

Pulmonary Hypertension

The questions signed by * are hard: read their explanation. ● Past Years ● TBL ● Others

This file belongs to the PULSE Cardiology question book: the explanations of the multiple choice answers and the model answers of the essay questions, in the words of the handout.

Answer Key | Multiple Choice Questions

Question no.	1	2	3	4	5	6
Answer	d	a	a	c	c	c

► Second | Questions with a Note

Question no.	1
Answer	b

► Why this answer?

- What is the most probable group of pulmonary hypertension in a patient with history of recurrent pulmonary...

(d) Group 4

Recurrent pulm. emboli cause obstructive (obliterative) pulmonary hypertension by obstruction of the pulm. arterioles. Chronic thromboembolic pulmonary hypertension is WHO group 4.
- Which of the following drugs could decrease pulmonary artery pressure in patients with idiopathic pulmonary...

(a) Bosentan

Bosentan is an endothelin antagonist; blocking endothelin dilates the pulmonary arteries and lowers pulmonary artery pressure. ACE inhibitors are usually of limited effect on pulm. pressure; trimetazidine is an anti-anginal drug and bisoprolol a beta blocker.
- The following drug could decrease pulmonary artery pressure in patients with idiopathic pulmonary...

(a) Sildenafil

For reduction of pulm. pressure, direct vasodilators, Ca-channel blockers and ACE inhibitors are usually of limited effect; recently, sildenafil can be used.
- The following drug should be avoided in patients with pulmonary hypertension who are non-responders to...

(c) Verapamil

Ca-channel blockers are usually of limited effect in reduction of pulm. pressure; they help only responders to vasoreactivity testing, and in non-responders verapamil lowers cardiac output. Sildenafil, bosentan and iloprost are pulmonary vasodilators.
- Which of the following statements is correct regarding pulmonary hypertension?

(c) Dyspnea on more than ordinary effort in a pulmonary...

WHO functional class I means no limitation of physical activity, so dyspnoea only on more than ordinary effort is class I. (a) is wrong because the definition uses resting pressure, mean pulm. pressure above 20 mmHg; congenital shunts are group I, and a positive vasoreactivity response needs unchanged or increased cardiac output.

6 Giant a-wave in neck vein is seen in:

(c) Pulmonary hypertension

Giant A wave is one of the general signs of pulmonary hypertension. In atrial fibrillation there is no atrial contraction, so there is no a wave.

► **Second | Questions with a Note: why this answer?**

1 What is the WHO classification of pulmonary hypertension on top of severe mitral stenosis?

(b) Type II

Severe mitral stenosis causes passive pulmonary hypertension due to pulm. congestion, as in all conditions causing left sided H.F.. In the WHO grouping, pulmonary hypertension due to left heart disease is group II; the handout's numbered list of causes is a different list.

Model Answers | Essay Questions

1 **Mention pathophysiology of pulmonary hypertension.**

Etiology

① **Passive pulm. HPN:**

- It is due to pulm. congestion (pulm. artery pressure rises to a degree equal to rise in pulm. venous pressure).
- It occurs in all conditions causing left sided H.F.

② **Hyperdynamic pulm. HPN:**

- It is due to increased pulm. arterial blood flow.
- It occurs in congenital heart disease with left to right shunt e.g. ASD, VSD & PDA.

③ **Vasoconstrictive pulm. HPN:**

- It is due to pulmonary arteriolar vasoconstriction.
- It occurs in:
 - Long standing passive pulm. HPN.
 - Long standing hyperdynamic pulm. HPN.
 - Hypoxia: in all conditions causing central cyanosis especially: respiratory failure e.g. COPD - ILD; pulm. A-V fistula; high altitudes.

④ **Obstructive (obliterative) pulm. HPN:**

- It is due to obstruction of pulm. arterioles or capillaries.
- It occurs in:
 - Long standing vasoconstrictive pulm. HPN.
 - Disease of the pulm. arteries e.g. recurrent pulm. emboli; pulm. bilharziasis and congenital syphilis, Ayerza's disease; pulm. vasculitis e.g. SLE, scleroderma; pulm. thrombosis in hypercoagulable states; SCA.

⑤ **Acute obstructive pulm. HPN:**

- It is due to acute obstruction of pulm. arterial blood flow.
- Occurs in: massive pulm. embolism; massive lung collapse; tension or bilateral pneumothorax.

⑥ **Idiopathic (primary) pulm. HPN:**

- Occurs more commonly in middle-aged females with repeated pregnancies.
- The aetiology is unknown, but may be due to: recurrent thrombo-embolism; pulm. arteritis; thrombosis of pulm. arteries due to increased blood coagulability.

Note The handout has no heading "pathophysiology"; the mechanisms are given under its Etiology.

2 Define the following: pathophysiology of pulmonary hypertension & clinical picture (symptoms & signs) of pericardial effusion.

(a) Pathophysiology of pulmonary hypertension

① Passive pulm. HPN:

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- It occurs in all conditions causing left sided H.F.

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- It is due to increased pulm. arterial blood flow.
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(b) Clinical picture of pericardial effusion

Pericardial effusion: 1. Symptoms

- * Symptoms of systemic congestion.
- * Symptoms of low COP.
- * **Precordial pain:** dull aching pain due to stretch of the parietal pericardium and may be referred to the shoulders.
- * **Pressure symptoms:** may occur with massive effusion.
 - Dyspnea improved by sitting and leaning forwards [Mohammedan prayer's position].
 - Cough and dysphagia (rare).
- * Symptoms of the cause e.g. T.B. toxemia.

Pericardial effusion: 2. General signs (signs of cardiac tamponade)

- * Decubitus: may be Mohammedan prayer's position.
- * Signs of low COP.

* **Pulsus paradoxus:**

- It is decreased pulse volume during inspiration [due to decrease systolic B.L. Pr. **> 10 mmHg**].
- It can be detected by palpation (esp. of the femoral or carotid artery), or by using the sphygmomanometer.
- Normally, during inspiration the lungs expand and accommodate more amount of blood. This amount is compensated by increased venous return to the right side, so venous return to the left side of the heart remains constant "nearly constant".
- Pulsus paradoxus is due to decrease of venous return to the left side of the heart because the expansion in the lungs during inspiration isn't compensated by increase of the right ventricular stroke volume.

* **Signs of systemic venous congestion:**

- Neck veins:
 - Congested pulsating neck veins.
 - May be severely congested with minimal pulsations giving a false impression of non-pulsating veins.
 - **Kussmaul's sign** [inspiratory filling]: due to failure of the right side of the heart to accept the increased venous return during inspiration with accumulation of blood in the veins.
 - **Friedreich's sign** [diastolic collapse]: deep Y descent due to rapid emptying of the congested neck veins in a short time after opening of the tricuspid valve [less marked than in constrictive pericarditis]; N.B. y descent may be blunted in tamponade.
 - **Gibson's sign**: deep X descent.
- Enlarged tender liver.
- Ascites which may precede oedema of LL (ascites praecox).

* Signs of the cause.

Pericardial effusion: 3. Cardiac signs

Examination	Findings
Inspection and palpation	The apical pulsations are weak or absent; there may be precordial bulge in children.
Percussion	Dullness outside the apex; shifting dullness over the pulmonary area; increased size of the bare area with stony dullness; dullness to the right border of the sternum; Ewart's sign : dullness over the left subscapular region due to compression of the base of the left lung by pericardial fluid.
Auscultation	Weak distant heart sounds.

Note The handout has no heading "pathophysiology"; part (a) is its Etiology of pulmonary hypertension.