose in patients with pulmonary emphysema; pulmonary congestion may not be evident on a chest x-ray. (2) Congenital heart disease: may be associated with acquired disease and may be misdiagnosed.

Misleading laboratory findings

• *Exudative pleural effusion:* An exudative pleural effusion may be found in CHF that leads to inflammatory changes in the pleura or following intensive diuresis with selective loss of water, in the absence of other causes of exudate.

• *Abnormal liver function tests:* Mild elevation of the levels of SGOT, alkaline phosphatase, bilirubin may result from hepatic congestion.

Misleading features of chest x-ray

• *Pulmonary congestion:* Can persist for 1 to 4 days after the wedge pressure has been restored to normal by therapy. Conversely, the wedge pressure may rise rapidly up to 12 h before the appearance of radiologic signs of pulmonary congestion.

• An enlarged heart may still seem to be *normal* if its size was previously near the lower limits of normal (cfr., cardiac inspection and palpation).

• *"Elevation" of the diaphragm:* May be due to subpleural fluid in basal space (cfr., lateral decubitus chest film).

Congestive heart failure Is not excluded by:

• Absence of peripheral edema: Pitting edema becomes manifest only after retention of about 4 to 5 kg of fluid (cfr., gain weight).

• Absent basilar rales: In interstitial edema, when fluid has not extravasated into the alveoli.

• A normal venous pressure (cfr., chest x-ray: interstitial pulmonary edema often precedes elevation of the systemic venous pressure).

• A normal circulation time: In acute CHF (myocardial infarction). Circulation time is shorter than normal in high output failure.

NOTE: Causes of high output failure: Increased body demands: thyrotoxicosis, anemia, pregnancy, arteriovenous fistulas, beriberi heart disease.

The elderly patient Absence of dyspnea, elevated venous pressure, third heart sound, rales, hepatomegaly, peripheral edema is not unusual.

• *Cheyne-Stokes breathing:* Alternating phases of apnea and hyperventilation; related to a lengthening of the circulation time between the lungs and the respiratory centers of the brain; this delay in turn interferes with the normal feedback mechanism that controls respiration. More likely to occur in the presence of cerebral vascular disease which is common in the elderly.

• *Lethargy, confusion:* May be due to decreased cerebral blood flow.

• *Jugular venous distention:* May be due to compression of the veins by an elongated tortuous aorta. (Cfr., Deep inspiration moves the aorta away from the dilated veins and causes them to collapse.)

• *Calcified aortic stenosis:* Is often overlooked as a cause of CHF in the elderly male.

• *Amyloidosis:* Should be considered in the elderly patient with otherwise unexplained CHF.

INDICATORS OF SEVERE OR LONG-STANDING HEART FAILURE

• Acute pulmonary edema: May be accompanied by hypotension and cardiogenic shock.

• Pitting edema of arms and face; ascites is a late manifestation, more frequent in tricuspid valve disease or constrictive pericarditis; oliguria indicates severe reduction of cardiac output; diminished pulse pressure reflects a reduction in stroke volume; splenomegaly; cachexia; Cheyne-Stokes breathing; S_3 gallop.

• Kerley A lines: Dilated interlobular septa in the upper lungs. These are more frequent in patients with acute left ventricular failure.

NOTE: The ECG is not useful in assessing the presence or degree of heart failure.

CLUES TO EARLY DIAGNOSIS

Early indicators of left ventricle dysfunction

• *Orthopneic cough:* A dry, nonproductive cough occurring at night, relieved by sitting up; may also occur with effort or emotional stress. Due to pulmonary congestion. (Cfr., Dyspnea is usually present; chest x-ray: cardiomegaly.)

• *Pulsus alternans:* Alternating weaker and stronger pulsations in the peripheral arterial system. Indicates poorly functioning left ventricle.

NOTE: Electrical alternans (alternation of the P wave, QRS complex, or T wave) is not a sign of heart failure as opposed to mechanical alternans. Pulsus alternans is not a feature of cardiac tamponade.

• S_3 *gallop:* May appear long before pulmonary rales; a paradoxic splitting of the second heart sound; orthopnea; paroxysmal nocturnal dyspnea.

Early indicators of right ventricular dysfunction

• *Palpable systolic lift* of the sternum reflects right ventricle hypertrophy.

• An S_3 gallop accentuated by inspiration and/or a loud pulmonic second sound indicate *pulmonary hypertension.*

• *Hepatojugular reflux:* Pressing on the liver produces increased venous return to the chest with distention of the neck veins.

• A rise (rather than the normal fall) in jugular venous pressure (JVP) with inspiration (*positive Kussmaul's sign*) may be seen even in the presence of normal JVP.

NOTE: Edema is a late sign of CHF.

Roentgenographic clues (upright chest film)

• Distention of the superior pulmonary veins (apical redistribution) indicates pulmonary venous hypertension. Probable postcapillary wedge pressure: 15 to 18 mmHg. May occur without auscultatory abnormalities.

• Interstitial pulmonary edema (septal edema, Kerley lines, perivascular edema, or subpleural edema) may appear before orthopnea and may be present with a normal peripheral venous pressure. Probable wedge pressure: 19 to 25 mmHg.