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Bedside Cardiology

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3rd Edition

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Contents

1. Bedside Cardiology: Is it Evidence-based? 00
2. Functional Classification 00
3. A Quartet: Cardinal Symptoms in the Cardiovascular System 00
4. A Triad: Minor Symptoms in the Cardiovascular System 00
5. A Triad: Cardinal Symptoms in Left-to-Right Shunt 00
6. A Triad: Cardinal Symptoms in Congenital Cyanotic Heart Disease 00
7. Syndrome and Measurements 00
8. Clinical Instruments 00
9. A Triad: Cardinal Signs in Congenital Cyanotic Heart Disease 00
10. Jugular Venous Pulse 00
11. Arterial Pulse 00
12. Blood Pressure 00
13. Precordium: Inspection, Palpation, and Percussion 00
14. First Heart Sound 00
15. Second Heart Sound 00
16. Third Heart Sound 00
17. Fourth Heart Sound 00
18. Ejection Sound 00
19. Nonejection Sound 00
20. Murmur 00
21. Continuous Murmur 00
22. Innocent Murmur and Sound 00
23. Dynamic Auscultation 00

24. Clinical Assessment: Congestion and Perfusion 00

25. Clinical Assessment: Pulmonary Hypertension 00

26. Segmental Approach in Congenital Heart Disease 00

27. Clinical Approach: Congenital Cyanotic Heart Disease 00

28. Clinical Approach: Tetralogy Physiology 00

29. Clinical Approach: Left-to-right Shunt and Eisenmenger Physiology 00

30. History Taking.....00

Index 00

3 CHAPTER

A Quartet: Cardinal Symptoms in the Cardiovascular System

INTRODUCTION

Dyspnea, palpitation, chest pain, and syncope are the most important cardinal symptoms in the cardiovascular system.

DYSPNEA

Definition

Dyspnea is a term used to characterize a subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity. The American Thoracic Society defines dyspnea as "a subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity." The experience derives from interactions among multiple physiological, psychological, social, and environmental factors and may induce secondary physiological and behavioral responses."[1] Braunwald defined dyspnea in simple words—an abnormally uncomfortable awareness of one's own breathing.[2]

Physiologic Components

- Mechanoreceptors (respiratory muscles)
- Hypoxia (carotid and aortic bodies)
- Changes in pCO_2/pH (medullary center)
- Medullary center (afferent input and efferent output)
- Cortical function (sense of effort)

Basic Mechanism

Circuit

The circuit of dyspnea consists of receptors, sensory cortex, and motor cortex **(Fig. 1)**. The mechanism of dyspnea lies in any incoordination of this complex circuit.

Receptor

Receptors include mechanoreceptors and chemoreceptors. Mechanoreceptors lie in the upper airway and face, lungs, chest wall, and skeletal muscles, including respiratory and exercising muscles. Receptors in the upper airway and face, particularly in the distribution of the trigeminal nerve, modulate

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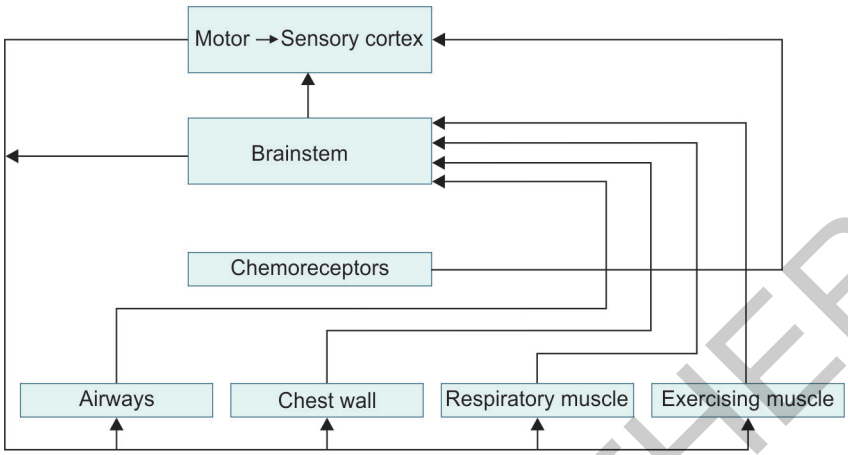


Fig. 1: Dyspnea circuit.

dyspnea.[2] A dyspneic patient gets relief when he is exposed to a fan or open window or cold air. Lungs consist of three types of mechanoreceptors:

1. Juxtacapillary receptors (J-receptors), supplied by C fibers, the unmyelinated nerve endings of the vagus nerve, lie in alveoli and blood vessels. They are stimulated by interstitial congestion.
2. Stimulation of irritant receptors increases the sensation of dyspnea in the form of chest constriction and tightness. Dynamic airway compression causes deformation of these receptors, which send afferent signals through the vagus nerve to the midbrain and sensory cortex.
3. Stimulation of pulmonary stretch receptors similarly sends signals through the vagus nerve only to reduce the sensation of dyspnea.

Receptors on chest wall muscles, including the respiratory muscles and diaphragm, deform on chest wall movement and send afferent signal.

Chemoreceptors play a lesser role in the mechanism of dyspnea. Hypoxia is not a major stimulus. Neither most of the hypoxic patients feel dyspneic nor most of the dyspneic patients are hypoxic. Moreover, correction of hypoxia does not produce much improvement in symptoms. However, hypercapnia may cause dyspnea independent of any reflex increase in respiratory muscle activity. In an experimental study,[3] patients on mechanical ventilation after induction of paralysis by neuromuscular-blocking drugs become dyspneic on the rise of end-tidal carbon dioxide to 7–11 mm Hg. Hypercapnia and consequential change of pH stimulate central chemoreceptors and induce the feeling of dyspnea.

Length-tension Inappropriateness or Afferent Mismatch or Efferent–afferent Dissociation[4,5]

Dyspnea results when there is an imbalance between the perceived need to breathe and the perceived ability to breathe, resulting from a mismatch

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between central respiratory motor activity and incoming afferent information from receptors in the airways, lungs, chest wall structures, and exercising muscle. This is a mismatch between the pressure (tension) generated by the respiratory muscle and the resulting tidal volume (change of length). The afferent feedback from peripheral sensory receptors may allow the brain to assess the effectiveness of the motor commands issued to the ventilatory muscles, i.e., the appropriateness of the response in terms of flow and volume for the command. When changes in respiratory pressure, airflow, or movement of the lungs and chest wall are not appropriate for the outgoing motor command, the intensity of dyspnea is heightened. Thus, any dissociation between the motor command and the mechanical response of the respiratory system may produce a sensation of respiratory discomfort and dyspnea.[6]

Dyspnea in Heart Failure

Lung Hypothesis

This is the time-old concept of explanation of exercise intolerance in heart failure, which leads to pulmonary venous hypertension and capillary congestion with interstitial edema. The lung becomes stiffer with reduced compliance. As mentioned earlier, the J-receptors are stimulated by this congestion, leading to activation of the Hering–Breuer reflex. This reflex does terminate the inspiratory effort before full inspiration is achieved, resulting in rapid and shallow breathing. On the other hand, increased tissue pressure leads to earlier closure of dependent airways with air trapping. These factors altogether behave as a restrictive ventilatory defect. Work of breathing is increased to distend the stiff lungs. Tidal capacity is reduced, and respiratory rate is increased as compensation. Both engorged blood vessels and peribronchiolar cuffing may cause bronchoconstriction with increased airway resistance. There is also ventilation–perfusion mismatch, increased alveolar-arterial oxygen difference, and increased ratio of dead space to tidal volume.

Muscle Hypothesis[7]

According to this hypothesis, skeletal muscle, both respiratory and peripheral, plays the key role in the pathophysiology of exercise intolerance in heart failure. Heart failure leads to a systemic inflammatory response along with an imbalance between anabolic and catabolic factors. There is muscle catabolism leading to respiratory muscle myopathy, causing dyspnea, and peripheral myopathy, causing fatigue.

Skeletal muscle abnormality induces the ergo reflex, a muscle reflex stimulated by work done, through the ergo receptors. Stimulation of the ergo reflex leads to a greater ventilatory response to exercise than normal.

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This results in the sensation of dyspnea. At the same time, this reflex leads to greater sympathetic activation, another feature common during exercise in heart failure.

Heart failure also results in increased levels of proinflammatory cytokines, interleukin-1, and interferon in skeletal muscles. Those cytokines increase the level of nitric oxide synthase. Intracellular nitric oxide level shoots up to a high level and inhibits mitochondrial oxidative phosphorylation. Exercise training reduces inflammation and inflammatory markers and increases mitochondrial volumes in skeletal muscle. Thus, muscle fatigue is reduced, and exercise tolerance is improved in heart failure.

Assessment of Dyspnea

- Mode of onset
- Duration
- Progress
- Severity
- Functional status
- Special character
- Relieving factor
- Associated symptoms

Severity

Severity of symptom is scaled by the guideline of the American Thoracic Society **(Table 1)**.

Formal Measurement of Dyspnea

Dyspnea can be measured formally by the provocative dyspnea scale and the modified Borg scale.

Provocative Dyspnea Assessment Scale

Heart Failure Syndrome Working Group[8] described the provocative dyspnea assessment (PDA) scale to measure dyspnea **(Box 1)**.

TABLE 1: Severity scale of dyspnea.

Grade	*Degree*	*Characteristics*
0	None	Only with strenuous activity
1	Slight	When hurrying on level ground or climbing a slight incline
2	Moderate	Needs to walk more slowly than others of the same age or has to stop for breath when walking at own pace on level ground
3	Severe	Stops for breath after 100 yards or after a few minutes
4	Very severe	Housebound or dyspnea when dressing or undressing

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BOX 1: Provocative dyspnea assessment scale.

- Sitting upright (>60°) with oxygen supplementation
- Sitting upright (>60°) without oxygen
- Supine (<20° head elevation) without oxygen
- Walking 50 m as fast as possible
- 6-minute walk test

TABLE 2: Modified Borg scale of dyspnea.

Scale	*Severity*
0	No breathlessness at all
0.5	Very very slight (just noticeable)
1	Very slight
2	Slight breathlessness
3	Moderate
4	Somewhat severe
5	Severe breathlessness
6	??
7	Very severe breathlessness
8	??
9	Very very severe (almost maximum)
10	Maximum

Modified Borg Scale

The modified Borg scale is a reliable assessment tool for dyspnea. Patients use to find that the language in this scale adequately expressed their dyspnea **(Table 2)**.

Ventilatory capacity is measured prior to exercise, ventilation is measured during exercise, and these are related to the intensity of dyspnea rated using a visual analog scale (VAS), which consists of a line, usually 100 mm in length, placed either horizontally or vertically upon a page, with anchors to indicate extremes of a sensation. The anchors on the scale have not been standardized, but "not breathless at all" to "extremely breathless" and "no shortness of breath" to "shortness of breath as bad as can be" are frequently used. Scoring is accomplished by measuring the distance from the bottom of the scale (or left side if oriented horizontally) to the level indicated by the subject. The reliability and validity of the VAS as a measure of dyspnea have been reported **(Fig. 2)**.

Orthopnea

Orthopnea is defined as dyspnea during recumbency. In the horizontal position, there is redistribution of blood volume from the lower extremities

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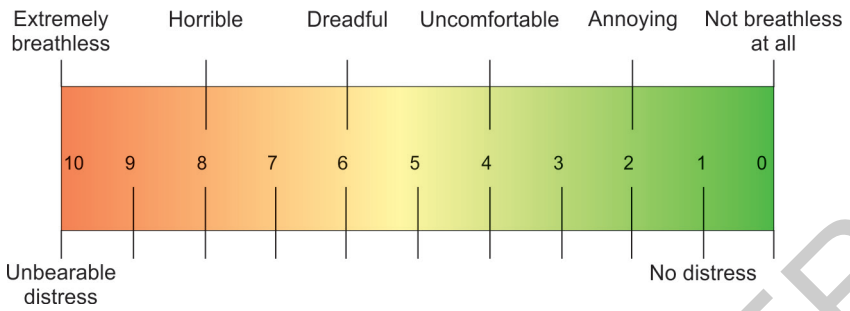


Fig. 2: Visual analog scale.

and splanchnic beds to the lungs. Moreover, lung compliance and vital capacity are reduced significantly, around 5% in the normal population and 20% in obese patients in the recumbent position.[9] In normal individuals, this has little effect, but in patients with heart failure, the dysfunctional left ventricle cannot pump the additional volume out. Besides, in heart failure, there may be reabsorption of edema fluid from previously dependent parts of the body. Venous return is increased by 250–500 cc of fluid.[10] In consequence, ventricular end-diastolic volume and pressure rise, leading to more pulmonary congestion and orthopnea.

Orthopnea also occurs in massive ascites, pleural effusion, bilateral diaphragmatic paralysis, and morbid obesity.

Traditionally, orthopnea is classified as orthopnea of choice and orthopnea of necessity.[9] In orthopnea of choice, the patient prefers to sit up, but they can lie down and remain comfortable. In orthopnea of necessity, the patient takes an upright position for self-preservation.

The correlation between orthopnea and raised pulmonary capillary wedge pressure (PCWP) is high, with a sensitivity nearing 90%. In the ESCAPE (Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness) study,[11] history of orthopnea requiring two or more pillows within 1 week predicted a PCWP of >30 mm Hg with an odds ratio of 3.6.

Dyspnea is said to be chronic when it lasts >1 month.[12]

Paroxysmal Nocturnal Dyspnea

In a typical episode of paroxysmal nocturnal dyspnea (PND), the patient is suddenly awakened 2–3 hours after going to sleep, gasping for air. He quickly sits up and puts his feet over the side of the bed, coughing, wheezing, and sweating or going to the window for fresh air. He gets relief only after sitting for 10–15 minutes and usually sleeps the rest of the night. If the symptoms recur, it usually occurs 2–3 hours later.[13]

Pathophysiology

Pathophysiology is complex and different from that of orthopnea and acute pulmonary edema. PND is usually an early feature of heart failure, when the

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patient is usually in New York Heart Association (NYHA) I or II. There is no gross fluid overload. Thus, it takes 2–3 hours for fluid redistribution to the lungs from the periphery. The lymphatic system takes around 10–15 minutes to get redistributed interstitial fluid back into tissue. PND occurs only at night because of diurnal physiology. Sympathoadrenal dominance is replaced by vagal dominance at night. A failing left ventricle cannot pump out additional preload. A fallacy of this theory is that PND occurs during the rapid eye movement phase of sleep, when there may occur an adrenergic surge.

The respiratory center is also unresponsive during night sleep. Nocturnal arrhythmia is another mechanism. During PND, there may be mild hypoxia and raised PCWP. A latest theory is that sleep apnea may precipitate PND.

Cheyne–Stokes respiration, another cause of nocturnal dyspnea in heart failure, may simulate PND. The patient may awaken during the rapid, deep-breathing phase. However, in contrast to PND, it occurs immediately after a patient goes to sleep.[13] Acute pulmonary edema, another nocturnal dyspnea, is more dramatic than PND. Sputum may be profuse, frothy, watery, or blood-tinged. Heart rate and blood pressure may be very high. Hypoxia and central cyanosis may be significant. Lungs may be flooded with bubbling rales, wheezing, and rhonchi.

Most Common Diseases

Common causes of PND are ischemic heart disease with heart failure, mitral stenosis and other valvular heart diseases, dilated cardiomyopathy, etc.

Noncardiac Causes

Noncardiac causes of nocturnal dyspnea are chronic pulmonary disease, including nocturnal asthma and chronic obstructive pulmonary disease, pulmonary embolism, obstructive sleep apnea, anxiety, and hyperventilation. Nocturnal dyspnea associated with chronic pulmonary disease is preceded by a paroxysm of cough before dyspnea and relieved by expectorating the secretion and not by sitting up. Anxious patients may wake from sleep, mostly after a bad dream, and show similar features of PND. However, the episode may occur at any time after going to sleep.

Trepopnea

Trepopnea may occur with asymmetric lung disease when the patient lies with the more affected lung down because of gravitational redistribution of blood flow. Left atrial myxoma, by obstructing mitral inflow in lateral decubitus, can cause trepopnea.

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Platypnea-orthodeoxia Syndrome

Platypnea-orthodeoxia syndrome (POS) is characterized by dyspnea and objective evidence of hypoxemia in an upright position that resolves in a supine posture. Patent foramen ovale (PFO) is present in around 25% of the normal population. The combination of PFO (and other interatrial combinations), Chiari malformation, prominent Eustachian valve, and change in right atrial hemodynamics prepares the stage for POS. There must be two factors to cause POS: First is the anatomic factor, including PFO and small or fenestrated atrial septal defect (ASD), and second are functional factors, including cardiac (pericardial effusion and constrictive pericarditis), pulmonary [pneumectomy, emphysema, and pulmonary arteriovenous (AV) malformation], abdominal (cirrhosis of liver or ileus), or vascular (aortic aneurysm or elongation) producing deformity of the atrial septum, resulting in a right-to-left shunt through PFO and small or fenestrated ASD in upright posture. More common conditions are after right pneumectomy, ascending aortic dilation, and arch surgery.[14]

How to explain a right-to-left shunt across interatrial communication? Standing upright could stretch interatrial communication, allowing redirection of flow from the inferior vena cava through stretched communication to the left atrium. When present, Chiari malformation or Eustachian valve may play an additional role in redirection. Normally, superior vena cava blood is directed to the tricuspid valve, whereas inferior vena cava blood is directed to the upper part of the interatrial septum, before turning downward toward the tricuspid valve. The combination of these different flow patterns produces a clockwise vortex inside the right atrium. The above-mentioned anatomical factors cause horizontal distortion of the heart, right atrium, and interatrial septum, which changes the hemodynamics of the vortex. There is higher dynamic pressure in the outer layer of the vortex, resulting in a differential pressure gradient across the interatrial septum. In consequence, there is a right-to-left shunt across a more stretched interatrial communication on upright posture.[15]

Bendopnea

Bendopnea is dyspnea within 30 seconds of bending. In the first study on bendopnea,[16] Thibodeau et al. measured ventricular filling pressure on bending and found higher filling pressure of both ventricles in all patients on bending. Those with increased baseline filling pressure became more dyspneic on bending. They found that abdominal girth did not play any role in bendopnea. Rather, bending increased intrathoracic pressure, leading to increased filling pressure of both ventricles. All the patients with heart failure in their series who had bendopnea also had orthopnea and increased jugular venous pressure (JVP). The triad—orthopnea, bendopnea, and increased

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TABLE 3: Statistical significance of three symptoms of heart failure.[17]

Symptom	*Sensitivity*	*Specificity*	*Positive predictive value*	*Negative predictive value*
Dyspnea on effort	79%	84%	18.5%	99%
Orthopnea	25%	99%	64%	97%
PND	27%	99%	62%	96%

(PND: paroxysmal nocturnal dyspnea)

JVP—may be the most specific and reasonably sensitive feature of chronic heart failure.

Three related symptoms in heart failure: Dyspnea, orthopnea, and PND have different statistical significance in heart failure **(Table 3)**.

PALPITATION

Palpitation can be defined as uncomfortable awareness of one's own heartbeat. The patient describes the feeling as a disagreeable sensation of palpitation, a skipped or missed beat, or a pounding sensation in the chest. Though occasionally it may indicate life-threatening arrhythmia, most often it is a benign symptom. Even if benign, palpitation is quite a common symptom. Palpitation accounts for 16% of the symptoms for which patients visit their general physician.[18] Palpitation as presenting symptom to a cardiologist is second only to chest pain.

Pathophysiology

The pathophysiology of palpitation is poorly understood. The circuit involves sensory receptors (myocardial, pericardial, and peripheral mechanoreceptors and baroreceptors), afferent pathway (parasympathetic or sympathetic pathway), and brain centers (subcortical area and frontal lobe). A patient feels palpitation when cardiac contraction is too rapid or very slow and irregular. Patients may also feel palpitation when vigorous contraction and anomalous movement of the heart occur in the chest.

Etiology

The most common causes of palpitation are arrhythmia and anxiety **(Table 4)**.

Cardiac Cause

Cardiac causes of palpitation include mostly arrhythmic and occasional nonarrhythmic. In one series,[18] arrhythmic causes of palpitation include atrial fibrillation (AF) (10%), supraventricular tachycardia (SVT) (9.5%), ventricular

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TABLE 4: Etiological classification of palpitation.[19]

Etiology	*%*
Cardiac (arrhythmic and structural)	43
Psychosomatic	30.5
Systemic causes and drugs	10
Unknown	31

premature beat (VPB) (7.9%), atrial flutter (5.8%), premature atrial beats (3.2%), ventricular tachycardia (VT) (2.1%), mitral valve prolapse (MVP) (1.1%), sick sinus syndrome (1.1%), and pacemaker (1.1%). Nonarrhythmic causes of palpitation are aortic regurgitation, mitral regurgitation, MVP, hypertrophic cardiomyopathy, atrial myxoma, ASD, ventricular septal defect, etc.

Psychosomatic Cause

Psychosomatic cause is characterized by recurrent unexpected panic attacks, more common in relatively younger women somatizing more frequently. However, one should not overdiagnose anxiety and should do a total evaluation, as arrhythmia can coexist with anxiety. Anxiety causes a catecholamine surge and may induce VT.

Systemic Cause

Fever, anemia, hypoglycemia, pheochromocytoma, and hyperthyroidism are some common noncardiac causes of palpitation.

Drugs

Beta agonists, theophylline, ephedrine, cocaine, alcohol, smoking, and caffeine cause palpitation.

Approach

The patient should be asked to describe the symptom in every detail. Its duration, onset and termination, slow or rapid, regular or irregular, activity and posture, associated giddiness, syncope, angina, or dyspnea should be evaluated. The examiner himself can demonstrate, or the patient may be asked to tap out the rhythm.

Clinical Types

The European Heart Rhythm Association, in a position statement,[20] classified the clinical types of palpitation **(Box 2)**.

The patient described extrasystolic palpitation as irregular skipped or missed beat or sinking of the heart. Onset and termination are sudden.

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BOX 2: Clinical classification of palpitation.[20]

- Extrasystolic
- Tachycardiac
- Anxiety-related
- Pulsation

Tachycardiac palpitation is described as regular or irregular accelerated beating wings in the chest with a sudden onset and termination and precipitated by physical effort. Anxiety-related palpitation is associated with anxiety and agitation, slightly accelerated, gradual onset and termination, and precipitated by stress and anxiety. Pulsation palpitation, mostly related to aortic regurgitation, or other structural heart disease with volume overload is described as pounding, regular, gradual onset and termination, nonrapid, and persistent.

Predictors of Cardiac Etiology

- Male sex
- Description of irregular heartbeat
- History of heart disease
- Duration of event >5 minutes

Clinical Evaluation

Symptoms

A feeling of flip-flopping in the chest is suggestive of atrial or ventricular ectopic, whereas fluttering in the chest is suggestive of atrial or ventricular tachyarrhythmia.

Rate and rhythm: A rapid, irregular, chaotic feeling, particularly in the elderly, is suggestive of AF. A fast-regular rhythm is suggestive of supraventricular or VT. A pounding sensation in the neck during tachycardia is suggestive that the atrium is contracting against the closed tricuspid valve and reflux of blood in the superior vena cava. This may be regular in atrioventricular (AV) nodal re-entrant tachycardia (AVNRT) and known as the frog sign (equivalent to the cannon wave in JVP). In VT, the frog sign is intermittent and indicative of AV dissociation, which produces intermittent simultaneous ventricular and atrial depolarization. Neck pounding produces a shirt-flipping sign. Visible neck pulsation only, as opposed to frog sign, is suggestive of accessory pathway-mediated AV re-entry tachycardia.

In the presence of significant left ventricular dysfunction, a rapid ventricular rate may not be sensed as palpitation due to poor ventricular contractility. In patients with dilated or ischemic cardiomyopathy, VT produces palpitation only in 6–15% of patients, whereas 93–100% of patients present with palpitation in case of right ventricular dysplasia or idiopathic VT.[20]

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Onset and Termination

Very sudden onset and termination are suggestive of paroxysmal tachycardia, in contrast to sinus tachycardia, which occurs and terminates slowly. The caveats are SVT and may become faster during exercise, giving an impression of gradual onset, and may have sinus tachycardia following termination, giving the impression of gradual cessation.

Circumstances

Palpitation and syncope: Palpitation turns into an ominous symptom when palpitation is associated with dizziness, presyncope, or syncope. VT is a strong probability. SVT can occasionally cause syncope immediately after the onset. Rapid heart rate with low cardiac output and acute vasodilatation are the mechanisms of syncope.

Palpitation and dyspnea: These usually indicate arrhythmia associated with ventricular dysfunction, aortic stenosis, hypertrophic cardiomyopathy, mitral stenosis, and ischemic heart disease.

Palpitation and posture: AVNRT may be initiated by standing posture from bending over and may be terminated after lying down. A patient with a regurgitant lesion feels the pounding sensation more in the lying or left lateral decubitus.

Palpitation and catecholamine excess: Exercise-induced disproportionate rapid palpitation may be due to catecholamine-induced idiopathic VT, right ventricular outflow tract (RVOT) tachycardia, and AVNRT. Postexercise AF may occur due to increased vagal tone just after cessation of sympathetic tone. Mild exertion or emotional stress can cause palpitation in young female patients due to hypersensitivity of beta-adrenergic stimulation. It is called inappropriate sinus tachycardia.

Sleep: Palpitation during sleep is vagal-mediated AF or certain long-QT syndrome.

Polyuria: Any paroxysmal atrial arrhythmia, more commonly AF, induces polyuria due to oversecretion of natriuretic peptide from atrial stretching. It is not a very common (<5%) symptom.

CHEST PAIN

Chest pain is the most common symptom of the cardiovascular system. In the United States, 7 million patients attend the emergency department annually with chest pain. It is an important task for the clinician to establish whether the chest pain is cardiac or noncardiac. In an outpatient setup, <25% of patients with chest pain will have a cardiac cause, and in an emergency setup, around 67% of patients will have a cardiac cause of chest pain **(Table 5)**.

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Cardiac Chest Pain

Stable Angina

One should determine three factors:

1. Substernal chest pain
2. Provoked by effort or emotional stress
3. Relieved by rest/nitroglycerine within minutes

When all three characteristics are present, it is typical anginal chest pain. When two features are present, it is atypical angina. When only one feature is present, it is nonanginal chest pain. The pretest probability of an obstructed coronary artery disease in 15,815 symptomatic patients, according to age, sex, and nature of symptom, was studied in a pooled analysis **(Table 6)**.[23]

Acute Coronary Syndrome

In an acute chest pain presentation, 3% of patients diagnosed as having noncardiac chest pain may suffer acute coronary syndrome (ACS) or death within 1 month. Hence, a clinical acumen added by optimum laboratory tests

TABLE 5: Common causes of chest pain.[21,22]

	Percentage of patients		
Diagnosis	*Primary care: United States (%)*	*Primary care: Europe (%)*	*Emergency (%)*
Musculoskeletal condition	36	29	7
Gastrointestinal	19	10	3
Serious cardiac cause (ACS, CHF, and pulmonary embolism)	16	13	54
Stable CAD	10	8	13
Psychosomatic disease	8	17	9
Lung cause (pneumonia, pneumothorax, and lung cancer)	5	20	12
Nonspecific chest pain	16	11	5

(ACS: acute coronary syndrome; CAD: coronary artery disease; CHF: congestive heart failure)

TABLE 6: Pretest probability of coronary artery disease according to the type of angina.

	Typical angina		*Atypical angina*		*Nonanginal chest pain*	
Age (years)	*Male*	*Female*	*Male*	*Female*	*Male*	*Female*
30–39	3%	5%	4%	3%	1%	1%
40–49	22%	10%	10%	6%	3%	2%
50–59	32%	13%	17%	6%	11%	3%
60–69	44%	16%	26%	11%	22%	6%
70+	52%	27%	34%	19%	24%	10%

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can only help the emergency clinician to diagnose whether the chest pain is cardiac or noncardiac **(Table 7)**.

There are several bedside scoring systems, which help to diagnose ACS with an added ECG or ischemic-injury biomarkers **(Tables 8 and 9)**.

Pericardial Pain

A major part of the pericardium is pain-insensitive. Pain due to pericarditis is due to adjacent pleural involvement. Pain may radiate to the neck, shoulder, or back. Radiation to the trapezius ridge is a specific symptom of acute pericardial pain. Most commonly, it is retrosternal, aggravated by coughing, deep breathing, and changes in posture, all of which cause pleural movement. Sitting upright and leaning forward relieve pain.

TABLE 7: Predictivity of symptoms for acute ischemic pain.[24]

Predictive	*Nonpredictive*
Central, pressure-like, and prolonged	Pressure-like pain
Radiation to one or both hands	Stabbing, pleuritic, or positional
Similar type of pain during a prior presentation	Reproducible on palpation
Associated nausea and sweating	Response to nitrate

TABLE 8: The HEART score for ACS at the emergency department.

History	Highly suspicious	2
	Moderately suspicious	1
	Slightly suspicious	0
ECG	Significant ST-segment depression	2
	Nonspecific repolarization abnormality	1
	Normal	0
Age (years)	≥65	2
	45–65	1
	≤45	0
Risk factors	3 or more	2
	1–2	1
	No risk factor	0
Troponin	≥3 normal unit	2
	1–3	1
	Normal limit	0

Note: Low risk: 0–3 points; non-low risk: ≥4 points.
(ACS: acute coronary syndrome; HEART: History, Electrocardiogram, Age, Risk factors, Troponin)

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TABLE 9: Simplified EDACS score for ACS.

Age (years)	18–39	0
	40–49	1
	50–59	2
	60–69	3
	70–79	4
	80–89	5
	90+	6
Known coronary artery disease or ≥3 cardiac risk factors		1
Male sex		1
Symptoms: Pain radiating to arm, shoulder, neck, or jaw		1

Note: Low risk 0–3 points; non-low risk ≥ 4 points.
(ACS: acute coronary syndrome; EDACS: Emergency Department Assessment of Chest pain Score)

Acute Aortic Syndrome

At the very beginning, pain reaches at peak, front, and back of the chest and is described as ripping, tearing, and severe sharp pain. Sudden-onset pain is very characteristic. Chest pain is more common in type A dissection compared to type B dissection. There may be an absence of pulse in one or both arms, and the patient may present with a cerebrovascular accident or paraplegia or acute aortic regurgitation.

Pulmonary Embolism

Chest pain due to pulmonary embolism is due to dilatation of the pulmonary artery or pulmonary infarction, leading to pleural involvement. One can follow the Well model for clinical diagnosis **(Tables 10 and 11)**.

Chest Wall Pain

A history of osteoarthritis and rheumatoid arthritis and the finding of local tenderness increase the likelihood of chest wall pain. Costochondral and costosternal syndrome are the common causes of chest wall pain. Tietze's syndrome, tenderness, swelling, and redness of costochondritis are infrequent findings. Chest wall pain distributed along a dermatome can be due to cervical root disease, intercostal muscle cramp, and herpes zoster.

Gastrointestinal Diseases

Esophageal pain due to acid reflux is a retrosternal burning pain, precipitated by some specific foods, more on lying down, early morning, and relieved by acid-reducing agents. Chest pain due to esophageal spasm is compressive

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TABLE 10: Well model for PE.

Clinical findings		*Points*
Clinical features of DVT		3
Heart rate >100 beats/min		1.5
Immobilization		1.5
Previous objectively diagnosed DVT or PE		1.5
Hemoptysis		1
Malignancy		1
Total points	***Risk of PE***	***Probability of PE (%)***
<2	Low	1–28
2–6	Moderate	28–40
>6	High	38–91

(DVT: deep vein thrombosis; PE: pulmonary embolism)

TABLE 11: Revised Geneva score for pulmonary embolism.

Clinical findings		*Points*
65 years		1
Previous DVT or PE		3
Surgery under anesthesia or lower limb fracture in the past month		2
Active malignant condition		2
Unilateral lower limb pain		3
Hemoptysis		2
Total point	***Risk of PE***	***Probability of PE (%)***
≤3	Low	7–9
4–10	Intermediate	10–60
≥11	High	>60

(DVT: deep vein thrombosis; PE: pulmonary embolism)

in nature, may radiate down arms, and sometimes is relieved by antianginal medications. Pain due to peptic ulcer disease, gallbladder disease, and pancreatitis can radiate to the lower chest wall.

Clinical Approach

A thorough history and clinical examination can diagnose most of the chest pain causes:

- Mode of onset, duration, and frequency
- Site of pain and radiation

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- Character of pain
- Aggravating and relieving factors
- Associated symptoms

SYNCOPE

Transient loss of consciousness is defined by the triad of amnesia for the period of unconsciousness, loss of motor control, and unresponsiveness **(Box 3)**. Syncope is transient loss of consciousness due to cerebral hypoperfusion, characterized by the triad of rapid onset, short duration, and spontaneous complete recovery. Presyncope is defined as a prodrome simulating syncope without loss of consciousness.

There are several etiopathologies with different mechanisms for syncope **(Table 12)**. Cessation of cerebral perfusion due to hypotension is the final common pathway for syncope. A cessation for 6–8 seconds can lead to syncope.

A detailed history and clinical examination can differentiate syncope from other causes of transient loss of consciousness in 60% of cases.[25] History taking is the most important part in diagnosing the etiology for syncope. Eyewitness accounts are very important. Reflex syncope **(Flowchart 1)** is the most common cause of syncope in all ages, followed by cardiac syncope **(Flowchart 2)**. Orthostatic hypotension **(Flowchart 3)** is also a common etiopathology.

Carotid Sinus Massage

The carotid sinus massage (CSM) test is recommended in patients above 40 years of age. Ventricular pause >3 seconds and/or a fall of systolic blood

BOX 3: Transient loss of consciousness.

- Syncope
- Seizure
- *Others:* Psychogenic, subclavian steal syndrome, vertebrobasilar TIA, and breath-holding spell

(TIA: transient ischemic attack)

TABLE 12: Etiopathologies of syncope.

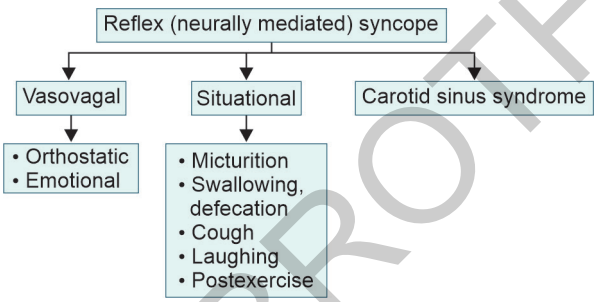
Syncope	*Conditions*
Reflex	• Vasovagal • Situational • Carotid sinus syncope • Low adenosine syncope
Cardiac	• Bradyarrhythmia • Tachyarrhythmia • Structural heart disease
Orthostatic hypotension	• Drug-induced • Autonomic failure

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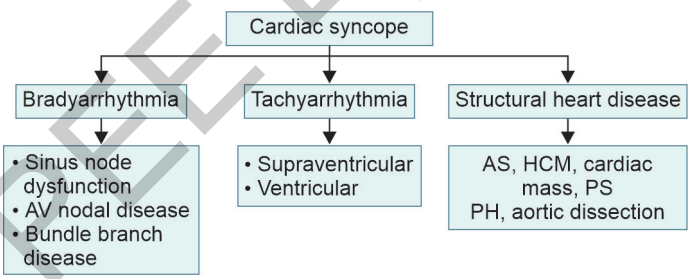
pressure >50 mm Hg is defined as carotid sinus hypersensitivity. To define carotid sinus syndrome, there must be syncope during CSM associated with ventricular pause, which is usually >3 seconds or asymptomatic pause >6 seconds. This response is a cardioinhibitory response. Only the fall of systolic pressure >50 mm Hg is a vasodepressor response. The complication of CSM is stroke or TIA in 0.24% of cases.[26]

CSM technique:[27] Head is turned contralaterally and posteriorly. Fingers are placed on the ipsilateral carotid artery at its maximum pulsation. Massage is done firmly up and down on the long axis of the artery for 10 seconds. Heart rate and blood pressure are monitored and recorded closely. After stabilization

Flowchart 1: Reflex syncope.

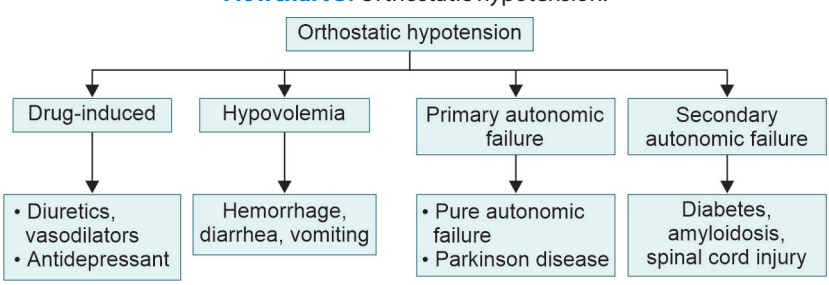


Flowchart 2: Cardiac syncope.



(AS: aortic stenosis; AV: atrioventricular; HCM: hypertrophic cardiomyopathy; PH: pulmonary hypertension; PS: pulmonary stenosis)

Flowchart 3: Orthostatic hypotension.



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of heart rate and blood pressure to baseline, CSM is done on the other side and then, in sequence, in an upright posture. CSM is contraindicated in case of TIA or stroke or acute myocardial infarction in the past 3 months. High-grade carotid artery stenosis is also a contraindication.

CONCLUSION

William Heberden in 1768 described cardiac chest pain as, "Those who are afflicted with it are seized, while they are walking, and more particularly when they walk soon after eating, with a painful and most disagreeable sensation in the breast, which seems as if it would take their life away, if it were to increase or to continue: The moment they stand still, all this uneasiness vanishes." After two and a half centuries, when coronary imaging is at its peak, effort angina is still a clinical diagnosis. Similarly, dyspnea, palpitation, and syncope help to initiate the diagnostic cascade in major cardiac diseases.

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