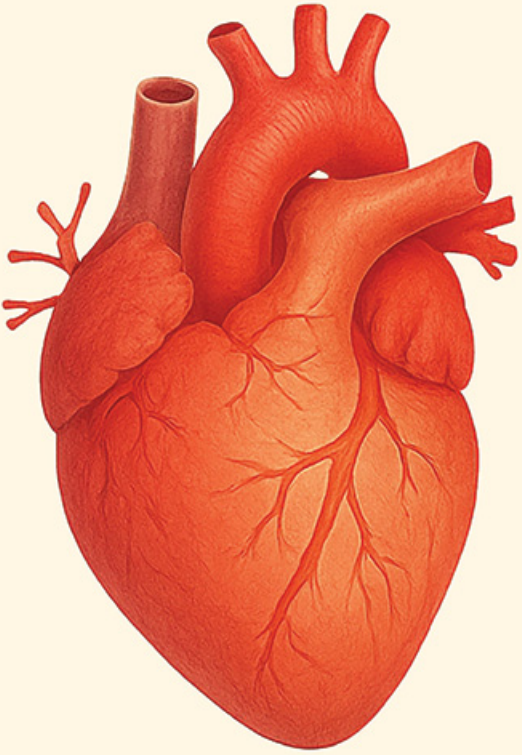

Manual of Cardiology

Kanu Chatterjee

JAYPEE

Contents

Chapter 2

Diagnosis

2.1 History and Physical Examination

The history and physical examination are essential, not only for the diagnosis of cardiovascular disorders but also to assess severity, to establish a plan of management and to assess the prognosis. Appropriate history and physical examination are also essential to decide what tests are necessary for a patient, as presently a plethora of tests is available for the diagnosis and management of the same cardiac disorder. It should also be appreciated that "history and physical examination" are cost-effective. During examination of the patient, the physician can also gain the confidence of the patient and of the family and can establish a good rapport that is necessary for the appropriate management of the problem of the patient.

❑ HISTORY

General Approach

During history taking, it is desirable to allow the patient to present the symptoms without interruption. Frequent interruptions give the impression that the physician is in hurry and impatient and disinterested. After the patient describes the symptoms, it is appropriate to discuss with the patient and the family to ascertain the chronology of symptoms and their severity. It is pertinent to enquire about each symptom. The major symptoms associated with cardiovascular disorders are chest pain or discomfort, dyspnea, palpitations, dizziness, and syncope.

Analysis of Symptoms

Chest Pain or Discomfort

Chest pain is one of the very common presenting symptoms that patients present to the cardiologists. The chest pain or discomfort can be caused by various cardiac and noncardiac causes, which are summarized in Tables 1 to 4.

Angina pectoris is a symptom of both of chronic coronary artery disease and of acute coronary syndromes. For the diagnosis of angina pectoris, it is imperative to enquire about the character, location, site of radiation, duration, and precipitating and relieving factors of the chest discomfort. While elucidating the history, the following thing should be noted:

Table 1: Cardiac chest pain

- Coronary artery disease
- Acute coronary syndromes
- Stable angina
- Ischemic cardiomyopathy
- Noncoronary artery disease
- Aortic dissection
- Acute pulmonary embolism

Table 2: Noncardiac chest pain

- Pulmonary
  - Pleuritis
  - Pneumonia
  - Tracheobronchitis
  - Pneumothorax
  - Mediastinitis
  - Tumor

Table 3: Cardiac causes of chest discomfort

- Aortic stenosis
- Aortic regurgitations
- Hypertrophic cardiomyopathy
- Restrictive cardiomyopathy
- Pulmonary hypertension

Table 4: Noncardiac chest pain

- Musculoskeletal
  - Costochondritis
  - Intercostal muscle cramps
  - Cervical disk disease
- Other causes
  - Herpes zoster
  - Emotional
  - Chest wall tumor
  - Disorders of breast

Character: Frequently, it is described as "heaviness", "pressure", "tightness", "squeezing" or "band across the chest". The character of angina pectoris is usually "dull and deep" and not "sharp and superficial".

Location: The location of the chest discomfort can be retrosternal, epigastric or left pectoral. When chest pain is much localized and can be indicated by one or two finger tips, it is unlikely to be angina pectoris.

Radiation: The radiation of angina pain may be to one or both shoulders, one or both arms and hands, one or both sides of the neck, lower jaw and interscapular area. The radiation can also occur to armpits, epigastrium and subcostal areas. The pain radiates from the center to the periphery (centripetal) and rarely centrifugal.

Intensity: The intensity of angina increases slowly and reaches its peak in minutes, not instantaneously. Similarly, it is relieved gradually, not abruptly. Analysis of the duration of chest discomfort is also helpful to decide whether it is ischemic or nonischemic pain.

Gestures: Patient's gestures during describing the chest discomfort have been thought to be useful in the diagnosis of its etiology.

Precipitating and relieving factors: The precipitating and relieving factors of the chest discomfort should be analyzed to determine its etiology. Classic angina (Heberden's angina) is precipitated by exercise or by emotional stress and is relieved when the exercise is discontinued. It tends to occur often after meals.

Exposure to cold weather precipitates angina more easily in patients with classic angina. Similarly, carrying heavy objects and heavy meals are also frequent precipitating factors. The character, location, and radiation of chest discomfort are similar in the different clinical subsets of angina. However, some distinctive features can be recognized in various subsets.

The atypical presentations frequently called "angina equivalents" are dyspnea, indigestion and belching, and dizziness and syncope without chest pain. The atypical presentations are more common in diabetics, women, and the elderly. A few clinical features of angina are summarized in Tables 5 and 6.

Table 5: Clinical features of stable angina

- Location
  - Usually retrosternal, can be epigastric, interscapular
- Localization
  - Usually diffuse, difficult to localize
  - When is very localized (point sign)—unlikely to be angina
- Quality
  - Pressure, heaviness, squeezing, indigestion
- Radiation
  - One or both arms, upper back, neck, epigastrium, shoulder
  - Lower jaw (upper jaw, head, lower back, lower abdomen, or lower extremities radiation is not feature of angina)
- Duration
  - Usually 1–10 minutes (not a few seconds or hours)
- Precipitating factors
  - Physical activity, emotional stress, sexual intercourse
- Aggravating factors
  - Cold weather, heavy meals
- Relieving factors
  - Cessation of activity, nitroglycerin (if relief is instantaneous it is unlikely to be angina)
- Associated symptoms
  - Usually none, occasionally dyspnea

Table 6: Clinical features of chest pain in acute coronary syndromes

- Location
  - Same as stable angina
- Quality
  - Same as stable angina
- Duration
  - Usually longer than stable angina
- Relieving factors
  - Usually unrelieved by rest or nitroglycerin
- Associated symptoms
  - Dyspnea, sweating, weakness, nausea, vomiting presyncope, or syncope

Chest pain resulting from acute aortic dissection is usually severe. The location can be anterior chest. Radiation to the back is common. The downward radiation along the spine is very suggestive of aortic dissection. The onset of pain is frequently instantaneous and the maximal severity may occur at the onset. A few clinical features of pain of acute pericarditis, pulmonary embolism and acute aortic dissection are summarized in Table 7.

The severity of angina is assessed by the Canadian Cardiovascular Society (CCS) functional classification (Table 8) or specific activity scale (Table 9). CCS is most frequently used to assess the severity of angina.

In suspected patients inquiries should be made about the risk factors. The modifiable and nonmodifiable risk factors for atherosclerotic coronary artery diseases are summarized in Table 10.

Dyspnea

Dyspnea is an uncomfortable sensation of breathing. It is also defined as "labored" breathing. Dyspnea can occur during exertion (exertional), during recumbency

Table 7: Clinical features of chest pain due to acute pericarditis, acute pulmonary embolism, and acute aortic dissection

Acute pericarditis	Acute pulmonary embolism	Acute aortic dissection
• Location ▪ Anterior chest, superficial • Character ▪ Sharp, can be pleuritic • Radiation ▪ Supraspinatus areas, shoulders, back • Relieving factors ▪ Worse in supine position, less severe in sitting and leaning forward position, relieved by analgesics, nonsteroidals and steroids	• Location ▪ Usually retrosternal • Quality ▪ Deep, may be similar to acute coronary syndromes • Associated symptoms ▪ Tachypnea and dyspnea	• Location ▪ Chest, back • Quality ▪ Shearing, tearing • Onset ▪ Instantaneous • Radiation ▪ Downwards along the spine

Table 8: Canadian Cardiovascular Society (CCS) functional classification

Class I
- Ordinary physical activity, such as walking and climbing stairs, does not cause angina
- Angina with strenuous or rapid or prolonged exertion at work or recreation

Class II
- Slight limitation of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, in cold, in wind or when under emotional stress, or only during the few hours after awakening
- Walking more than two blocks on the level, and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions

Class III
- Marked limitation of ordinary physical activity
- Walking 1–2 blocks on the level and climbing more than one flight in normal conditions

Class IV
- Inability to carry on any physical activity without discomfort—anginal syndrome may be present at rest

Table 9: Specific activity scale

Class I
- Patients can perform to completion any activity requiring ≤7 metabolic equivalents [e.g. can carry 24 lbs up eight steps; carry objects that weigh 80 lbs, do outdoor work (shovel snow, spade soil); do recreational activities (skiing, basketball, squash, handball, jog/walk 5 mph)]

Class II
- Patients can perform to completion any activity requiring ≤5 metabolic equivalents (e.g. have sexual intercourse without stopping, garden, rake, weed, roller skate, dance fox trot, walk 4 mph on level ground), but cannot and do not perform to completion activities requiring ≥7 metabolic equivalents

Class III
- Patients can perform to completion any activity requiring ≥2 metabolic equivalents (e.g. shower without stopping, strip and make bed, clean windows, walk 2.5 mph, bowl, play golf, dress without stopping), but cannot and do not perform to completion any activities requiring≥ 5 metabolic equivalents

Class IV
- Patients cannot or do not perform to completion activities requiring ≥2 metabolic equivalents. Cannot carry out activities listed above (Specific Activity Scale Class III)

Table 10: Cardiac and pulmonary causes of dyspnea

Differential diagnosis of dyspnea	
Cardiac	*Pulmonary*
• CHF	• COPD
• CAD	• Asthma
• Cardiomyopathy	• Restrictive lung diseases
• Valvular dysfunction	• Hereditary lung diseases
• LVH	• Pneumothorax
• Pericardial diseases	
• Arrhythmias	
• Congenital HD	

(Abbreviations: CHF: Congestive heart disease; HD: Heart disease; CAD: Coronary heart disease; LVH: Left ventricular hypertrophy)

Table 11: Cardiac cause of dyspnea

Dyspnea
Cardiac or noncardiac dyspnea
Physical examination:
• Signs of heart failure—diagnostic of cardiac cause, e.g. S3, elevated JVP, positive HJR
Presence of cardiac pathology:
• Very suggestive of cardiac cause
Chest X-ray:
• Very helpful when findings of pulmonary venous congestion or pulmonary hypertension are present
ECG:
• Normal electrocardiogram
• A negative predictive value has over 90%

(Abbreviations: JVP: Jugular venous pressure; HJR: Hepatojugular reflux)

(orthopnea) or even with standing (platypnea). There are both cardiac and noncardiac (Table 10) causes of dyspnea. Many patients have both cardiac and pulmonary disease. To distinguish between cardiac and noncardiac dyspnea, the measurements of serum B-type natriuretic peptide (BNP) or N-terminal ProBNP (NTBNP) is helpful. In noncardiac dyspnea, the natriuretic peptide levels are normal, and in patients with heart failure, they are substantially elevated.

Exertional dyspnea can be caused by both cardiac and noncardiac causes. Exertional dyspnea is an important symptom of chronic heart failure. Orthopnea is defined when patients develop dyspnea lying flat and feel better when the upper part of the torso is elevated.

There are many cardiac conditions, which can be associated with episodic severe dyspnea. In between the episodes of dyspnea, these patients are relatively asymptomatic and may have good exercise tolerance. Careful cardiovascular examination may also reveal the etiology of dyspnea. For example, evidence of valvular and myocardial heart diseases suggests cardiac cause of dyspnea (Table 11).

Palpitation

Palpitation is perceived as an uncomfortable sensation in the chest associated with heartbeats. The most frequent cause of palpitation is premature atrial or ventricular contractions. The premature beat itself is not felt; the normal beat following the

compensatory pause is felt as a strong beat. The patients usually describe this uncomfortable sensation as "a thump", "skipped beat", "the heart is coming out of chest", "heart stops" and "heart stops beating". The frequent premature beats may also be associated with other symptoms, such as dizziness, sinking feeling, shortness of breath and chest pain.

Syncope

Syncope is defined as transient loss of consciousness. Patients with presyncope complain of dizziness and near fainting, although they do not loose consciousness completely. The mechanism of cardiac syncope is reduced cerebral perfusion resulting from decreased cardiac output and hypotension.

A careful history is helpful for the diagnosis of the cause of syncope. Enquiry should be made about the circumstances in which syncope occurred, whether it was accompanied or preceded by palpitation, whether it occurs during exertion or it can also occur at rest, and whether it occurs only during upright position or it is unrelated to body position.

Edema

Patients with edema present with the symptom of "swelling", usually of the lower extremities. Both cardiac and noncardiac conditions may be associated with edema. Enquiries should be made regarding the initial location and progression of edema. Right heart failure with systemic venous hypertension causes dependent edema, such as in the ankles, feet, and legs. In bedridden patients, edema can be predominantly in the back. Generalized edema is uncommon in heart failure and usually suggests permeability edema, such as in patients with hypoalbuminemia.

Cough

Paroxysms of cough may be the presenting symptoms in cardiac and noncardiac patients. Patients with left heart failure may complain of nocturnal cough with or without dyspnea. Paroxysms of nonproductive cough are bothersome complications of angiotensin-converting enzyme inhibitor therapy.

Hemoptysis

Hemoptysis is an uncommon presenting symptom of cardiac patients. Patients with hemodynamic pulmonary edema may present with history of frothy pink, blood-tinged sputum. These patients also have dyspnea.

Recurrent hemoptysis may be a presenting symptom in patients with precapillary pulmonary arterial hypertension and Eisenmenger's syndrome. Hemoptysis associated with pleuritic chest pain should raise the suspicion of pulmonary embolism. Patients on anticoagulation therapy may present with hemoptysis.

It should be appreciated, however, that frank hemoptysis is an uncommon presenting symptom of cardiac patients, and primary bronchopulmonary disease, such as malignancy, should always be suspected.

❑ PHYSICAL EXAMINATION

Physical examination like history taking, is an integral part of evaluation of a patient with suspected or established cardiovascular disorders.

General Appearance

The physical examination starts with the inspection. During inspection, the physician has the opportunity to observe the patient's expression, skin color, posture, and general health status. During inspection, the nutritional status of the patient can be determined. Obesity or cachexia can easily be recognized. Obesity is a risk factor for metabolic syndrome, coronary artery disease, and heart failure.

Examination of the Skin

Examination of the color of the skin can reveal presence of cyanosis, jaundice and slaty, and bronze discoloration. Cyanosis is characterized by bluish discoloration of the skin and mucous membrane. Most frequently cyanosis is due to presence of abnormal amount of reduced hemoglobin. Cyanosis can be central, peripheral or mixed. Central cyanosis is due to intracardiac or intrapulmonary right-to-left shunt. The amount of desaturated hemoglobin is increased in central cyanosis and best recognized inspecting the buccal mucous membrane, tongue, and oropharyngeal mucous membrane.

Jaundice due to hyperbilirubinemia is occasionally seen in patients with severe right heart failure associated with congestive hepatopathy. Patients with portopulmonary hypertension may also have jaundice. A few abnormalities of skin, which can be associated with cardiovascular disorders are summarized in Table 12.

Examination of the Musculoskeletal System

The majority of congenital heart disease with musculoskeletal abnormalities is encountered in the pediatric population. In adult cardiology practices a few conditions, although distinctly uncommon, may be seen (Table 13).

Table 12: Skin abnormalities and cardiovascular disorders

- Cyanosis
  - Central—intracardiac and intrapulmonary right-to-left shunt
  - Peripheral—low cardiac output, increased peripheral oxygen extraction
- Methemoglobinemia—bluish discoloration of the skin
  - Hereditary (rare) and acquired (nitrate and nitrite toxicity)
- Jaundice—yellow discoloration
  - Prosthetic valve malfunction—hemolysis
  - Portopulmonary hypertension
  - Severe congestive hepatopathy
- Bronze discoloration—slaty color of the skin
  - Primary or secondary hemochromatosis
  - Atrial or ventricular arrhythmias
  - Restrictive cardiomyopathy, dilated cardiomyopathy
- Amiodarone skin toxicity—benign
- Butterfly rash of the face—lupus erythematosus
  - Valvular disease (Libman–Sack endocarditis)
  - Pulmonary arterial hypertension
- Malar flush of the face—
  - Severe mitral stenosis
  - Severe precapillary pulmonary hypertension
- Plantar and palmar keratosis and wooly hair—naxos disease
  - Arrhythmogenic right ventricular dysplasia

Contd...

Contd...

Table 12: Skin abnormalities and cardiovascular disorders

- Telangiectasia of lips, tongue and buccal mucous membrane—
  - Osler–Weber–Rendu syndrome
  - Arteriovenous malformations
- Xanthomatosis—tendon xanthoma, xanthoma in the palmar crease, with or without xanthelasma—familial hyperlipidemia
  - Premature coronary artery disease
- Cutaneous lentiginosis—LEOPARD syndrome
  - Conduction defects, congenital pulmonary stenosis
- Petechiae and purpuric skin rash—bacterial endocarditis
  - Valvular heart disease
- Blotchy cyanosis—carcinoid heart disease
  - Right-sided valvular heart disease
- Livid reticularis—reticular purplish skin rash
  - Lupus erythematosus
  - Valvular heart disease, pulmonary hypertension
  - Blue toes syndrome
  - Cholesterol emboli
- Atrophic skin lesions—necrobiosis diabeticorum
  - Increased risks of cardiovascular disease
- Annular skin rash with clear center—Lyme disease
  - Heart block, myocarditis
- Tightening of the skin, flexion contractures of the fingers, telangiectasia-scleroderma, CREST syndrome
  - Pulmonary arterial hypertension

Table 13: Musculoskeletal abnormalities associated with cardiovascular disorders

- Marfan's syndrome
  - Aortic regurgitation, mitral regurgitation, aortic disease
- Ehler–Danlos syndrome
  - Mitral regurgitation, aortic disease
- Turner's syndrome
  - Coarctation of aorta
- Holt–Oram syndrome
  - Atrial septal defect
- Down syndrome
  - Ventricular septal defect, atrioventricular cushion defects
- Rheumatoid arthritis
  - Aortic regurgitation, heart block
- Ankylosing spondylosis
  - Aortic and mitral valve disease, heart block
- Clubbing of the fingers and toes
  - Congenital cyanotic heart disease, bacterial endocarditis
- Straight back syndrome
  - Mitral valve prolapse
- Clubbing of fingers and toes
  - Congenital cyanotic heart disease, bacterial endocarditis

Finger and toes should be examined for clubbing. The drumstick type of clubbing is seen in cardiovascular diseases, such as congenital cyanotic heart disease and bacterial endocarditis. Bacterial endocarditis can be also associated

with splinter hemorrhage, Janeway and Osler nodes and valvular regurgitations. Heberden's nodes, which are usually seen in the fingers, result from osteoarthritis and are not associated with cardiovascular disorder.

Measurement of Arterial Pressure

At present, in most institutions, automated techniques are used for the measurement of blood pressure. The various techniques of measuring blood pressure, their advantages and disadvantages, and pitfalls are discussed in the section on clinical hypertension.

Examination of the Jugular Venous Pulse

Careful examination of the jugular venous pulse and pressure provides information regarding the hemodynamic changes in the right side of the heart. It has been suggested that for estimation of jugular venous pressure, it is easier and preferable to examine the external jugular vein. However, it has also been suggested that if pulsation is present and visible, the examination of the internal jugular veins is preferable to that of the external jugular veins, as the internal jugular veins are in a direct line with the superior vena cava.

Estimation of Jugular Venous Pressure

The jugular venous pressure can be estimated by examining either external or internal jugular veins (Fig. 1). Conventionally, the upper torso is elevated to 30–40 degrees and the top of the venous pulsation is determined and 5 cm is added to the height assuming that right atrium is located 5 cm below the sternal angle (angle of Louis). However, it has been suggested that 10 cm should be added rather than 5 cm, if the torso is elevated to 45 degrees or greater. It should be appreciated that the external jugular venous pulse may not be recognized in patients with a fat and short neck. Kinking and thrombotic obstruction of the external jugular veins may also cause a spuriously higher central venous pressure.

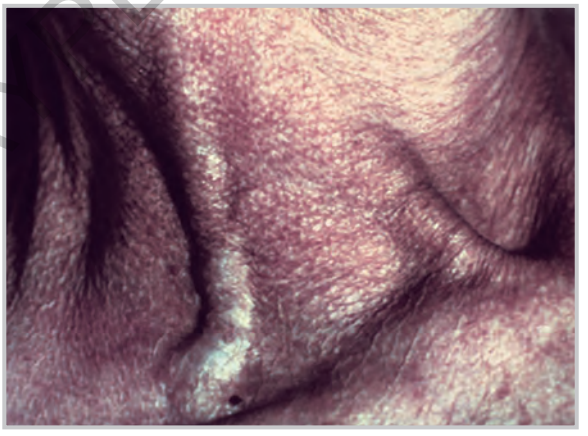

FIGURE 1: Courses of the external and internal jugular veins. The external jugular vein runs from lateral to the medial side of the neck across the sternocleidomastoid muscle. The internal jugular vein starts at the root of the neck in between the two heads of the sternocleidomastoid muscle and runs superiorly toward the angle of the jaw

Table 14: Causes of increased central venous and right atrial pressures

- Tricuspid valve obstruction—
  - Rheumatic tricuspid valve stenosis (usually associated with mitral and/or aortic valve disease)
  - Right atrial myxoma
  - Carcinoid heart disease
  - Neoplastic disease
- Right ventricular failure—
  - Systolic—
    Primary-RV infarction
    Secondary-pulmonary hypertension
  - Diastolic—
    Right ventricular hypertrophy
  - Pericardial disease
  - Pericardial effusion
  - Constrictive pericarditis
- Primary tricuspid regurgitation—
  - Traumatic
  - Ruptured chordae
  - Ebstein's anomaly
  - Carcinoid heart disease
  - Rheumatic heart disease
  - Neoplastic disease
- Generalized volume overload—
  - Glomerulonephritis
  - Anemia
  - Large atriovenous communications
  - Isolated right ventricular volume overload
  - Atrial septal defects

Elevated central venous pressure suggests that the right atrial pressure is elevated. The upper limit of normal right atrial pressure in the supine position is about 7 mm Hg. Some of the causes of increased central venous and right atrial pressures are summarized in Table 14.

Jugular Venous Pulsations

The jugular venous pulse characters are best analyzed by examining the internal jugular veins. When the right atrial pressure waveforms are recorded during cardiac catheterization, three positive waves (a, c and v) and two negative waves (x and y descents) are recognized. The "a" wave occurs during atrial systole with increased right atrial pressure due to atrial contraction (Fig. 2). The "c" wave is related to bulging of the closed tricuspid valve into the right atrium at the beginning of the right ventricular systole. The "x" descent is primarily due to atrial relaxation with a fall in right atrial pressure. The downward descent of the tricuspid valve apparatus also contributes to the genesis of the "x" descent.

After complete relaxation of the right atrium and the nadir of "x" descent, the right atrial pressure rises as the systemic venous return to the right atrium continues. With the onset of right ventricular systole when the tricuspid valve closes, the right atrial pressure rises and the "v" wave begins. The right atrial pressure continues to rise as the systemic venous return to the right atrium continues.

The peak of the "v" wave coincides with the end of the right ventricular systole and can be recognized by timing with the down slope of the carotid pulse. The "y" descent begins with the opening of the tricuspid valve and continues during the rapid filling phase of the right ventricle with a concurrent pressure decline in the right atrium.

The hepatojugular reflux, also called abdominojugular reflux, is assessed during sustained abdominal compression by applying firm pressure over the abdomen for 10–15 seconds. Normally, during abdominal compression, the jugular venous pressure increases transiently by 1–3 cm.

Arterial Pulse

The contour, character and amplitude of the arterial pulses are related to left ventricular stroke volume, left ventricular velocity of ejection, aortic dP/dT,

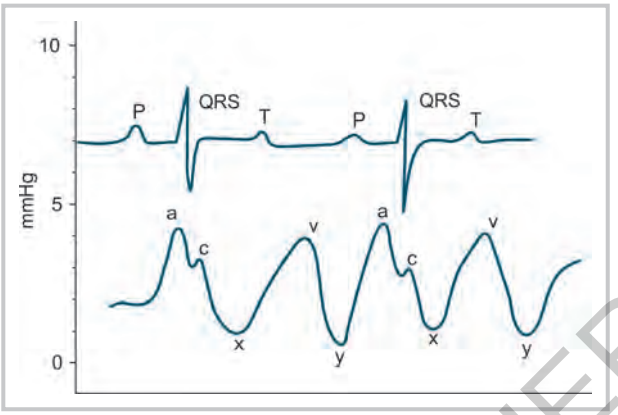

FIGURE 2: The schematic illustrations of right atrial pressure waveforms, which reveal three positive "a", "c" and "v" waves and two negative waves "x" and "y" descent. The "a" wave occurs during atrial systole following P wave of the electrocardiogram. The "c" wave occurs at the onset of right ventricular systole when the closed tricuspid valve bulges into the right atrium. It occurs just after the QRS complex of the electrocardiogram. The "x" descent is related to atrial relaxation. The peak of the "v" wave coincides with the end of right ventricular systole. It coincides with the end of the T-wave of the electrocardiogram. The "y" descent is caused by the opening of the tricuspid valve and during the rapid filling phase (P: P wave; QRS: QRS complex; T: T wave. Normally the magnitude of "a" wave is greater than "v" wave and is less than 7 mm Hg)

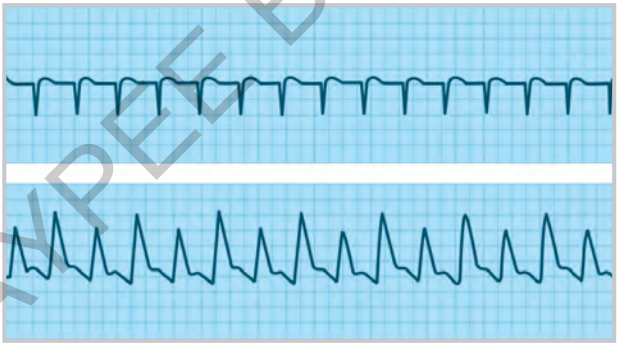

FIGURE 3: The characteristic "strong and weak alternating sequences of arterial pulses" in a patient with systolic heart failure are illustrated. The upper panel is an electrocardiogram showing normal sinus rhythm. The lower panel is directly recorded arterial pressure, showing alternating higher and lower arterial pressure

systemic vascular resistance, and the capacity and compliance of the arterial vascular system.

Examination of the volume and contours of arterial pulses provides important diagnostic clues regarding the underlying etiology of the pathophysiologic condition. Decreased amplitude may reflect reduced stroke volume irrespective of its etiology. Pulsus alternans in presence of regular rhythm indicates reduced left ventricular ejection fraction (Fig. 3).

Table 15: Cardiovascular physical examination

Palpable precordial impulses

- Prominent systolic left parasternal impulse
  - RV failure
- Sustained LV apical impulse
  - Reduced LVEF
  - Increased LV mass
- Palpable PA impulse
  - Left-to-right shunt
  - Pulmonary hypertension
  - Pulmonary stenosis

(Abbreviations: LV: Left ventricle; RV: Right ventricle; LVEF: Left ventricle ejection fraction; PA: Pulmonary artery)

It should be appreciated that the absence of pulsus alternans does not exclude systolic heart failure. However, the presence of pulsus alternans is almost diagnostic of reduced left ventricular ejection fraction, although it is present in only about 10% of patients with chronic systolic heart failure. In patients with acute coronary syndrome, in approximately 25% of patients pulsus alternans can be appreciated.

The pulsus paradoxus is characterized by a fall in arterial pressure during inspiration more than 10 mm Hg. Normally systolic arterial pressure falls during inspiration by an average of 8–12 mm Hg. In cardiac tamponade there is a substantially greater decrease in the arterial pressure during inspiration. The magnitude of the pulsus paradoxus can be better appreciated if the blood pressure is recorded by the sphygmomanometer. The cuff pressure should be decreased slowly. The systolic pressure at expiration is noted. With a further reduction of cuff pressure, the systolic pressure during inspiration is noted and the difference between these two systolic blood pressures provides an estimate of pulsus paradoxus. More severe the tamponade, a greater fall in arterial pressure occurs during inspiration.

Examination of the Precordial Pulsation

Precordial cardiovascular pulsations are best appreciated with the patient in supine position with the upper torso elevated not more than 45 degree. During inspection, the left ventricular apical impulse is usually visible over the left fifth intercostal space medial to the anterior axillary line. In patients with severe volume overload of the left ventricle, such as due to severe mitral or aortic regurgitation, an accentuated left ventricular apical impulse along with pulsation of the entire precordium may be visible. The left ventricular apical impulse, also called apex beat, is examined with the patient in a partial left lateral decubitus position. A few clinical conditions and mechanisms of precordial impulses are summarized in Table 15.

Auscultation

The heart sounds are schematically illustrated in Figure 4. The analysis of the heart sounds should precede analysis of the heart murmurs. The high-frequency heart sounds, such as first (S1) and second (S2) and murmurs due to aortic and mitral regurgitation are better appreciated with the use of the diaphragm of the stethoscope. The lower frequency heart sounds, such as third (S3) and fourth (S4) and mid-diastolic rumbles are better heard with the bell of the stethoscope.

The S1 occurs just before the upstroke of the carotid pulse at the beginning of the isovolumic systole. At the bedside, S1 and S2 are best recognized by timing with carotid pulse upstroke and down stroke, respectively.

Manual of Cardiology

Key Features

- A concise resource which provides up-to-date information in a streamlined easy-to-follow format
- Provides practical information on how to manage cardiac diseases with a focus on hands-on therapeutic guidance for the clinicians
- A primer for those interested in cardiology as well as students pursuing the specialty
- Useful to the clinician for urgent reference in an easy-to-carry format; this book is your constant companion in practice
- Discusses in details topics on cardiovascular pharmacology, diagnostic techniques, cardiovascular electrophysiology, heart failure, acute coronary syndrome, valvular heart disease, systemic disease, and heart and preventive cardiology
- Modified guidelines for the management of angina, arrhythmias, heart failure, valvular heart diseases, and perioperative cardiac evaluations are incorporated
- Written by experts with an enormous cumulative experience, both in practise as well as research
- Numerous tables, figures, and algorithms to assist you for quick reference and decision making.

Kanu Chatterjee MBBS FRCP (London)
FRCP (Edin) FCCP FACC MACP
Clinical Professor of Medicine
Division of Cardiology
The Carver College of Medicine
University of Iowa, Iowa City, Iowa, USA
Emeritus Professor of Medicine
University of California, San Francisco, California, USA

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