HARRISON

EDITION 22nd

CARDIOLOGY

PART 1

Cardiology

Approach To The Patient With Possible Cardiovascular Disease	Completed
Basic Biology Of The Cardiovascular System	Completed
Epidemiology Of Cardiovascular Disease	Completed
Physical Examination Of The Cardiovascular System Part 1	Completed
Physical Examination Of The Cardiovascular System Part 2	Completed
Physical Examination Of The Cardiovascular System Part 3	Completed
Chapter 247 - Electrocardiography	Completed
Chapter - 248 Noninvasive Cardiac Imaging Echocardiography, Nuclear Cardiology, And Magnetic Resonance Computed Tomography Imaging Part - 1	Completed
Chapter - 248 Noninvasive Cardiac Imaging Echocardiography, Nuclear Cardiology, And Magnetic Resonance Computed Tomography Imaging Part - 2	Completed
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Chapter - 250 Principles Of Clinical Cardiac Electrophysiology	Completed
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Cardiology

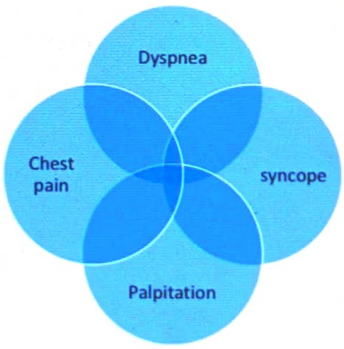
Chapter 252 - The Bradyarrhythmias Disorders Of The Atrioventricular Node Part 1	Completed
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Chapter 253 - Approach To Supraventricular Arrhythmias	Completed
Chapter 254 - Physiologic And Nonphysiologic Sinus Rhythm	Completed
Chapter 255 - Focal Atrial Tachycardia	Completed
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Chapter 257 - Common Atrial Flutter And Macroreentrant And Multifocal Atrial Tachycardias	Completed
Chapter 258 - Atrial Fibrillation	Completed
Chapter 259-263 Ventricular Arrhythmias	Completed
Chapter 264 Heart Failure Pathophysiology And Diagnosis	Completed
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Chapter 266 - Classification Of Cardiomyopathy	Completed
Chapter 267 - Genetic Cardiomyopathies	Completed

HARRISON 22nd EDITION BASED CARDIOLOGY
SECTION 1: Introduction to CV system

243. Approach to the Patient with Possible Cardiovascular Disease

THE CARDINAL SYMPTOMS

- Dyspnoea
- Palpitation
- Angina
- Fatigue
- Syncope



DYSPNOEA

- *Abnormally uncomfortable awareness of breathing*
- Conscious sensation that results from an unusual perception of discomfort during breathing.

- **Grading**
- **Mechanism**
- **Timing**
- **Onset/offset**
- **Heart vs Lung**
- **Localizing value**
- **Systemic (Anemia)**

A 72-year-old man with a history of myocardial infarction and congestive heart failure is comfortable at rest. While ordinary physical activity causes dyspnoea, walking to his car makes him develops chest pain. He must rest for several minutes before these symptoms resolve. His New York Heart Association classification is which of the following?

a. Class I

b. Class II

c. Class III

d. Class IV

Symptoms

S

D

P

A

NYHA III → III B

TABLE 243-1 New York Heart Association Functional Classification

Class I	**Class III**
No limitation of physical activity	Marked limitation of physical activity
No symptoms with ordinary exertion	Less than ordinary activity causes symptoms
Class II	Asymptomatic at rest
Slight limitation of physical activity	**Class IV**
Ordinary activity causes symptoms	Inability to carry out any physical activity without discomfort
	Symptoms at rest

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Answer: C

NYHA FUNCTIONAL CLASS

CLASS	NYHA functional class
NYHA I	Patients with cardiac disease but without resulting limitations of physical activity Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain.
NYHA II	Patients with cardiac disease resulting in slight limitation of physical activity They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or angina pain.
NYHA III	Patients with cardiac disease resulting in marked limitation of physical activity They are comfortable at rest. **Less than ordinary physical activity causes fatigue, palpitation, dyspnea, or anginal pain.**
NYHA IV	Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort Symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.

Functional assessment of children with heart failure is done using

a. NYHA classification

b. WHO classification

c. Ross classification

d. MRC classification

Dexmedetomidine?

NYHA III → IV

NYHA IIIB

Answer: C

INISS September 2023 recall

NYHA and Modified Ross Heart Failure Classification for Children

	NYHA	Ross
Class I	No limitations of physical activity	No limitations or symptoms
Class II	May experience fatigue, palpitations, dyspnea, or angina during moderate exercise but not during rest	Infants: Mild tachypnea or diaphoresis with feeding Older children: Mild to moderate dyspnea on exertion
Class III	Symptoms with minimal exertion that interfere with normal daily activity	Infants: Growth failure and marked tachypnea or diaphoresis with feeding Older children: Marked dyspnea on exertion
Class IV	Unable to carry out any physical activity because they typically have symptoms of HF at rest that worsen with any exertion	Symptoms at rest such as tachypnea, retractions, grunting, or diaphoresis

HF: Heart failure; NYHA: New York Heart Association.

THE MODIFIED MEDICAL RESEARCH COUNCIL DYSPNEA SCALE

TABLE 39-1 An Example of a Clinical Method for Rating Dyspnea: The Modified Medical Research Council Dyspnea Scale[a]

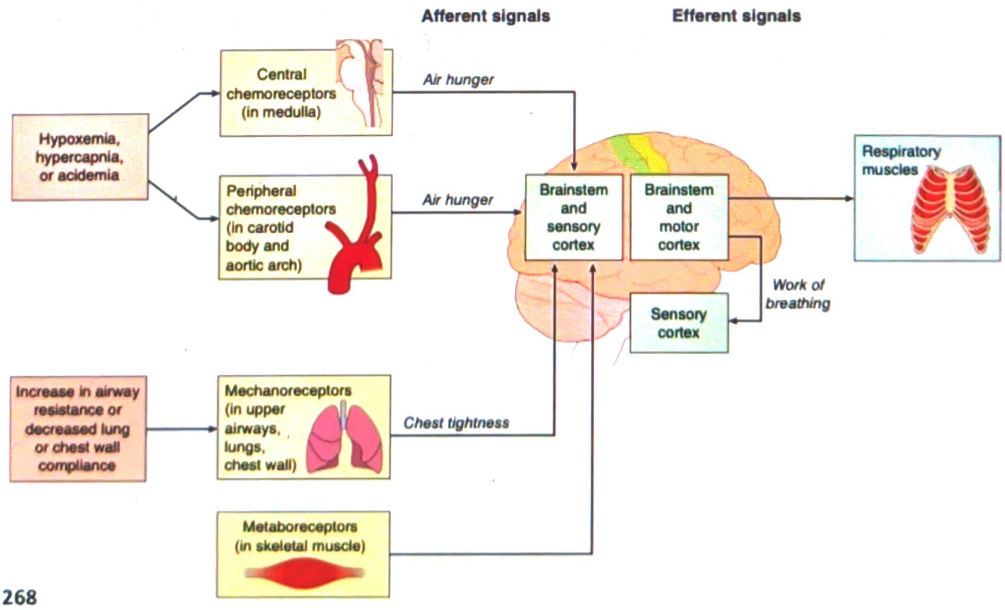
GRADE OF DYSPNEA	DESCRIPTION
0	Not troubled by breathlessness, except with strenuous exercise
1	Shortness of breath walking on level ground or with walking up a slight hill
2	Walks slower than people of similar age on level ground due to breathlessness, or has to stop to rest when walking at own pace on level ground
3	Stops to rest after walking 100 m or after walking a few minutes on level ground
4	Too breathless to leave the house, or breathless with activities of daily living (e.g., dressing/undressing)

[a]This scale has been integrated into the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines clinical classification scheme.

Source: Reproduced with permission from DA Mahler, CK Wells: Evaluation of clinical methods for rating dyspnea. Chest 93:580, 1988.

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POTENTIAL SIGNALING PATHWAYS UNDERLYING THE SENSATION OF DYSPNEA



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GENERAL MECHANISMS OF DYSPNEA

- Decreased Lung compliance
- Decreased air volume
- Increased Airway resistance
- Respiratory muscle fatigue

PULMONARY CAUSE VERSUS CARDIAC CAUSE

Pulmonary cause

- Cough with expectorant
- Wheezing
- Fever
- Weight loss
- Pleuritic type of pain
- Seasonal variation
- Responds to bronchodilator

Cardiac cause

- PND and orthopnea
- Asso syncope/palpitation/angina
- Rapid progression of symptoms
- Little cyanosis with severe dyspnea
- Response to diuretics
- Cheyne-stokes breathing

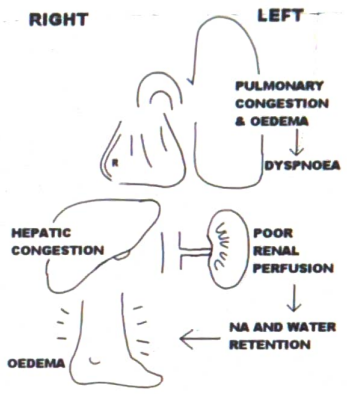
RIGHT HEART VERSUS LEFT HEART FAILURE

Right heart

- Peripheral edema
- Hepatic congestion
- Raised JVP

Left heart

- Pulmonary congestion
- Edema
- Breathlessness



Biventricular failure?

DYSPNOEA IN LEFT HEART DISEASE:

- Orthopnea
- PND
- Systolic dyfunction
- Diastolic dysfunction

Other forms of dyspnoea

- Platypnea → LYING DOWN
- Trepopnea → LATERAL DECUBITUS
- Bendopnoea → BENDING FORWARD
- Blockopnea →
- Platypnoea/orthodeoxia → LYING DOWN + DESATURATION

Dyspnoea equivalent

- Cough
- Extreme fatigue
- Other signs of hypoxia
- Violent behavior , ACS delirium

ANGINA EQUIVALENT

1) Dyspnoea
2) Jaw pain
3) Radiation of pain

NYHA - All Heart failure.

WHICH OF THE FOLLOWING IS NOT A COMPONENT OF WHO FUNCTIONAL CLASS FOR PAH?

a. Fatigue

b. Dyspnea

c. Palpitation

d. Presyncope

NYHA = ↓ CO ⓐ A ↓ Dyspnea

WHO FC = PD Syncope (presyncope) WHO FC

ANSWER: C

WHO FUNCTIONAL CLASS FOR PAH

Syncope in PAH
1. ↓ TE
2. Arrhythmia's
3. RV Ischemia/↓ C.O
4. MPA → LVCA ↓ Syncope.

Class	Description
I	Patients with PAH where there is no limitation of usual physical activity, ordinary physical activity does not cause increased dyspnea, fatigue, chest pain, or presyncope
II	Patients with PAH who have mild limitation of physical activity. There is no discomfort at rest, but normal physical activity causes increased dyspnea, fatigue, chest pain, or presyncope
III	Patients with PAH who have marked limitation of physical activity. There is no discomfort at rest, but less than normal physical activity causes increased dyspnea, fatigue, chest pain, or presyncope
IV	Patients with PAH who are unable to perform any physical activity at rest and who may have signs of right ventricular failure. Dyspnea and/or fatigue may be present at rest, and symptoms are increased by almost any physical activity

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Bendopnea :-

A 62-year-old man with a history of ischemic cardiomyopathy presents with progressive dyspnea and fatigue. During examination, he reports the onset of shortness of breath within 30 seconds of bending forward to tie his shoes. His heart rate is 92 bpm, blood pressure is 110/70 mmHg, and jugular venous pressure is elevated. Which of the following is the most accurate interpretation of this finding?

a. It suggests reduced probability of elevated left heart filling pressure (LR <0.3).

b. It is a sign of increased probability of elevated left heart filling pressure (LR >3).

c. It is diagnostically unhelpful (LR between 0.3 and 3).

d. It rules out heart failure with preserved ejection fraction.

AJR = ABSENT
FALSE NEG = IVC D/Bcs

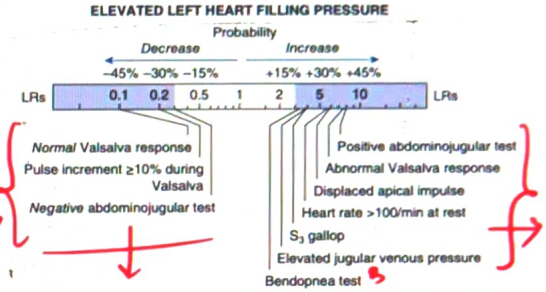


FIGURE 8-1 With just a glance, the clinician can immediately identify those few findings that significantly increase probability of heart failure (*right* side of ruler, likelihood ratio [LR] = 3 or more) and those that significantly decrease it (*left* side of ruler, LR = 0.3 or less). For example, the figure shows that the *presence* of S_3 gallop (LR = 3.9) and *presence* of displaced apical impulse (LR = 5.8) significantly increase probability of heart failure. On the other hand, the *absence* of either finding does not appear on the ruler because these LRs lie between 0.3 and 3.0 (Table 8-3) and therefore are diagnostically unhelpful. *(Reproduced with permission from SR McGee: Evidence-Based Physical Diagnosis, 4th ed. Philadelphia, Elsevier, 2022.)*

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Answer: B

Bendopnea is characterized by all except :

a. Increased PCWP (wedge ↑)

b. Decreased cardiac index

c. Raised JVP (↑)

d. Increased abdominal girth

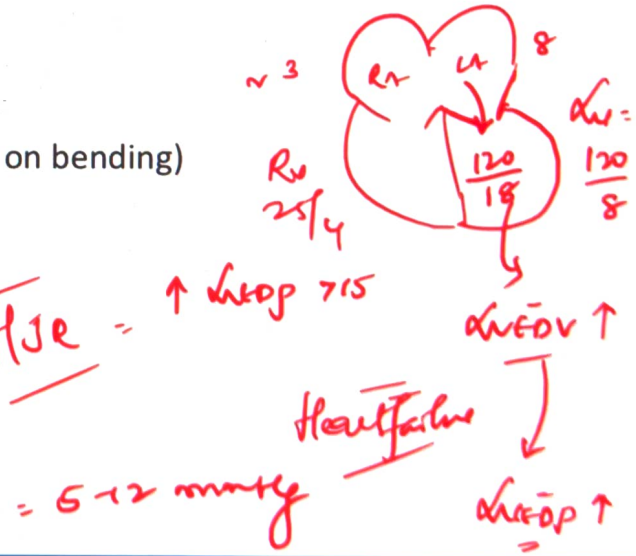
Answer: D

BENDOPNEA

- Shortness of breath, specifically when bending forward, such as when putting on their shoes or socks
- **Mechanism:**
  - During bending, Increased intrathoracic pressure leads to further increase in ventricular filling pressures and subjects with bendopnea (who start with higher filling pressures) are more likely to reach a threshold pressure necessary to induce shortness of breath.
- It is likely caused by elevation of left sided filling pressure or PCWP as has been demonstrated with shortness of breath in patients with heart failure.

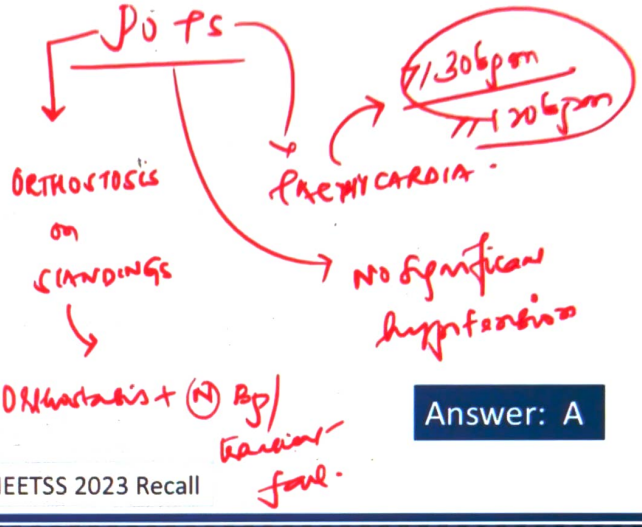
HEMODYNAMICS IN BENDOPNEA

1. Increased intrathoracic pressures
2. Elevated filling pressures (RV and LV on bending)
3. Elevated PCWP
4. Reduced Cardiac Index
5. Raised JVP



A 30-year-old generally healthy female presents with ongoing fatigue, mental "clouding," and sleep disturbances. She also reports experiencing her heart racing on standing with associated dizziness. She does not take any medications or illicit substances. You make a diagnosis of postural orthostatic tachycardia syndrome. What is the heart rate cut off for the same?

a. Rise in HR by > 30 beats per minute
b. Rise in HR by > 20 beats per minute
c. Rise in HR by > 15 beats per minute
d. Rise in HR by > 10 beats per minute



Answer: A

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SBP 20/10
DBP
Either of them
3 mins.

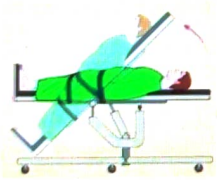
ORTHOSTATIC HYPOTENSION

- Orthostatic hypotension (OH) is defined as a decrease of more than 20 mm Hg in systolic pressure or a decrease of more than 10 mm Hg in diastolic pressure after rising to a standing position from a supine position
- BP and heart rate should be measured after symptoms develop or once 3 minutes has passed after rising to the standing position
- OH is an inability to maintain sufficient BP and adequate cerebral perfusion against gravity

POSTURAL TACHYCARDIA SYNDROME

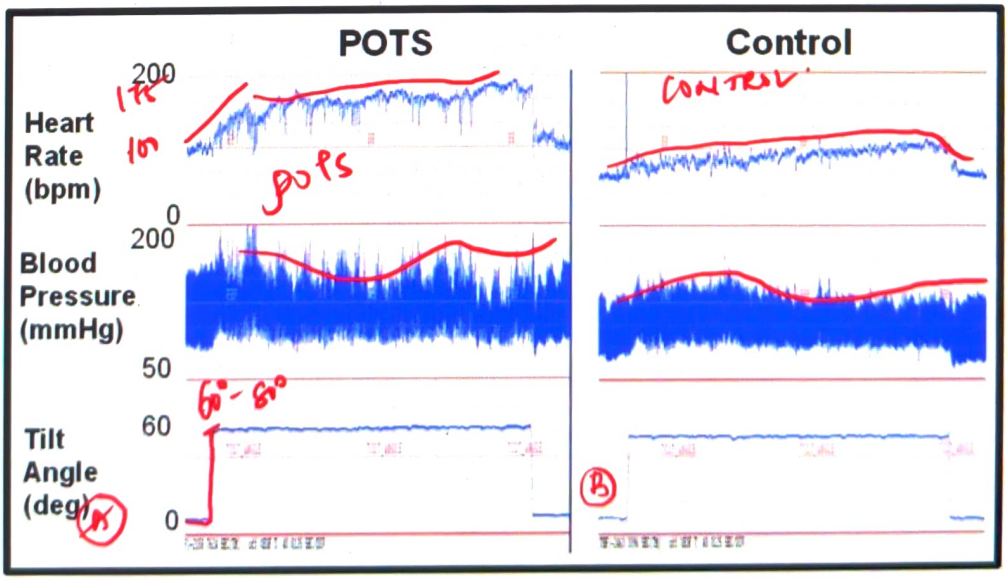
- Characterized by orthostatic symptoms, tachycardia, and the absence of significant hypotension
- Major mechanisms are denervation (neuropathic POTS), deconditioning, and a hyperadrenergic state NE > 600pg/mL.
- **Diagnostic criteria**
  - –Orthostatic tachycardia greater than 30 beats/min, usually to a rate of 120 beats/min or higher
  - –Transient decrease in SBP of more than 20 mm Hg, with recovery within first minute of tilt
  - –Standing plasma norepinephrine level higher than 600 pg/mL
  - –Severe orthostatic symptoms

Females (5: 1)
20-40 years



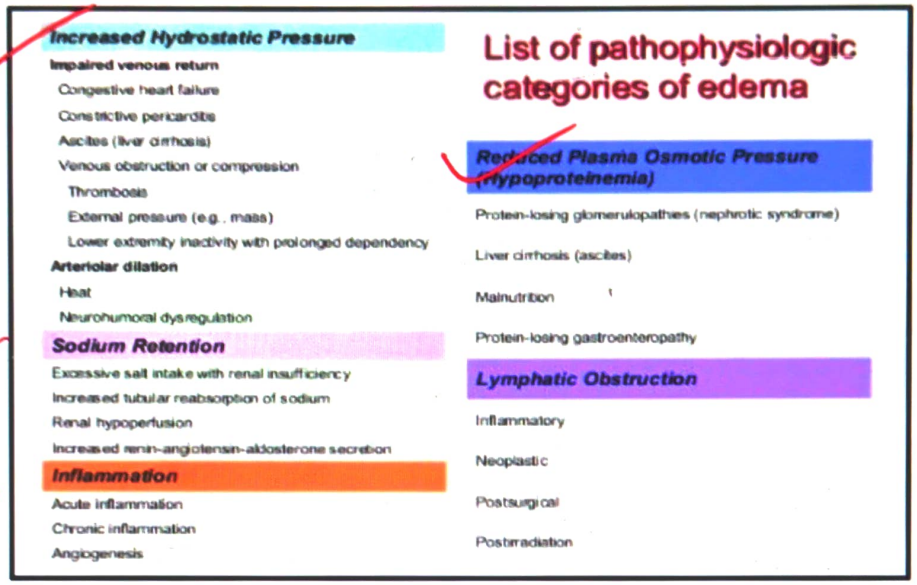
POSTURAL TACHYCARDIA SYNDROME

TILT TABLE TEST



Excessive increase in HR both initially and throughout the tilt

PATHOLOGICAL EDEMA



Increased Hydrostatic Pressure

Impaired venous return
- Congestive heart failure
- Constrictive pericarditis
- Ascites (liver cirrhosis)
- Venous obstruction or compression
  - Thrombosis
  - External pressure (e.g., mass)
- Lower extremity inactivity with prolonged dependency

Arteriolar dilation
- Heat
- Neurohumoral dysregulation

Sodium Retention
- Excessive salt intake with renal insufficiency
- Increased tubular reabsorption of sodium
- Renal hypoperfusion
- Increased renin-angiotensin-aldosterone secretion

Inflammation
- Acute inflammation
- Chronic inflammation
- Angiogenesis

List of pathophysiologic categories of edema

Reduced Plasma Osmotic Pressure (Hypoproteinemia)
- Protein-losing glomerulopathies (nephrotic syndrome)
- Liver cirrhosis (ascites)
- Malnutrition
- Protein-losing gastroenteropathy

Lymphatic Obstruction
- Inflammatory
- Neoplastic
- Postsurgical
- Postirradiation

HIGH SAAG ASCITES IS A FEATURE OF ALL OF THE FOLLOWING EXCEPT?

a. Nephrotic syndrome

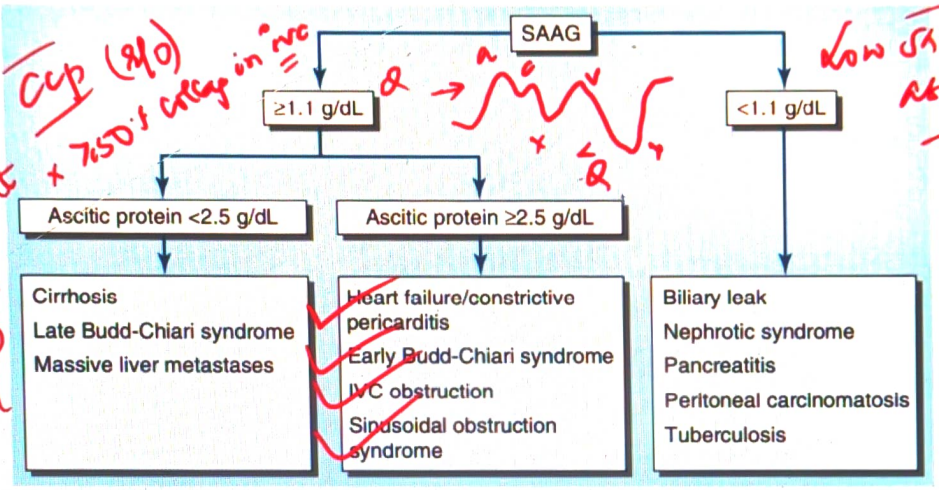
b. IVC Obstruction

c. Congestive heart failure

d. Constrictive pericarditis

ANSWER: A

ALGORITHM FOR THE DIAGNOSIS OF ASCITES ACCORDING TO THE SERUM-ASCITES ALBUMIN GRADIENT (SAAG)



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ANTIHYPERTENSIVES ASSOCIATED WITH PEDAL EDEMA INCLUDE ALL EXCEPT ?

a. Minoxidil

b. Hydralazine

c. Clonidine

d. Enalapril

ANSWER: D

INITIATION OF MEDICATIONS THAT WORSEN THE SYMPTOMS OF HF/CAUSE PEDAL EDEMA

- Calcium antagonists (verapamil, diltiazem)
- Beta blockers (Cardioselective)
- Nonsteroidal anti-inflammatory drugs (Not aspirin)
- Thiazolidinediones (Pioglitazone and rosiglatzone)
- Antiarrhythmic agents (all class I agents, sotalol [class III])
- Anti-TNF antibodies
- Minoxidil
- Trastuzumab

TABLE 43-2 Drugs Associated with Edema Formation

- Nonsteroidal anti-inflammatory drugs
- Antihypertensive agents
  - Direct arterial/arteriolar vasodilators
    - Hydralazine
    - Clonidine
    - Methyldopa
    - Guanethidine
    - Minoxidil
  - Calcium channel antagonists → AMLODIPINE
  - α-Adrenergic antagonists
  - Thiazolidinediones
- Steroid hormones
  - Glucocorticoids
  - Anabolic steroids
  - Estrogens
  - Progestins
- Cyclosporine
- Growth hormone
- Immunotherapies
  - Interleukin 2
  - OKT3 monoclonal antibody

Source: Reproduced with permission from GM Chertow, in E Braunwald, L Goldman (eds): Approach to the patient with edema, in Primary Cardiology, 2nd ed. Philadelphia, Saunders, 2003.

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PRINCIPAL CAUSES OF GENERALIZED EDEMA

TABLE 43-1 Principal Causes of Generalized Edema: History, Physical Examination, and Laboratory Findings

ORGAN SYSTEM	HISTORY	PHYSICAL EXAMINATION	LABORATORY FINDINGS
Cardiac	Dyspnea with exertion prominent—often associated with orthopnea—or paroxysmal nocturnal dyspnea	Elevated jugular venous pressure, ventricular (S_3) gallop; occasionally with displaced or dyskinetic apical impulse; peripheral cyanosis, cool extremities, low pulse pressure when severe	Elevated urea nitrogen-to-creatinine ratio common; serum sodium often decreased; elevated natriuretic peptides
Hepatic	Dyspnea uncommon, except if associated with significant degree of ascites; most often a history of ethanol abuse	Frequently associated with ascites; jugular venous pressure normal or low; blood pressure lower than in renal or cardiac disease; one or more additional signs of chronic liver disease (jaundice, palmar erythema, Dupuytren's contracture, spider angiomata, male gynecomastia; asterixis and other signs of encephalopathy) may be present	If severe, reductions in serum albumin, cholesterol, other hepatic proteins (transferrin, fibrinogen); liver enzymes elevated, depending on the cause and acuity of liver injury; tendency toward hypokalemia, respiratory alkalosis; macrocytosis from folate deficiency
Renal (CRF)	Usually chronic: may be associated with uremic signs and symptoms, including decreased appetite, altered (metallic or fishy) taste, altered sleep pattern, difficulty concentrating, restless legs, or myoclonus; dyspnea can be present, but generally less prominent than in heart failure	Elevated blood pressure; hypertensive retinopathy; uremic fetor; pericardial friction rub in advanced cases with uremia	Elevation of serum creatinine and cystatin C; albuminuria; hyperkalemia, metabolic acidosis, hyperphosphatemia, hypocalcemia, anemia (usually normocytic)
Renal (NS)	Childhood diabetes mellitus; plasma cell dyscrasias	Periorbital edema; hypertension	Proteinuria (≥3.5 g/d); hypoalbuminemia; hypercholesterolemia; microscopic hematuria

Abbreviations: CRF, chronic renal failure; NS, nephrotic syndrome.

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Hypoxia induced vasoconstriction in pulmonary vasculature is mediated by

a. Opening of KATP channels
b. Inhibition of K+ channels
c. Adenosine receptors
d. Inhibition of voltage-gated Ca2+ channels

Answer: B

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DIFFERENTIAL EFFECTS OF HYPOXIA ON PULMONARY AND SYSTEMIC VASCULATURE

- During hypoxia, systemic arterioles dilate, at least in part, by opening of KATP channels in vascular smooth-muscle cells due to the hypoxia-induced reduction in ATP concentration.
- By contrast, in pulmonary vascular smooth-muscle cells, inhibition of K+ channels causes depolarization which, in turn, activates voltage-gated Ca2+ channels raising the cytosolic [Ca2+] and causing smooth-muscle cell contraction.
- Hypoxia-induced pulmonary arterial constriction shunts blood away from poorly ventilated portions toward better ventilated portions of the lung; however, it also increases pulmonary vascular resistance and right ventricular afterload.

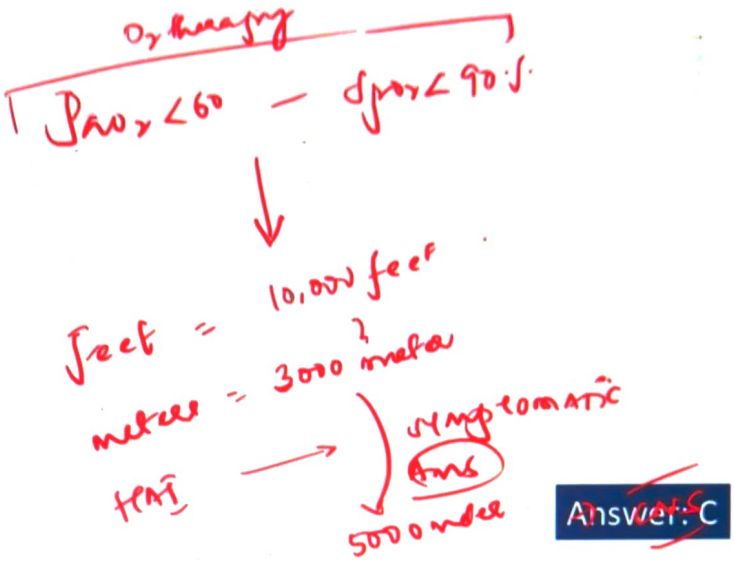
Ascent to what altitude results in reduction in Pao2 levels to 60 mm Hg or less?

a. 1000 meters

b. 2000 meters

c. 3000 meters

d. 7000 feet



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HYPOXIA SECONDARY TO HIGH ALTITUDE

- As one ascends rapidly to 3000 m (~10,000 ft), the reduction of the O2 content of inspired air (Fio2) leads to a decrease in alveolar Po2 to ~60 mmHg, and a condition termed high-altitude illness develops.
- At higher altitudes, arterial saturation declines rapidly and symptoms become more serious; and at 5000 m, unacclimated individuals usually cease to be able to function normally owing to the changes in CNS function described above

B. In persons with chronic hypoxemia secondary to prolonged residence at a high altitude (>13,000 ft, 4200 m), a condition termed chronic mountain sickness develops.

PHYSIOLOGICAL RESPONSE TO HYPOXIA

1. Erythrocytosis
2. Hyperventilation
3. Systemic arterial vasodilation
4. Pulmonary vasoconstriction
5. Rise in cardiac output

Cyanosis usually becomes manifest in an ascent to an altitude of 4000 m (13,000 ft)

CAUSES OF CYANOSIS?

Central Cyanosis
Decreased arterial oxygen saturation
Decreased atmospheric pressure—high altitude
Impaired pulmonary function
Alveolar hypoventilation
Inhomogeneity in pulmonary ventilation and perfusion (perfusion of hypoventilated alveoli)
Impaired oxygen diffusion
Anatomic shunts
Certain types of congenital heart disease
Pulmonary arteriovenous fistulas
Multiple small intrapulmonary shunts
Hemoglobin with low affinity for oxygen
Hemoglobin abnormalities
Methemoglobinemia—hereditary, acquired
Sulfhemoglobinemia—acquired
Carboxyhemoglobinemia (not true cyanosis)
Peripheral Cyanosis
Reduced cardiac output
Cold exposure
Redistribution of blood flow from extremities
Arterial obstruction
Venous obstruction

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A 23-year old female presents to emergency after her friends notice bluish discoloration of her lips. On examination, she has normal S1, S2 split and no audible murmurs or clubbing. Her saturation is 88% on room air. Her ECG and Echo are unremarkable, What is the likely cause for her symptoms?

a. Excess nitrates in water

b. Lead poisoning

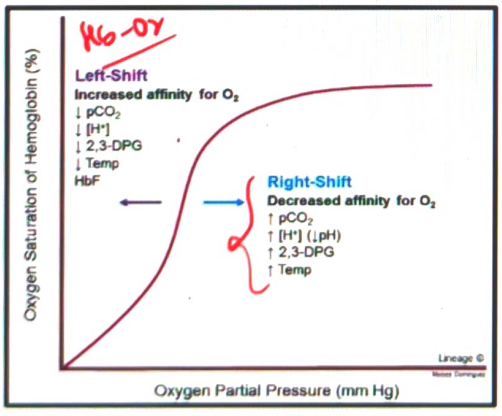
c. CO poisoning

d. Anemia

NEETSS 2022 Recall

Answer: A

OXY-HAEMOGLOBIN DISSOCIATION CURVE



Cyanosis: Cut off's

O_2 saturation	< 85%
Reduced Hb	> 4 gm/100 ml
Methaemoglobin	> 1.5 gm/100 ml
Sulphaemoglobin	> 0.5 gm/100 ml
R-L shunt	> 25% of LV output.