UNDERSTANDING CARDIOLOGY

Davies · Nelson

CARDIOLOGY

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FOREWORD

The authors have compiled an excellent manuscript on pertinent aspects of cardiovascular disease. The title, *Understanding Cardiology*, is one that is most appropriate. After reading this book one will, indeed, understand a great deal of important fundamentals of cardiovascular disease which are covered in a concise fashion. In fact, Drs. Davies and Nelson are to be commended for their ability to condense medical knowledge without sacrificing the essential characteristics of a particular disease. The authors' purpose has been admirably accomplished through liberal use of figures, diagrams, and photographs which aid greatly in the comprehension of the material presented.

I am sure you will all enjoy *Understanding Cardiology* as I have.

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PREFACE

The learning of any subject is made much easier if the teacher's aim is to provide understanding as well as to impart knowledge. While this is a statement of the obvious, the principle is often lost sight of in the increasingly complex educational process. Our own teaching activities over a number of years have made us aware of the lack of a suitable text on cardiology to which to refer students, and accordingly one of us provided such a text for local use. Its acceptance provided the stimulus for rewriting and expanding it into its present form.

The content is addressed primarily to medical students entering clinical apprenticeship, and the book will succeed in its purpose if it provides for them. However, our hope is that its usefulness will extend to others, including nurses, technicians, house-staff, cardiac fellows and perhaps physicians in practice.

Certain omissions are deliberate. For example, we have chosen not to elaborate on the subject of hypertension. The omission is not intended to decry either the importance or interest of the subject, but rather to acknowledge the growing contributions of nephrology and endocrinology to its understanding. We have also chosen to omit detailed considerations of therapy; since these are amply covered elsewhere. Some might argue that a consideration of digitalis is of more relevance than that of supravalvar aortic stenosis; however, the understanding of the latter is a hemodynamic exercise in itself, whereas the subject of digitalis therapy, after two hundred years, remains ridden with doubt, dogma, and personal bias.

Cardiac evaluation is logical and is based on sound, yet simple, principles. Throughout the text emphasis is placed on bedside observation and physical diagnosis, for these methods remain the cornerstone of

good practice. The good cardiologist is still the one who has the following attributes:

1. Attentiveness and accuracy in the observation of physical signs.
2. Ability to synthesize information gained from different sources.
3. Ability to assess the validity or, perhaps more importantly, the invalidity of laboratory data, and to reject at times those that are not in keeping with the *clinical* picture.
4. Knowledge of the limitations and the toxic properties of drugs.
5. Understanding of the timeliness of surgical intervention and the chances of its success.
6. Understanding of the importance of psychological factors in the patient with heart disease.

All of these qualities are necessary. Most can be gained only with experience, but the experience must be founded on basic understanding and knowledge of the ground rules. These foundations are what we have sought to impart.

Our debt to our teachers, as well as to our colleagues and students through the years, requires no emphasis. We hope that in passing on a number of their ideas, albeit without specific acknowledgment, we do justice to the spirit, as well as the letter, of the dialogue that has gone between us.

March 1978

H. D.
W. P. N.

CONTENTS

1

UNDERSTANDING SYMPTOMS

By definition, symptoms are subjective and thus are not amenable to rigorous analysis. The main symptoms of heart disease, when they exist, are: dyspnea, chest pain, fatigue, palpitation, swelling, fainting. We will discuss each of these symptoms in order.

DYSPNEA

The symptom of dyspnea has a component of unpleasantness. Dyspnea is not the same as hyperventilation, hyperpnea, or tachypnea. Sometimes there may be considerable hyperpnea, e g, during skiing or a game of tennis, but the sensation is usually pleasant and is not dyspnea. Dyspnea is a response that is inappropriate. This symptom of heart disease is the most common, and the underlying mechanisms are complex and unclear at times.

Nevertheless, certain associations emerge that are of great help in the practice of clinical cardiology.

Left Ventricular Failure

Failure of the left heart is characterized by elevation of the left atrial, pulmonary venous, and pulmonary capillary pressures. The failure may occur at rest in more severe cases, or only on effort in milder ones, with a likelihood that the patient would experience *dyspnea on exertion* (DOE). Normal mean left atrial pressure does not exceed 10 to 12 mm Hg (the zero-level reference is at the midthorax with the patient supine). In left heart failure it rises to higher levels, sometimes to 30 mm or more mean pressure at rest.

As the left atrial pressure rises, the lungs become congested and a sequence of events occurs that is characteristic. First, at a mean left atrial pressure of 20 mm or so, dyspnea appears. This development is probably due to a stiffening of the lungs (loss of compliance). At 30 mm the hydrostatic pressure within the pulmonary capillaries begins to exceed the oncotic pressure of the plasma proteins, and fluid begins to transude into the extravascular space, i e, the interstitium of the lung. Now dyspnea is marked and *orthopnea* appears; the patient is more comfortable sitting up. However, a most important point to emphasize is that the lungs are silent on auscultation; there are no rales.* The x-ray shows hilar congestion (see page 387 for an illustration). Next, fluid enters the alveoli and the dyspnea becomes intense; fine rales are heard first at the bases and then at higher levels. The patient develops a dry cough, and then one that is productive of pink frothy sputum. This stage characterizes overt *pulmonary edema*, and the x-ray shows evidence of this (see Figure 14.18, page 387). This sequence of events is followed, no matter what the cause of the rising left atrial and pulmonary capillary pressure, whether it be left ventricular failure (for instance, due to aortic stenosis, hypertension, coronary disease, or cardiomyopathy) or mitral stenosis.

The symptom of dyspnea in patients with disease of the left side of the heart can be used as a guide to the degree of elevation of left atrial pressure, and the symptom must be so interpreted in the absence of other obvious causes, such as an acute or chronic pulmonary disease.

The second situation in which dyspnea may be marked in cardiovascular disease is in *primary [idiopathic] pulmonary hypertension*. Although rare, this condition may cause the most intense persisting dyspnea of any cardiovascular condition. The cause of this disease, as well as the cause of the dyspnea, is obscure; there is obviously an important pathophysiologic mechanism operating, even though we do not fully understand what it is. Owing to widespread occlusion of small radicles of the pulmonary vascular bed, the corresponding alveoli cease to be perfused and a gross imbalance between ventilation and perfusion ensues, with much wasted ventilation. The important realization is that the presence of pulmonary hypertension per se is not necessarily associated with

* Much confusion is caused by the misinterpretation of rales at the lung bases. It is true that rales are heard in left heart failure as previously described. In practice, however, it happens that most rales are due to sputum in the air passages, and the inference that they are due to heart failure is more often wrong than right, especially if the rales are coarse, or disappear with several coughs. In the early stages, when it is important to diagnose left heart failure, rales might be absent; when present, they are commonly due to something else. It is better not to use rales as a major criterion of heart failure.

dyspnea, although there usually is an association. There are numerous causes of pulmonary hypertension (see Chapter 6), and the effect on lung function will vary with the cause.

Dyspnea is marked in *cyanotic congenital heart disease*, in which there is right-to-left shunting of venous blood into the arterial circuit. The mechanism of the dyspnea appears to be different from those cases already mentioned. Clearly, there is a gross ventilation-perfusion ($\dot{V}/\dot{Q}$) abnormality in that a good deal of blood does not become exposed to the alveolar air. However, the immediate mechanism for the shortness of breath appears here to be chemical. On exercise, CO_2-rich blood pours into the arterial circuit and the brain is perfused with blood that is not only hypoxemic but acidemic too—the arterial pH may fall as low as 7.2 on exercise. Under these circumstances the respiratory stimulation is likely to be intense, and may be interpreted as the symptom of dyspnea; which is, incidentally, protective in that it prevents gross and life-threatening deviations of the metabolic state.

In congenital heart disease with *left-to-right shunt* (e g, atrial septal defect, ventricular septal defect, patent ductus arteriosus), dyspnea is sometimes absent, but usually is mild to moderate. The mechanism is probably that of loss of lung compliance due to the increased pulmonary blood flow and pressure.

Dyspnea is an important symptom, the pathophysiologic basis for which is interpreted according to the disease state present. Special forms of dyspnea are: cardiac asthma, which is synonymous with pulmonary edema of cardiac origin; paroxysmal nocturnal dyspnea, which has the same connotation; and the dyspnea of Cheyne-Stokes respiration.

Paroxysmal nocturnal dyspnea (PND), as the name implies, occurs at night, when the subject is supine, (a position that increases the intrathoracic blood content) and when there may be emotional stress because of dreaming. The patient is awakened dyspneic or orthopneic, with a dry cough, and may sit on the edge of the bed, walk around the room, or go to an open window or door. The history of such recurrent episodes is characteristic of significant pulmonary venous congestion. The presence of wheezing and widespread rhonchi, as evidence of accompanying bronchospasm, has given rise to the term *cardiac asthma*. It should be emphasized that in some patients with lung disease the bronchi may become occluded with secretions during sleep, causing the development of the same symptoms of breathlessness, orthopnea, and cough. Hence there may be com difficulty in differential diagnosis, but the problem is usually resolved when it becomes clear that the dyspnea is immediately relieved by coughing up a plug of tenacious sputum.

The symptom of dyspnea involves perception of and reaction to stimuli, and not all patients react alike. Further, adaptation takes place to

some degree. Thus in the early days of mitral valve surgery, it became clear that a reduction of mean left atrial pressure from 35 to 20 mm Hg often left the patient without symptoms, whereas 20 mm mean LAP in a subject who was unaccustomed to it would be accompanied by dyspnea. Accommodation to the factors causing dyspnea can also be the result of organic change, e g, the thickening of pulmonary capillary walls that occurs in mitral stenosis and tends to impede the transudation of fluid.

Cheyne-Stokes respiration in cardiac disease signifies left ventricular failure; periodic breathing also occurs in patients with cerebrovascular disease and in normal subjects at high altitude. There are periods of apnea that may seem to be frighteningly long to observers unaccustomed to them. Then the patient begins to take small breaths, gradually increasing the amplitude of these until there is considerable hyperpnea; dyspnea may be present at this time. The condition is troublesome to patients at night because they tend to wake during the hyperpneic phase and sometimes cry out. (Sedation and hypnotics often make the condition worse, but aminophylline may have a powerful effect in abolishing the periodic respiration.)

CHEST PAIN

Chest pain in cardiovascular disease is most commonly the consequence of hypoxia of the myocardium. This condition can result from too little supply of oxygen, too much demand for it, or both. Other cardiovascular causes of chest pain are *pericarditis*, *pulmonary embolism*, *dissection*, and *neurosis*; noncardiovascular causes from which they have to be distinguished are pneumothorax, esophageal disease, and musculoskeletal abnormality. Each of these types of pain has its own characteristics, and the distinction of one from another is not difficult in most cases. Ischemic cardiac pain is also known as *angina pectoris*, which means literally "squeezing of the breast." Its usual cause was defined as long ago as 1799 by Caleb Parry, and the symptom is brought on by effort or emotion in most cases. The pain is substernal in location with radiation to the arms, to the left more frequently than to the right. The sensation in the chest may be expressed as pain, heaviness, ache, squeezing, or tightness. The sensation in the arms may be pain, heaviness, ache, or numbness, and may also radiate to the neck, the jaw, or the teeth, and occasionally through into the back. Angina pectoris is characteristically short-lived, i e, minutes, and goes away on cessation of the precipitating cause, i e, exertion or emotion. The pain is *not* left inframammary, and is *not* stabbing or knife-like in character; it is more readily provoked after meals, in cold weather, and in the wind. The patient illustrates the

symptom usually by laying the flat of the hand across the sternum, though sometimes he may use his clenched fist. The symptom is often accompanied by dyspnea, which signifies acute left ventricular failure. The symptom is rapidly relieved within a minute or two by nitroglycerine administered sublingually. The pain of acute myocardial infarction often begins with what is described as "indigestion", and may start below the diaphragm before assuming its typical substernal location. Pain that lasts for hours or days is rarely angina, although preinfarction angina, or coronary insufficiency, may be prolonged. Resting (decubitus) angina is of serious import, and often signifies impending infarction. The physiologic determinants of myocardial oxygen demand are heart rate, intramyocardial tension, and the contractile state of the myocardium—hence the angina of paroxysmal tachycardia, severe hypertension, or some varieties of cardiomyopathies. Angina due to increased demand is indistinguishable from that due to decreased supply and may be present also in any condition that causes enough hypertrophy of the myocardium, usually of the left ventricle, e g, aortic stenosis, but sometimes of the right ventricle, e g, pulmonary hypertension. Nocturnal angina occurs in the Prinzmetal variant (see page 113) and in aortic insufficiency.

A summary of both the symptoms that are characteristic of angina pectoris and those that are not is given in the following table. This information is well worth committing to memory.

	Angina Pectoris	
	Is	*Is not*
Location	Diffuse, substernal with radiation	Localized, left inframammary
Quality	Dull, deep, aching, pressing	Shooting, sharp, cutting
Intensity	Mild to moderate with gradual fluctuation	Excruciating, rapidly fluctuating
Duration	Minutes	Seconds or hours
Precipitated by	Effort, emotion, cold	Posture, respiration
Relieved by	Brief rest and nitroglycerine	Lengthy rest and most other measures

The pain of *pericarditis* is precordial, and is closely related to breathing and posture. Thus the pain is accentuated by full inspiration, and the

need to take rapid shallow breaths can give the sensation of dyspnea. Leaning forward may abolish the pain. The pain can be sudden in onset and, on occasion, may be substernal, thus mimicking that of acute myocardial infarction.

The pain of *pulmonary embolism* is classically sudden in onset and then pleuritic; i e, it may be felt anywhere in the chest, but is accentuated by inspiration. Depending on the size of the embolus, dyspnea and cardiovascular collapse may also be present (see Chapter 10).

Dissection of the aorta is signaled by the sudden onset of severe chest pain that goes through the back or to the abdomen and persists for several hours. Such a symptom in a hypertensive patient should immediately raise the question of dissection, and the appropriate clinical signs and laboratory evidence should be sought to differentiate the cause from myocardial infarction (see Chapter 10).

The pain of *cardiac neurosis* is left inframammary, stabbing or prolonged, and may also radiate to the left arm.

FATIGUE

Fatigue is a highly variable symptom, as it is in people without heart disease. It correlates best with impairment of cardiac output, but also with personality and motivation.

PALPITATION

Awareness of the heartbeat is physiological in most of us after strenuous exercise. In cardiac patients palpitation is usually due to arrhythmia, paroxysmal or otherwise. Patients with atrial fibrillation complain of palpitation on effort if the ventricular rate speeds unduly. Extrasystoles are often felt as a turning over or a flip-flop of the heart in the chest, but many patients are unaware of them. Patients are often conscious of the sudden onset or cessation of a paroxysmal supraventricular or ventricular tachycardia. Arrhythmias that are especially rapid or that compromise circulatory function may be accompanied by considerable anxiety or by *angor animi* (the feeling of impending doom), and may lead to syncope or collapse. Patients can sometimes describe the rhythm or rate of their arrhythmias.

SWELLING

Swelling in heart disease is usually the consequence of edema formation. In this instance, the swelling is always associated with elevation of the venous pressure—except after diuresis. This fact is important to realize;

the patient who is admitted to the hospital in the afternoon may, after a considerable diuresis, show a normal venous pressure by the following morning, but may still have ankle or sacral edema. Enlargement of the liver, if rapid, is painful. This pain may occur only on effort, and may be confused with angina pectoris, although its abdominal location usually gives the clue. Ascites occurs especially with disease of the tricuspid valve and with constrictive pericarditis.

Fluid retention of from 5 to 10 pounds gives clinical evidence of edema. Before this point is reached, fluid retention may be expressed in subtle ways, e g, tightness of the trousers, increase in belt size, fullness of the head, tightness of rings on the hands, or puffiness of the face in the morning. If there is doubt as to whether or not there is fluid retention, it is good practice to perform a therapeutic test with a diuretic such as furosemide, 20 or 40 mg. Diuresis, weight loss, and relief of dyspnea are usually prompt if excess fluid is present.

FAINTING

The medical word for sudden faint is *syncope*. The swoon and the smelling salts with which it was treated are far less fashionable today than they were in the Victorian era; swooning is usually vasovagal in origin.

Syncope is defined as temporary loss of consciousness due to generalized cerebral ischemia. Syncope is usually due to a fall in cardiac output, as occurs in complete heart block, asystole, rapid ventricular tachycardia, or ventricular fibrillation, or to marked peripheral vasodilatation in the presence of a fixed, low cardiac output, as probably occurs in aortic stenosis. In the latter case, syncope occurs on effort, and may be due to the stimulation of baroceptors in the left ventricular wall in response to the high intracavitary pressure. This stimulation leads to peripheral vasodilatation and bradycardia, a response that is appropriate in hypertension, but disastrous in aortic stenosis where the arterial pressure is already low. Other forms of syncope are reflex vagotonic ones that are associated with cough, micturition, or posture.

Adams-Stokes (or Stokes-Adams) seizures are episodes of sudden loss of consciousness, sporadic and unpredictable, that occur in the setting of atrioventricular block as a consequence of either extreme slowing or quickening of the ventricular pacemaker. The attacks may or may not be associated with seizures. Pallor is a feature of the attacks, with reactive rubor (redness of the face) following the resumption of effective cardiac output.

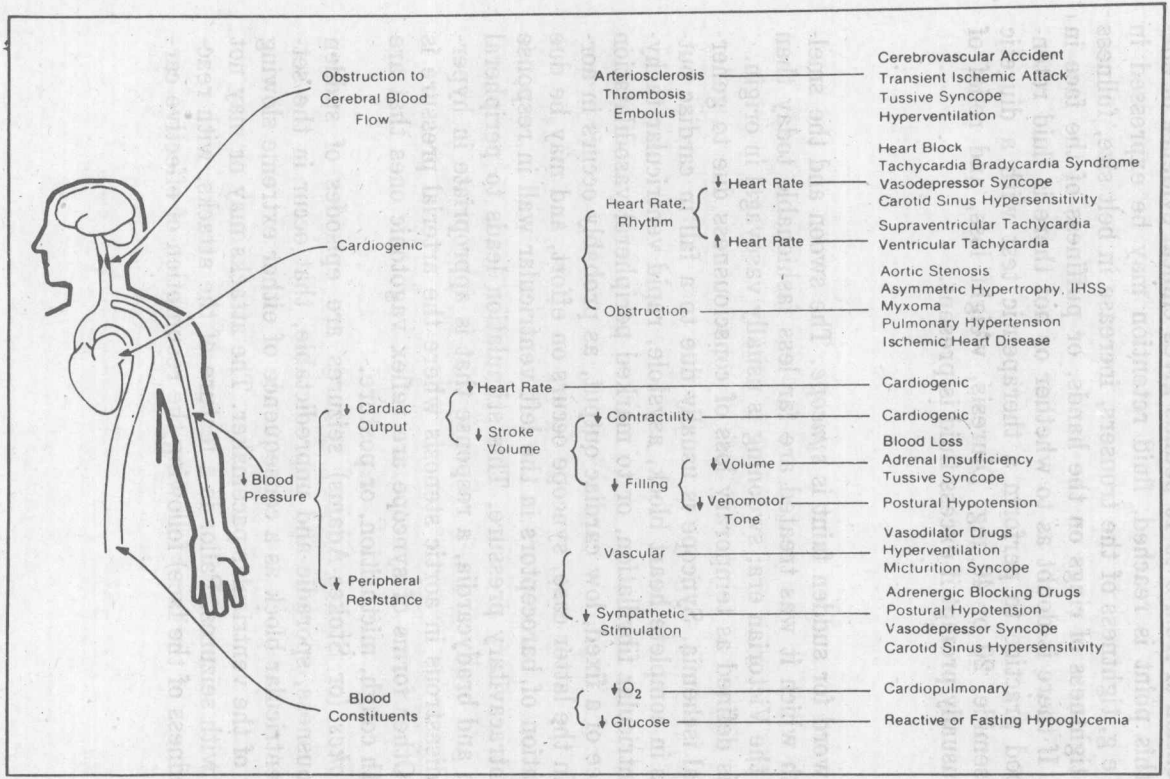

Fig. 1.1 Causes of syncope. Reprinted by permission. JAMA 237:1372-1376, ©1977. (March 28, 1977)