

Cardiac Symptomatology

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	c	e	a	c	c	c

► Second | Questions with a Note

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

► Why this answer?

- Which of the following is not a cause of unilateral lower limb oedema?
(c) Liver cell failure.
Liver cell failure is a cause of generalized oedema, so it does not give oedema of one limb. Venous obstruction, lymphatic obstruction and congenital causes are listed under local oedema.
- Differential diagnosis of acute dyspnea with clear back doesn't include the following:
(e) Acute left ventricular failure
Acute left ventricular failure gives acute pulmonary oedema with bilateral crepitations, at first basal then generalized, so the back is not clear. Pulmonary embolism is the cause whose clue is tachycardia, desaturation and clear back.
- Haemoptysis may be found in:
(a) Left ventricular failure
Haemoptysis in a cardiac patient comes with left-sided failure: frothy blood-tinged sputum of acute pulmonary oedema, winter bronchitis from pulmonary congestion, and pulmonary apoplexy from rupture of bronchial varices. Haemoptysis is also one of the backward manifestations of Lt-sided heart failure.
- Regarding Kerley's B lines, all of the following are true except:
(c) Its presence indicates left atrial pressure >10 mmHg
Kerley's B lines are linear horizontal shadows near the costophrenic angles due to oedema of the interlobular septa, and mitral stenosis is a cause of cardiogenic pulmonary oedema. A pressure just above 10 mmHg is still inside the normal wedge pressure of 5-12 mmHg, so (c) is the false statement.
- Which of the following disorders is not associated with ventricular tachycardia as a cause of syncope?
(c) Atrial myxoma
Left atrial myxoma causes syncope by obstruction of blood flow, listed with ball and valve embolus, not by ventricular tachycardia. HOCM commonly causes ventricular tachycardia and an old infarction scar is an ischaemic cause of arrhythmia; aortic stenosis is a listed cause of cardiac syncope.

6 Cardiac syncope is characterized by:

(c) Rapid recovery

Syncope is a transient loss of consciousness due to acute cerebral ischaemia, so recovery is rapid. Only prolonged ischaemia leads to convulsions, coma and brain damage, so a residual neurodeficit does not fit.

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

1 Regarding oedema in case of heart failure, which of the following is not true:

(c) Increased capillary permeability

The handout lists all four in cardiac oedema: oedema is pitting, and its pathogenesis includes salt and water retention with renin and aldosterone secretion and increased capillary permeability due to hypoxia. So strictly no option is false, and (c) is kept only as the printed answer.

2 Ascites could precede lower limb oedema in case of:

(d) All of the above

Ascites may develop before oedema (ascites precox) in pericardial effusion and constrictive pericarditis, and in tricuspid incompetence or stenosis, which gives marked liver congestion and portal hypertension and may end in cardiac cirrhosis. So (b) and (c) are true and the printed answer is 'all of the above'.

3 Regarding oedema in case of heart failure, which of the following is false:

(c) Hypoalbuminemia

The handout lists all four in cardiac oedema: oedema is pitting, and its pathogenesis includes salt and water retention with renin and aldosterone secretion and hypoalbuminaemia from low intake, low absorption, low liver synthesis and urinary loss. So strictly no option is false, and (c) is kept only as the printed answer.

4 In a heart failure patient, ascites could precede lower limb oedema in case of:

(e) All of the above

Ascites may develop before oedema (ascites precox) in pericardial effusion and constrictive pericarditis, and in tricuspid incompetence or stenosis, which gives marked liver congestion and portal hypertension and may end in cardiac cirrhosis. So (b) and (c) are true and the printed answer is 'all of the above'.

5 Syncopal attack is associated with all of the following except:

(a) Myocarditis

Arrhythmias (severe tachycardia such as ventricular fibrillation), aortic stenosis and HOCM are listed as causes of cardiac syncope. Myocarditis is not in that list, so it is the expected answer, although the handout gives it as a cause of acute left-sided failure and of arrhythmia.

Model Answers | Essay Questions

1 Enumerate causes of life-threatening chest pain.

Cardiac pain

- * Ischaemic heart disease:
 - M.I.
 - Angina pectoris.
- * Pericardial disease:
 - Dry pericarditis.
 - Massive pericardial effusion.
- * Massive pulmonary embolism and pulmonary infarction.
- * Aortic dissection and aortic aneurysm.

- * Cardiac neurosis:
 - Occurs in neurotic individuals.
 - Site: Lt inframammary and localized.
 - Radiation: no specific radiation (may be to Lt arm).
 - Character: stitching or stabbing.
 - No relation to exertion and no relief by rest.
 - Variable duration (seconds or hours).
 - Associated with local tenderness and may be other features of neurosis.
 - Normal cardiac exam.
- * Huge cardiomegaly: may rarely cause retrosternal heaviness.
- * Mitral valve prolapse, AS, AR.

N.B. Huge lt. atrium → sawing pain "erosion of spine".

Differential diagnosis of acute chest pain

	Cause	Clue in ABCDE approach
Cardiac causes	Pericarditis	Pericardial rub
	Myocarditis	Pericardial rub
	Pulmonary embolism	Tachycardia, desaturation, and clear back
Chest causes	Tension pneumothorax	Diminished air entry on one side
	Pleuritis	Diminished air entry on one side if with effusion
Vascular causes	Aortic dissection	Unequal pulse and BP

	Cause	Clue in ABCDE approach
GIT causes	Cholecystitis	Epigastric tenderness
	Pancreatitis	Epigastric tenderness
	Esophageal spasm	
Chest wall causes	Myositis	Tenderness
	Chest wall trauma	Tenderness and visible injury
	Herpes zoster infection	Rash on the skin

Note The handout has no list titled life-threatening chest pain; its causes of cardiac pain and of acute chest pain are given.

2 Enumerate the differential diagnosis of cases with chest pain.

Cardiac pain

- * Ischaemic heart disease:
 - M.I.
 - Angina pectoris.
- * Pericardial disease:
 - Dry pericarditis.
 - Massive pericardial effusion.
- * Massive pulmonary embolism and pulmonary infarction.
- * Aortic dissection and aortic aneurysm.

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Differential diagnosis of acute chest pain

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Chest wall causes	Myositis	Tenderness
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3 How would you approach such a case?

Anginal chest pain

- * Constricting / heavy discomfort to the chest, jaw, neck, shoulders, or arms.
- * Symptoms brought on by exertion, emotion, cold weather and heavy meals.
- * Symptoms relieved within 5 min by rest or nitro-glycerine (GTN).

Number of features	Type of chest pain
All the 3 features	Typical angina
Only 2 features	Atypical angina
0-1 feature	Non-anginal chest pain

- * Anginal pain usually lasts for less than **15 minutes**.
- * Anginal pain could be only in the chest, in the chest and referring, or only in the referred areas.

Angina equivalents

Dyspnoea, fatigue, nausea, sweatiness, and faintness.

Features that make angina less likely

- * Pain that is continuous or lasting for seconds is less likely related to ischemic origin.
- * Pleuritic localized chest pain or that worse with swallowing is mostly non-ischemic pain.

Cardiac neurosis

- * Occurs in neurotic individuals.
- * Site: Lt inframammary and localized.
- * Radiation: no specific radiation (may be to Lt arm).
- * Character: stitching or stabbing.
- * No relation to exertion and no relief by rest.
- * Variable duration (seconds or hours).
- * Associated with local tenderness and may be other features of neurosis.
- * Normal cardiac exam.

Note The handout gives no set approach to this case, answer incomplete.

Heart Failure

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Question no.	1	2	3	4	5	6	7	8	9	10	11	12
Answer	a	c	c	a	b	c	e	c	d	c	b	c

Question no.	13	14	15	16	17	18	19	20	21	22	23	24
Answer	b	c	c	d	d	d	d	c	d	c	a	a

Question no.	25	26	27	28	29	30	31	32	33	34	35	36
Answer	c	d	c	c	a	c	c	d	b	d	c	a

Question no.	37	38	39	40	41	42	43	44	45	46	47	48
Answer	a	d	a	d	c	a	c	a	c	c	b	b

Question no.	49
Answer	a

► Second | Questions with a Note

Question no.	1	2	3	4	5	6	7	8
Answer	a	b	d	d	a+b	c	d	c

► Why this answer?

1 Which of the following is not a manifestation of heart failure in children?

(a) Large volume pulse

In heart failure the pulse is weak because of the low COP, so a large volume pulse is not a feature. Tachycardia, oliguria and an enlarged tender liver are all features of H.F..

- 2 Causes of acute right sided heart failure are all of the following except:
(c) Malignant hypertension
 Malignant hypertension is systemic HPN, a pressure overload on the left ventricle. Pulm. HPN, tricuspid incompetence and right ventricular myocardial disease (the same like Lt. v., e.g. infarction) are causes of right ventricular failure.
- 3 Systemic congestive manifestations (right-sided heart failure) do not include:
(c) Bilateral basal crepitations
 Bilateral basal crepitations are a backwards sign of left-sided H.F., due to pulmonary congestion. Oedema of the lower limbs, ascites and pain in the Rt. hypochondrium are systemic congestion features of right-sided H.F..
- 4 Regarding selective beta-1 blockers use in heart failure, which is incorrect?
(a) Beta-1 blockers increase the deleterious effects of...
 Nothing in the handout says beta-blockers increase the harmful effects of the sympathetic system; they can be used in treatment of stable HF, so (a) is incorrect. -ve inotropic agents e.g. B-blockers are a precipitating factor of H.F., which supports (c) and (d).
- 5 Which of the following is not considered a cause of acute left-sided heart failure?
(b) Acute pulmonary embolism-induced pulmonary hypertension.
 Pulm. HPN is a pressure overload on the right ventricle, so pulmonary embolism-induced pulmonary hypertension causes right-sided, not left-sided, failure. Systemic HPN, myocardial infarction and myocarditis are causes of left ventricular failure.
- 6 What is the primary role of diuretics in the management of heart failure?
(c) Control pulmonary or systemic congestion.
 Diuretics promote loss of salt and H₂O, so they reduce the preload, circulatory and pulmonary congestion and oedema. The only drug class that showed mortality benefit in HFpEF is SGLT2i, not diuretics.
- 7 Causes of acute right sided heart failure are all of the following except:
(e) None of the above
 Pulm. HPN is a pressure overload and tricuspid incompetence a volume overload on the right ventricle, and its myocardial disease is 'the same like Lt. v.' (e.g. infarction), so a, b and d are causes of right ventricular failure. Pulmonary regurgitation is not in this list, but it also returns blood to the right ventricle (a volume overload), so all four are causes and the answer is 'None of the above'.
- 8 Which of the following drugs is not a treatment of heart failure in children?
(c) Hydrocortisone
 Corticosteroids are an iatrogenic precipitating factor of heart failure. Diuretics, digitalis (digoxin) and ACE inhibitors such as captopril are lines of treatment of H.F..
- 9 The drug classes that showed benefit in heart failure management are:
(d) All of the above
 ACE inhibitors in H.F. have a better effect than other vasodilators, and ARBs act as ACE inhibitors. SGLT2i is the only drug class that showed mortality benefit in HFmrEF and HFpEF. So all three classes have benefit.
- 10 Regarding selective beta blockers, all are true except:
(c) BB increase the deleterious effects of sympathetic nervous...
 Beta-blockers e.g. carvedilol and bisoprolol can be used in treatment of stable HF; nothing in the handout says they increase the harmful effects of the sympathetic system, so (c) is false. Options b and d are true: -ve inotropic agents e.g. B-blockers are an iatrogenic precipitating factor of H.F..
- 11 All of the following are symptoms of right-sided heart failure except:
(b) Left hypochondrial pain
 The pain of right-sided H.F. is in the Rt. hypochondrium and epigastrium, not the left. Oedema of the lower limbs, congested neck veins and an enlarged tender liver are right-sided features.
- 12 Switching from ACE inhibitor to an ARB improves:
(c) Dry cough
 Cough with ACE inhibitors is due to increased bradykinin; ARBs act as ACE inhibitors with fewer side effects and no cough. Hyperkalaemia, hypotension and renal failure are side effects of ACE inhibitors that ARBs share.

- 13 The main antidote in digitalis toxicity is:
(b) Fab fragments
 Treatment of digitalis toxicity includes digitalis antibodies, and Fab fragments are these antibodies. Naloxone, flumazenil and BAL are antidotes of other poisons.
- 14 All the following are signs of digitalis toxicity except:
(c) Sinus tachycardia
 Digitalis toxicity gives a slow heart rate, arrhythmias and heart block, vomiting, and blurring of vision with coloured vision; digitalis slows the heart (-ve chronotropic effect). So sinus tachycardia is not a sign.
- 15 A 6-month-old infant presenting to the ER with respiratory distress. Which of the following is true in heart...
(c) Tachycardia
 Heart failure causes tachycardia because of increased sympathetic activity, and in an infant with respiratory distress tachycardia with tachypnea is the typical finding. Cyanosis is not a feature of heart failure itself in an infant; it points to cyanotic heart disease or severe lung disease. Bradycardia and polyuria do not occur (heart failure causes oliguria).
- 16 Which of the following drugs is NOT used in management of heart failure?
(d) Calcium channel blockers (CCBs)
 Ca-channel blockers are not used in H.F. due to their -ve inotropic effect. ACE inhibitors, ARBs and SGLT2i are used.
- 17 Which of the following is NOT a feature of low cardiac output symptoms of heart failure?
(d) Bilateral pitting lower limb edema
 Oedema of the lower limbs is a backwards manifestation of right-sided H.F., due to systemic congestion. Easy fatigability, dizziness and syncope, and coldness of the extremities are forwards (low COP) manifestations.
- 18 Which of the following is NOT a feature of left ventricular enlargement?
(d) The apex usually is diffuse and is shifted outwards
 A diffuse apex displaced outwards is a sign of right ventricular enlargement. In left ventricular enlargement the apex is directed outwards and downwards and is localized, sustained (heaving) in hypertrophy and hyperdynamic in dilatation, as in aortic stenosis and aortic regurgitation.
- 19 Low cardiac output and hypotension lead to reflex sympathetic activation which leads to all of the following...
(d) Vasoconstriction decreases the pressure overload in front...
 In the neurohormonal diagram, neurohumoral activation causes vasoconstriction, which leads to increased afterload on the failing heart, so it does not improve H.F.. This is why vasodilators are used to decrease the after-load.
- 20 As regards heart failure pathophysiology, angiotensin II leads to all of the following except:
(c) Increased urine output
 Angiotensin II causes salt and water retention, peripheral vasoconstriction and activation of the sympathetic system, which ACE inhibitors counteract, and the renin-angiotensin system leads to sodium and water retention. So urine output falls, it does not rise.
- 21 * Digitalis poisoning can develop as a result of:
(d) All of the above
 The handout gives only that digoxin is excreted mainly by the kidney; the answer is inferred from this line, as renal malfunction lets the drug accumulate. Accidental ingestion and deliberate overdose are the acute forms of poisoning, so all three are true.
- 22 The following are signs of right-sided heart failure except:
(c) Pulmonary edema
 Acute pulmonary oedema is a backwards sign of left-sided H.F., due to pulmonary congestion. Oedema of the lower limbs and ascites are backwards signs of right-sided H.F., and oliguria is a forwards (low COP) manifestation found in both sides.
- 23 Which of the following is not a cause of systolic dysfunction in the pediatric population?
(a) Hypertrophic cardiomyopathy
 The handout puts HOCM under diastolic failure, while DCM causes systolic failure. Myocarditis is a myocardial disease that causes ventricular failure. Malnutrition is not mentioned in the handout.

- 24** What is the most probable diagnosis?
(a) Heart failure with preserved ejection fraction (HFpEF)
 The ejection fraction is 60% with poor diastolic function, and the handout defines HFpEF as EF > 50% and HFrEF as EF < 40%. Basal crepitations (pulmonary congestion) with congested neck veins and an enlarged tender liver (systemic congestion) show that both sides are affected, so it is not isolated right ventricular failure.
- 25** Which of the following is the most approved to improve the prognosis in such a patient?
(c) Dapagliflozin
 This patient has HFpEF. The only drug class that showed mortality benefit in HFmrEF and HFpEF is SGLT2i, e.g. empagliflozin / dapagliflozin; the role of the other disease-modifying drugs in HFpEF is still unclear.
- 26** The following medication is recommended for our patient, but hypokalemia is a common side effect:
(d) Torsemide
 Torsemide is a loop diuretic, and loop diuretics cause hypokalaemia. Spironolactone causes hyperkalaemia; ACE inhibitors cause hyperkalaemia and ARBs such as valsartan act as ACE inhibitors. Nebivolol is a beta-blocker with no effect on potassium in the handout.
- 27** What is the added value of sacubitril to valsartan in ARNI?
(c) Has indirect diuretic and vasodilator effect
 Sacubitril is an endopeptidase inhibitor that increases ANP. It is the natriuretic peptide, not sacubitril itself, that inhibits Na reabsorption and increases Na and H₂O excretion, so its effect is indirect.
- 28** Which of the following could predict higher risk of stroke in such a patient?
(c) Dilated left atrium
 As left atrial pressure remains elevated, the left atrium increases in size and there is a greater chance of developing atrial fibrillation, and stroke is a complication of AF. A dilated left atrium > 50 mm is an indication for oral anticoagulation.
- 29** Tachycardia in children with congestive heart failure is due to:
(a) Increased catecholamine release
 Tachycardia in H.F. occurs as a result of sympathetic stimulation by Marey's law & Bainbridge reflex, that is, by catecholamine release. The renin-angiotensin system leads to sodium and water retention and vasoconstriction, not tachycardia.
- 30** Which of the following could lead to increased volume overload on the left ventricle?
(c) Aortic regurgitation
 Aortic incompetence is a volume overload on the left ventricle. Aortic stenosis is a pressure overload, mitral stenosis causes left atrial failure, and tricuspid incompetence is a volume overload on the right ventricle.
- 31** The following medications are approved to improve the prognosis of heart failure except:
(c) Digoxin
 The handout states that the disease-modifying drugs decrease mortality in patients with HFrEF. Ramipril (ACE inhibitor), metoprolol succinate (beta-blocker) and eplerenone (aldosterone antagonist) are disease-modifying drugs, while digoxin improves symptoms but does not improve survival.
- 32** The following medication is recommended for this patient, but hypokalemia is a common side effect:
(d) Frusemide
 Frusemide is a loop diuretic, and loop diuretics cause hypokalaemia. Spironolactone causes hyperkalaemia, and candesartan, an ARB, acts as ACE inhibitors, which cause hyperkalaemia.
- 33** The patient started medical treatment of heart failure and started to experience dry cough. Which of the...
(b) Sacubitril-valsartan
 Cough with ACE inhibitors is due to increased bradykinin, and ARBs such as valsartan give no cough. Sacubitril is an endopeptidase inhibitor; the same enzyme also breaks down bradykinin, so sacubitril-valsartan can cause dry cough. Eplerenone, carvedilol and dapagliflozin do not cause cough.
- 34** Which of the following could cause congestive heart failure in an 8-month-old infant?
(d) All of the above
 VSD is a volume overload and myocarditis a myocardial disease cause of heart failure, and anaemia causes hyperdynamic circulation with high COP failure. So all three can cause heart failure.

- 35 * The following drug/s improve prognosis of patient with dilated cardiomyopathy:
(c) Carvedilol
 Inferred from the handout: DCM causes systolic failure, and carvedilol is a beta-blocker that can be used in treatment of stable HF. Nicorandil and ranolazine are 3rd line anti-anginal drugs.
- 36 Following up creatinine showed that it rose to 3.5 mg/dl. Which of the following drugs must be stopped:
(a) Candesartan
 ACE inhibitors can cause acute renal failure (vasodilatation of the efferent arteriole → decreased GFR), and renal functions must be monitored; ARBs such as candesartan act as ACE inhibitors. So candesartan must be stopped when creatinine rises to 3.5 mg/dl. Aspirin, clopidogrel and metoprolol do not act this way.
- 37 Hepatomegaly in children with congestive heart failure is due to:
(a) Systemic venous congestion
 Enlarged tender liver is a backwards manifestation of right-sided H.F., due to systemic congestion. Pulmonary congestion gives the backwards manifestations of left-sided H.F., and low COP gives the forwards manifestations.
- 38 The following medication must be avoided in the treatment of such a patient:
(d) Amlodipine
 Ca-channel blockers are not used in H.F. due to their -ve inotropic effect, and amlodipine is a Ca-channel blocker. In mitral stenosis, diuretics and betablockers (to slow heart rate and increase the diastolic time) are used to control pulmonary congestion.
- 39 As regards natriuretic peptides, all of the following are true except:
(a) Decreased intracardiac pressures lead to increased release...
 Overload on the left ventricle increases BNP, so natriuretic peptides rise with increased, not decreased, intracardiac pressure. The natriuretic hormone inhibits Na reabsorption and increases Na and H₂O excretion, and sacubitril is an endopeptidase inhibitor that increases ANP.
- 40 Sodium-glucose cotransporter-2 inhibitors (SGLT2i): all are true except:
(d) SGLT2i are diuretics that cause severe electrolyte...
 SGLT2i work by inhibiting SGLT-2 in the proximal tubules, reducing renal reabsorption of glucose and increasing urinary excretion of glucose. The handout does not describe them as diuretics with severe electrolyte disturbance, and they are the only drug class that showed mortality benefit in HFmrEF and HFpEF.
- 41 Objectives of treatment of heart failure include all of the following except:
(c) No role for interventional and surgical options
 Interventional and surgical options do have a role: surgical treatment e.g. for valvular and congenital lesions, mechanical assistance of circulation using intra-aortic balloon counterpulsation, and cardiac transplantation.
- 42 Which of the following medication must be avoided in the treatment of this patient?
(a) Verapamil.
 Non-DHP CCBs (verapamil and diltiazem) must be avoided in heart failure with reduced systolic function as they worsen heart failure; Ca-channel blockers are not used in H.F. due to their -ve inotropic effect. Metoprolol, spironolactone and candesartan are used in H.F..
- 43 Which of the following is NOT a feature of right-sided heart failure?
(c) Paroxysmal nocturnal dyspnea (PND)
 PND is due to aggravation of pulmonary congestion and occurs more commonly in Lt. VF, so it belongs to left-sided H.F. Oedema of the lower limbs, enlarged tender liver and pain in the Rt. hypochondrium are right-sided features.
- 44 In medical treatment of heart failure, which is false?
(a) Selective beta-1 blockers (carvedilol / bisoprolol / ...)
 Beta-blockers e.g. carvedilol and bisoprolol can be used in treatment of stable HF, so (a) is false. Loop diuretics are the most potent diuretics, ACE inhibitors prevent conversion of angiotensin I to II, and sacubitril is an endopeptidase inhibitor that increases ANP.

- 45 *Tachycardia in children with congestive heart failure is due to:*
(c) Increased catecholamine release from suprarenal medulla
 Tachycardia in H.F. occurs as a result of sympathetic stimulation by Marey's law & Bainbridge reflex, that is, by catecholamine release. Low COP and venous congestion act only through these reflexes, and renin acts through salt and water retention.
- 46 *A 9-month-old infant presenting to the ER with congestive heart failure. Which of the following is true?*
(c) Tachycardia
 Tachycardia is present in H.F. due to sympathetic stimulation. H.F. gives coldness of the extremities and oliguria, so warm extremities, polyuria and bradycardia are the opposite.
- 47 *Pulsus alternans is produced by:*
(b) Left-sided heart failure
 Pulsus alternans (alternating strong & weak beats) is a cardiovascular sign of left-sided H.F.. Pericardial effusion gives low voltage ECG and sometimes electrical alternans, not pulsus alternans.
- 48 *In a patient with history of shortness of breath, which of the following signs indicates left heart failure?*
(b) Basal crepitations
 Bilateral basal crepitations are a backwards sign of left-sided H.F., due to pulmonary congestion. Ascites, oedema of the lower limbs and congested neck veins are backwards signs of right-sided H.F..
- 49 *The action of digitalis is augmented by:*
(a) Calcium
 Digitalis inhibits Na-K/ATPase → increased intracellular Na → increased intracellular Ca → increased force of contraction, so calcium augments its action. Hypokalaemia must be corrected in digitalis toxicity.

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

- 1 *Which of the following is not a manifestation of heart failure in children?*
(a) Splenomegaly
 Oliguria (low COP), tachycardia and an enlarged tender liver (systemic congestion) are manifestations of H.F.. Splenomegaly is not among them; the handout mentions hepatosplenomegaly only when pulmonary hypertension is accompanied by RV failure.
- 2 *Heaving apex is found in:*
(b) Aortic regurgitation
 A sustained (heaving) apex is a sign of left ventricular hypertrophy, while dilatation gives a hyperdynamic apex. In AR the left ventricle is enlarged, so (b) is the expected answer, although the handout gives AR a hyperdynamic apex. Pulmonary stenosis loads the right ventricle and gives a left parasternal heave, not a heaving apex.
- 3 *In case of acute digitalis poisoning, toxicity is mainly due to its action on:*
(d) Cardiac muscle
 Digitalis increases the excitability of atria and ventricles, which may be complicated by arrhythmia, and its toxicity is shown mainly by arrhythmias and heart block. The handout also gives a vagal action, so (c) is the tempting option.
- 4 *The following are signs of digitalis toxicity except:*
(d) Diarrhea
 The GIT manifestations of digitalis toxicity are anorexia (usually the 1st symptom), nausea and vomiting, and arrhythmias are its cardiovascular manifestations. Diarrhoea is not in this list.
- 5 *The following are heart failure causes with intermittent intracardiac obstruction (choose all that apply):*
(a) Left atrial myxoma + (b) Hypertrophic obstructive cardiomyopathy
 A left atrial myxoma moves with each beat and blocks the mitral valve only at times (ball valve), and the outflow obstruction in HOCM is dynamic, changing with load and contractility. Tetralogy of Fallot has right ventricular outflow obstruction, but it is mainly fixed and Fallot does not usually cause heart failure, because the large VSD lets the right ventricle empty into the aorta. Calcific aortic stenosis is a fixed obstruction.

6 The following are manifestations of acute congestive heart failure in children except:

(c) Splenomegaly

Tachycardia, tachypnea and an enlarged tender liver are manifestations of H.F. Splenomegaly is not among them; the handout mentions hepatosplenomegaly only when pulmonary hypertension is accompanied by RV failure.

7 Drug therapy of congestive heart failure in children includes all of the following drugs except:

(d) Alpha adrenergic blockers

Furosemide, digitalis (digoxin) and captopril are drugs of H.F.. (d) is the printed answer, although the handout lists alpha blockers e.g. prazosin among the types of vasodilators in H.F..

8 A 6-month-old infant presenting to the ER with congestive heart failure. Which of the following is not true?

(c) Splenomegaly

Oliguria, tachypnea and an enlarged tender liver are manifestations of H.F. Splenomegaly is not among them; the handout mentions hepatosplenomegaly only when pulmonary hypertension is accompanied by RV failure.

Model Answers | Essay Questions

1 Outline the management of a patient with acute heart failure.

- ① Hospitalization:** preferably in ICU and bed rest in sitting position.
- ② Oxygen inhalation:** 100% if no pre-existing lung disease.
- ③ Furosemide:** **40-80 mg** I.V. and could be repeated every 30 minutes according to the response.
- ④ Morphine:** **5-10 mg** I.V. [caution in liver failure & COPD]. Action:
 - Relieves anxiety.
 - Decreases sensitivity of the respiratory centre to reflex stimulation from the congested lungs.
 - Decreases venous return (through venodilatation).
- ⑤ Vasodilators:** Na nitroprusside 0.5-10 µg/kg/min I.V. infusion or nitroglycerine I.V. infusion (Tridil) or sublingually [not given if systolic BP < **90 mm Hg**].
- ⑥ Aminophylline:** 5.6 mg/kg slowly I.V. over 20 min (average dose: 250-500 mg).
- ⑦ Positive inotropics** [if systolic BP < **100 mm Hg**]:
 - I.V. digitalization may be used especially in cases associated with AF.
 - Other parenteral inotropics may be needed e.g. dopamine and dobutamine.
- ⑧ Correction of the cause and precipitating factors** e.g. AF.
- ⑨ Aspiration of respiratory secretions.**
- ⑩** If there is no response, the following measures may be used:
 - Mechanical ventilation [e.g. CPAP].
 - Mechanical removal of fluid using phlebotomy or dialysis.
 - Mechanical assistance of circulation using intra-aortic balloon counterpulsation: the balloon is automatically inflated with CO₂ at the onset of diastole and rapidly deflated just before the onset of systole. The diastolic inflation will help in pushing blood in the aorta and tissues.

Note The handout has no section on acute heart failure; this is its treatment of acute pulmonary oedema.

2 Classify heart failure according to anatomy and cardiac output.

Classification of H.F.

- ①** Left-sided H.F., right-sided H.F. and combined (left → right = **CHF**).

- ② According to cardiac output (COP):
- (A) **Low COP failure:**
 - There is decrease of COP below normal.
 - This type occurs in most cases of H.F.
 - (B) **High COP failure:**
 - There is increase of COP, however insufficient for metabolic needs of tissues.
 - Occurs in hyperdynamic circulation (HDC):
 - 3 A: Anaemia, A.V. fistula, AR.
 - 3 H: Hyperthyroidism, hypoxic cor pulmonale and hepatic failure.
 - 3 P: PDA, Paget's disease of bone, beriberi.

3 Mention the causes of acute heart failure.

Precipitating factors (3 I, 3 P, 2 A)

- ① **Infections:** esp. infective endocarditis, rheumatic activity, chest infections.
- ② **Myocardial infarction.**
- ③ **Iatrogenic** factors:
 - Discontinuation of anti-failure therapy.
 - Excessive salt intake.
 - I.V. fluids.
 - Corticosteroids.
 - -ve inotropic agents e.g. B-blockers.
- ④ **Physical & emotional stress.**
- ⑤ **Pregnancy & labour.**
- ⑥ **Pulmonary embolism.**
- ⑦ **Arrhythmias** e.g. marked tachycardia or bradycardia.
- ⑧ **Anaemia**, thyrotoxicosis & other causes of hyperdynamic circulation.

Acute H.F.

- * Acute H.F. e.g. in **M.I.**
- * In patients with **acute severe AR**, left ventricular diastolic pressures rise rapidly and may lead to acute heart failure (acute pulmonary edema ± cardiogenic shock).
- * **Pulmonary embolism:** rapid appearance of manifestations of pulmonary hypertension & right ventricular failure (acute cor pulmonale).

Note The handout has no single list of the causes of acute heart failure; the answer is collected from several topics.

4 Heart failure with reduced EF is a multifactorial process. Enumerate 3 underlying pathophysiological mechanisms and their effects.

Compensatory mechanisms (cardiac reserve)

Several mechanisms become operable to compensate for the reduction in the cardiac output. These mechanisms are beneficial within certain limits. If they exceed these limits, they will aggravate H.F.

	When / why it occurs	Benefit	If it exceeds the limits
(1) Tachycardia	Occurs as a result of sympathetic stimulation by Marey's law & Bainbridge reflex.	It increases COP.	If tachycardia is marked (e.g. > 160/m) → decreases COP due to shortening of diastole & impairment of cardiac filling.
(2) Dilatation	Occurs mainly in volume overload.	It increases the force of contraction (according to Starling's law).	It will diminish contraction.
(3) Hypertrophy	Occurs mainly in pressure overload.	It increases the force of contraction.	The cardiac muscle will be ischaemic leading to decreased contraction.
(4) Hypervolemia	Occurs due to salt & H ₂ O retention.	It increases the preload & force of contraction.	It will aggravate heart failure.
(5) Redistribution of blood flow	There is diversion of blood from less vital organs (e.g. skin) to more vital organs (e.g. brain & heart).		

Neurohormonal and compensatory mechanisms in heart failure

- * Reduced cardiac output → **neurohumoral activation**: sympathetic nervous system, renin-angiotensin system, vasopressin system, endothelin system.
- * Vasoconstriction → increased afterload.
- * Sodium and water retention → increased intravascular volume.

Rheumatic Fever

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	7	8	9	10	11	12
Answer	d	c	a	c	a	a	d	d	a	a	a	c

Question no.	13	14	15	16	17	18	19	20	21
Answer	a	a	c	b	c	b	c	b	c

► Second | Questions with a Note

Question no.	1	2
Answer	b	a

► Why this answer?

- What is the recommended duration for secondary prophylaxis after rheumatic fever?
(d) At least until 40 years of age / for life in patients...
 Secondary prevention lasts till age 40 years or lifelong in patients with severe valvular disease or after valvular replacement. Without carditis it is till age 21 (at least 5 years) and with mild regurgitant lesion till age 25 (at least 10 years), so a, b and c mix up the groups.
- What is the most commonly affected valve in rheumatic heart disease?
(c) Mitral valve
 Mitral valve is commonly involved, followed by aortic valve then tricuspid valve.
- Which of the following is a minor criterion of acute rheumatic fever?
(a) Fever
 Fever is a minor criterion. Erythema marginatum and carditis are major criteria, and a raised ASO titre is evidence of recent streptococcal infection.
- Which of the following is false regarding rheumatic chorea in children?
(c) Associated with hypertonia and hyperreflexia
 Sydenham's chorea gives hypotonia and hyporeflexia, not hypertonia and hyperreflexia. It is more common in girls over 8 years, gives emotional instability and interrupted speech, and could occur alone weeks or months after the streptococcal infection.
- Major manifestations of rheumatic fever are the following EXCEPT:
(a) Fever
 Fever is a minor criterion. Arthritis, carditis and Sydenham's chorea are major criteria.

- 6 Regarding acute rheumatic fever, which of the following is not true?
(a) Raised antistreptolysin O titre is a minor criterion
 A raised ASO titre is evidence of recent streptococcal infection, not a minor criterion. Arthralgia and raised ESR are minor criteria, and the arthritis affects big joints.
- 7 Which of the following is not a risk factor for the development of rheumatic fever?
(d) Streptococcal skin infection
 Rheumatic fever follows streptococcal pharyngitis with group A beta hemolytic streptococci, not skin infection. Good nutrition, proper hygiene, good housing and adequate ventilation are the primordial prevention of recurrent pharyngitis, so their absence is a risk.
- 8 Which of the following is true regarding secondary prevention of rheumatic fever?
(d) It aims to prevent colonization of the upper respiratory...
 Secondary prevention is of recurrent attacks, by monthly benzathine penicillin G or daily erythromycin. Preventing the first attack is primary prevention, the 10 days course belongs to the acute phase, and patients with carditis need the longest prophylaxis.
- 9 Regarding acute rheumatic fever, which of the following is not true?
(a) Cardiomegaly is a minor criterion
 Cardiomegaly is not a minor criterion; the minor criteria are fever, arthralgia, raised CRP, ESR and/or total leucocytic count, and prolonged PR interval. Arthralgia and raised ESR are minor criteria, and the arthritis affects big joints.
- 10 Regarding acute rheumatic fever, which of the following is not true?
(a) Hepatomegaly is a minor criterion
 Hepatomegaly is not a minor criterion; the minor criteria are fever, arthralgia, raised CRP, ESR and/or total leucocytic count, and prolonged PR interval. Arthralgia and raised ESR are minor criteria, and the arthritis affects big joints.
- 11 The minor Jones criterion for the diagnosis of acute rheumatic fever in the ECG is:
(a) Prolonged P-R interval
 Prolonged PR interval in ECG is the ECG minor criterion.
- 12 Primary prevention of rheumatic fever (RF) is defined as adequate antibiotic therapy of:
(c) Group A streptococcal URT infection
 Primary prevention is good treatment of acute pharyngitis: complete course of antibiotic in treatment of any acute streptococcal pharyngitis. The organism is group A beta hemolytic streptococci.
- 13 The minor criterion of acute rheumatic fever in the ECG is:
(a) Prolonged PR interval
 The ECG minor criterion is prolonged PR interval. 'Heart block' is wider, as it includes first degree, second degree and complete heart block, so (a) is the exact answer.
- 14 Which of the following is a primary prevention of rheumatic fever in individuals with streptococcal...
(a) Proper treatment with antibiotics
 Primary prevention is complete course of antibiotic in treatment of any acute streptococcal pharyngitis. Hygiene belongs to primordial prevention of recurrent pharyngitis.
- 15 Which of the following is a major criterion of acute rheumatic fever?
(c) Erythema marginatum
 Erythema marginatum is a major criterion. Fever and prolonged PR interval are minor criteria, and a raised ASO titre is evidence of recent streptococcal infection.
- 16 Regarding acute rheumatic fever, which of the following is true?
(b) PR interval is prolonged
 Prolonged PR interval in ECG is a minor criterion. Cardiomegaly is not a criterion, the arthritis affects big joints such as knee, wrist and elbow, and ESR is increased.
- 17 A 9-year-old boy is newly diagnosed with acute rheumatic fever. Which of the following is a major criterion?
(c) Erythema marginatum
 Erythema marginatum is a major criterion. Fever, arthralgia and prolonged PR interval are minor criteria.

- 18** A 7-year-old boy has newly diagnosed acute rheumatic fever. Which of the following is NOT true?
(b) 1st hour ESR 10 mm
 ESR is increased in acute rheumatic fever (minor criterion), so a first-hour ESR of 10 mm does not fit. Fleeting (migrating) polyarthritis and fever are typical, and endocarditis gives an audible murmur of the valve lesion.
- 19** In the diagnosis of rheumatic fever, which of the following is considered one of the major criteria?
(c) Polyarthritis
 Polyarthritis is a major criterion. The rash of rheumatic fever is erythema marginatum, the organism is group A beta hemolytic streptococci, and the ASO titer must be increased.
- 20** All of the following are minor criteria in the Modified Jones criteria for the diagnosis of rheumatic fever...
(b) Subcutaneous nodules
 Subcutaneous nodules are a major criterion. Fever, arthralgia and raised ESR/CRP are minor criteria.
- 21** Earliest valvular lesion in acute rheumatic carditis is:
(c) MI
 Mitral valve is commonly involved, and early affection of the valve could lead to regurgitation. Stenosis comes long term from destruction, fibrosis and adhesion of the valve.

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

- 1** * In adults, rheumatic fever mostly bites:
(b) The joints
 Arthritis is the commonest major criterion (75%), more than carditis (50%). The answer is inferred from these two figures, as the handout has no statement about adults.
- 2** A 9-year-old boy is newly diagnosed with acute rheumatic fever. Which of the following is a major criterion?
(a) Polyarthralgia
 Fever is a minor criterion, a raised ASO titre is evidence of streptococcal infection, and the PR interval is prolonged, not short. Polyarthralgia is accepted as a major criterion only in high-risk populations; the handout lists arthralgia as a minor criterion.

Model Answers | Essay Questions

- 1** Mention the treatment of acute rheumatic fever in children.

Treatment of acute phase

- * Supportive measures:
 - Bed rest in patients with carditis and arthritis till control of HF and normalization of ESR.
 - Low salt diet in heart failure.
- * Eradicate streptococcal infection with one of the following options:
 - Single dose of IM benzathine penicillin G.
 - 10 days course of oral penicillin V or amoxycillin.
 - In penicillin allergic patients: 10 days course of erythromycin, azithromycin, or clindamycin.

Anti-inflammatory drugs

Indication	Drug	Regimen
Arthritis	Salicylates	50-70 mg/kg/day (maximum 6 gram/day) for 5 days; then 50 mg/kg/day for 3 wks; then 25 mg/kg/day for 2-4 wks
Carditis	Corticosteroids: prednisone	2 mg/kg/day (max 60 mg) for 2-3 weeks; then 1 mg/kg/day for 2-3 weeks; then gradual tapering 5 mg/day every 2-3 days with introduction of 50 mg aspirin/day for 6 weeks

Other lines of treatment

- * Rheumatic chorea: haloperidol.
- * Heart failure: see heart failure.
- * Treatment of complications could include surgical replacement of diseased valve.

Secondary prevention of recurrent attacks

- * Monthly benzathine penicillin G or daily erythromycin in penicillin allergic patient.

Duration of secondary prevention

Duration	Patient
Till age 21 years (at least 5 years)	Patients without carditis
Till age 25 years (at least 10 years)	Patients with mild regurgitant lesion
Till age 40 years or lifelong	Patients with severe valvular disease or patients underwent valvular replacement

Prophylaxis against infective endocarditis

See infective endocarditis.

2 Discuss 4 major criteria of acute rheumatic fever.

A. Arthritis (75%)

- * Usually polyarthritis (≥ 4 joints).
- * Affecting big joints (e.g., knee, wrist, elbow...).
- * Fleeting: migrating from one joint to the other.
- * The affected joint shows: redness, hotness, swelling, limitation of movement $\pm$ effusion.
- * Good response to salicylates.
- * Spontaneous remission without deformity.

B. Clinical or subclinical carditis (50%)

- * Endocarditis:
 - Its presence is a must to diagnose rheumatic carditis.
 - Mitral valve is commonly involved followed by aortic valve then tricuspid valve.
 - Poly-valvular affection is the rule.
 - Early affection of the valve could lead to regurgitation.
 - Destruction, fibrosis, and adhesion of the valve will lead to long term stenosis, regurgitation, or combined stenosis and regurgitation.
 - Subclinical endocarditis is major criteria.

Type	Symptom	Sign	Investigation
Endocarditis	Asymptomatic or dyspnea	Audible murmur of valve lesion	Echocardiography will pick up valvular lesions (subclinical endocarditis is defined when detected in echocardiography despite being asymptomatic with no audible murmur)
Dry pericarditis	Stitching chest pain	Pericardial rub on auscultation	ECG: diffuse ST elevation in all leads with PR depression in V5 and V6
Pericardial effusion	Dull aching pain and dyspnea	Distant heart sounds on auscultation	Echocardiography will pick up pericardial effusion
Myocarditis	Dyspnea and palpitations	Gallop rhythm & muffled heart sounds	Echocardiography will pick up systolic dysfunction associated with myocarditis

C. Sydenham's chorea

- * More common in girls > 8 years due to affection of the basal ganglia.
- * Could occur alone (diagnostic alone) after weeks or months of streptococcal infection.

Symptom

- * Involuntary movements: sudden involuntary jerky movements of:
 - The limbs (fall of objects from the hand and unsteady gait).
 - The tongue (interrupted speech).
 - The face (abnormal movements).
- * Emotional instability (sudden laughing or crying).

Sign

- * Hypotonia and hyporeflexia:
 - Inability to keep the arm protruded but wrist flexion and finger hyperextension occurs (dinner fork appearance).
 - Milkmaid's grip (the inability to maintain voluntary contraction of hand muscles on active grip).
 - Inability to keep the tongue protruded.
 - Hypotonia may be severe enough to cause paralysis.

D. Erythema marginatum (<5%)

- * Evanescent wavy rings of erythema with sharp margins, not itchy nor painful.
- * Most common site is the trunk and proximal part of the extremities.

E. Subcutaneous nodules (1%)

- * Firm, painless, mobile nodules under the skin present mainly over the extensor surfaces and bony prominences e.g. scapula, elbow & shin of the tibia.
- * It is usually associated with severe carditis.

3 Discuss briefly clinical features of rheumatic chorea.

- * More common in girls > 8 years due to affection of the basal ganglia.
- * Could occur alone (diagnostic alone) after weeks or months of streptococcal infection.

Symptom

- * Involuntary movements: sudden involuntary jerky movements of:
 - The limbs (fall of objects from the hand and unsteady gait).
 - The tongue (interrupted speech).
 - The face (abnormal movements).
- * Emotional instability (sudden laughing or crying).

Sign

- * Hypotonia and hyporeflexia:
 - Inability to keep the arm protruded but wrist flexion and finger hyperextension occurs (dinner fork appearance).
 - Milkmaid's grip (the inability to maintain voluntary contraction of hand muscles on active grip).
 - Inability to keep the tongue protruded.
 - Hypotonia may be severe enough to cause paralysis.

4 Discuss briefly how to diagnose acute rheumatic fever.

Diagnostic criteria (Revised Jones criteria)

Required criteria

Attack	Criteria
First attack	[(2 major) or (1 major + 2 minor)] + Evidence of recent streptococcal infection
Recurrent attacks	[(2 major) or (1 major + 2 minor) or (3 minor)] + Evidence of recent streptococcal infection

Major criteria

- 1 Arthritis (**75%**).
- 2 Clinical or subclinical carditis (**50%**).
- 3 Sydenham's chorea.
- 4 Erythema marginatum (<5%).
- 5 Subcutaneous nodules (**1%**).

Minor criteria

- * Fever.
- * Arthralgia (is not considered in presence of arthritis).
- * Increased CRP, ESR, and/or total leucocytic count.
- * Prolonged PR interval in ECG (is not considered in presence of carditis).

Evidence of recent streptococcal infection

- * Positive throat culture.
- * Increased ASO titer or anti-DNase titer.
- * History of scarlet fever.

5 Enumerate three minor manifestations of acute rheumatic fever.

- * Fever.
- * Arthralgia (is not considered in presence of arthritis).
- * Increased CRP, ESR, and/or total leucocytic count.
- * Prolonged PR interval in ECG (is not considered in presence of carditis).

6 Mention pathogenesis, diagnosis, and treatment of rheumatic fever.

(a) Etiology and pathogenesis

Rheumatic fever occurs after prior (**3-4 weeks**) streptococcal pharyngitis with group A beta hemolytic streptococci (GABHS) by one of the following theories:

- * Crossed reactivity due to similar antigenicity:
 - There is a similarity between protein of GABHS and some proteins in heart, joints, brain, and skin.
 - Antibodies against GABHS cross-react with human tissues producing immune complexes that start inflammatory process that damage human tissue.
- * Altered antigenicity:
 - Less acceptable theory that suppose that there is direct infection of heart, joints, skin, and brain with subsequent alteration in the antigenic properties that stimulates autoimmune inflammatory response.

(b) Diagnosis: diagnostic criteria (Revised Jones criteria)

Required criteria

Attack	Criteria
First attack	[(2 major) or (1 major + 2 minor)] + Evidence of recent streptococcal infection
Recurrent attacks	[(2 major) or (1 major + 2 minor) or (3 minor)] + Evidence of recent streptococcal infection

Major criteria

- ① Arthritis (75%).
- ② Clinical or subclinical carditis (50%).
- ③ Sydenham's chorea.
- ④ Erythema marginatum (<5%).
- ⑤ Subcutaneous nodules (1%).

Minor criteria

- * Fever.
- * Arthralgia (is not considered in presence of arthritis).
- * Increased CRP, ESR, and/or total leucocytic count.
- * Prolonged PR interval in ECG (is not considered in presence of carditis).

Evidence of recent streptococcal infection

- * Positive throat culture.
- * Increased ASO titer or anti-DNase titer.
- * History of scarlet fever.

(c) Treatment

Treatment of acute phase

- * Supportive measures:
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 - Low salt diet in heart failure.
- * Eradicate streptococcal infection with one of the following options:
 - Single dose of IM benzathine penicillin G.
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 - In penicillin allergic patients: 10 days course of erythromycin, azithromycin, or clindamycin.

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Other lines of treatment

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- * Heart failure: see heart failure.
- * Treatment of complications could include surgical replacement of diseased valve.

Secondary prevention of recurrent attacks

- * Monthly benzathine penicillin G or daily erythromycin in penicillin allergic patient.

Duration of secondary prevention

Duration	Patient
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Till age 25 years (at least 10 years)	Patients with mild regurgitant lesion
Till age 40 years or lifelong	Patients with severe valvular disease or patients underwent valvular replacement

Prophylaxis against infective endocarditis

See infective endocarditis.

Adult Congenital Heart Disease & Cyanosis

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	7	8	9	10	11	12
Answer	a	d	b	b	b	d	b	d	c	a	a	a

Question no.	13	14	15	16	17	18	19	20	21	22	23	24
Answer	d	b	b	d	b	c	a	b	d	c	c	d

Question no.	25	26	27	28	29	30	31	32	33	34	35
Answer	d	d	b	a	a	c	e	d	c	a	c

► Second | Questions with a Note

Question no.	1	2	3	4	5
Answer	a	a	d	b	d

► Why this answer?

- 1 * The most common congenital cyanotic heart disease in children is:

(a) Fallot tetralogy

The handout calls TOF one of the most common CHDs, and it is one of the two cyanotic CHDs it studies, with reduced pulmonary blood flow. The answer is inferred from these lines; the handout does not rank the cyanotic lesions.

- 2 Which of the following is not used in treatment of cyanotic spells of Fallot's tetralogy?

(d) Hydrocortisone

For cyanotic spells the handout gives the knee-chest position, oxygen, IV morphine and IV propranolol; hydrocortisone is not among them. Sodium bicarbonate is used to correct the acidosis of a severe spell.

- 3 In MOST patients with an ASD, the characteristic physical finding is:

(b) A wide and fixed splitting of 2nd heart sound

The RV impulse is also increased in ASD, but wide and fixed splitting of S2 is the most characteristic sign of ASD. There is no audible murmur because of flow across the ASD, so a pansystolic murmur is not expected.

- 4 Infant 8 months old diagnosed as secundum atrial septal defect ASD, the characteristic clinical sign is:
(b) Wide fixed splitting of the 2nd heart sound
 A soft ejection systolic murmur at the left upper sternal border is also heard in ASD, but wide and fixed splitting of S2 is the most characteristic sign.
- 5 A 6-month-old child presented with respiratory distress (RD), tachycardia, difficult feeding and pansystolic...
(b) VSD
 Medium and large VSDs present with congestive heart failure (dyspnea, tachypnea, sweating and failure to gain weight) and a holosystolic murmur best heard at the left lower sternal border. In ASD there is no audible murmur because of flow across the defect.
- 6 Regarding pulmonary stenosis in children, which is false?
(d) Left ventricular hypertrophy
 Pulmonary stenosis is a right ventricular outflow obstruction and a pressure overload on the right ventricle, and hypertrophy occurs mainly in pressure overload. So the right ventricle hypertrophies, not the left.
- 7 The cardiovascular disease that is diagnosed by just palpation:
(b) Coarctation of the aorta
 Palpation of femoral pulses to detect radio-femoral delay picks up coarctation. VSD and PDA are diagnosed by their murmurs.
- 8 Differential Cyanosis is a possible complication of:
(d) Patent ductus arteriosus (PDA)
 Differential clubbing may occur in a patient with patent ductus arteriosus associated with pulmonary artery hypertension and right to left shunt, sparing the upper limbs. The PDA joins the descending aorta at the level of the left subclavian artery.
- 9 Which of the following is considered an acyanotic congenital heart disease?
(c) Ventricular septal defect.
 Acyanotic CHDs are classified to left to right shunting and obstructive lesions, and VSD is one of the left-to-right shunts. Fallot is a cyanotic CHD.
- 10 * Which of the following is the most common congenital cyanotic heart lesion in children?
(a) Fallot's tetralogy.
 The handout calls TOF one of the most common CHDs, and it is one of the two cyanotic CHDs it studies, with reduced pulmonary blood flow. The answer is inferred from these lines; the handout does not rank the cyanotic lesions.
- 11 * The most common cyanotic congenital heart disease is:
(a) Fallot tetralogy
 The handout calls TOF one of the most common CHDs, and it is one of the two cyanotic CHDs it studies, with reduced pulmonary blood flow. The answer is inferred from these lines; the handout does not rank the cyanotic lesions.
- 12 Tetralogy of Fallot consisted of the following, except:
(a) ASD.
 TOF comprises RVOTO (infundibular stenosis), VSD, aorta dextroposition (overriding aorta) and RV hypertrophy. ASD is not one of the four components.
- 13 Infant 20 months old diagnosed as Tetralogy of Fallot, which of the following is not true:
(d) Small VSD
 RV hypertrophy is one of the four components of TOF, and polycythemia is linked to cyanosis. A large VSD is the usual defect of Fallot, so 'small VSD' is the wrong statement; the handout does not give the VSD size or squatting.
- 14 Infant 20 months old diagnosed as Tetralogy of Fallot, which of the following is not true:
(b) Left ventricle hypertrophy
 TOF has RV hypertrophy, not left ventricular hypertrophy. Squatting, polycythemia and a large VSD fit a cyanotic child with Fallot.

- 15 The most common valvular regurgitation in ventricular septal defect is:
(b) AR
 Aortic insufficiency develops in approximately 5% of VSD patients, from prolapse of an aortic valve cusp into the VSD or lack of support to the aortic root.
- 16 Cyanotic spells may be a complication of:
(d) Tetralogy of Fallot
 Cyanotic spells are treated under tetralogy of Fallot: knee-chest position, oxygen, IV morphine and IV propranolol. PDA, VSD and ASD are acyanotic left-to-right shunts.
- 17 Pulmonary oligemia is seen in X-ray in the following congenital heart lesion:
(b) Tetralogy of Fallot's
 TOF is a cyanotic CHD with reduced pulmonary blood flow, seen on CXR as decreased vascularity (oligemia). VSD is a left-to-right shunt with increased pulmonary vascular markings.
- 18 The followings are cyanotic heart lesions with lung oligemia except:
(c) Transposition of the great vessels
 Diminished pulmonary vasculature represents obstruction of blood flow to the lungs, as in Fallot with reduced pulmonary blood flow. Transposition of the great vessels is a cyanotic lesion with plethoric lungs, not oligemic.
- 19 Which of the following diseases is characterized by wide and fixed splitting of S2?
(a) Atrial septal defect.
 S2 is widely split and fixed (splitting does not vary with respiration) and is the most characteristic sign of ASD.
- 20 The wide and fixed splitting of the second heart sound occurs in:
(b) Atrial septal defect
 S2 is widely split and fixed, does not vary with respiration, and is the most characteristic sign of ASD.
- 21 A large ventricular septal defect (VSD) is likely to:
(d) Be commonly associated with plethoric lung.
 A large left-to-right shunt gives increased pulmonary vascular markings. Wide and fixed splitting belongs to ASD, and cyanosis from shunt reversal (Eisenmenger) makes the patient inoperable.
- 22 6-month-old baby with PDA, which of the following is true:
(c) Continuous murmur at upper left sternal border
 PDA gives a continuous murmur heard best at the left upper sternal border and bounding pulses. Higher arm pressure and rib notching belong to coarctation.
- 23 The characteristic clinical sign of Patent ductus arteriosus is:
(c) Continuous machinery murmur at upper left sternal border
 Bounding pulses are also found in PDA, but the continuous (machinery) murmur at the left upper sternal border is its characteristic sign. Rib notching belongs to coarctation.
- 24 Patients with a small PDA have the following criteria EXCEPT:
(d) Risk for endocarditis is extremely low.
 Isolated PDA is closed mainly to prevent bacterial endocarditis, so the risk is not extremely low. A small PDA has no symptoms and is detected by a murmur, a continuous murmur at the left upper sternal border.
- 25 Which of the following is a complication of PDA?
(d) All of the above
 Moderate to large PDA may present with congestive heart failure, PDA is closed mainly to prevent bacterial endocarditis, and a right-to-left shunt from raised pulmonary vascular resistance is Eisenmenger's physiology.
- 26 Regarding pulmonary stenosis (PS), the most likely is:
(d) It causes cyanosis if critical.
 Critical pulmonary stenosis can cause cyanosis by right-to-left shunting at atrial level. The lungs are not plethoric because diminished pulmonary vasculature represents obstruction of blood flow to the lungs; the murmur is not at the apex.

- 27** The cardiovascular disease that is diagnosed by just palpation:
(b) Coarctation of the aorta
 Palpation of femoral pulses (to detect radio-femoral delay) is necessary to avoid a delay in the diagnosis of coarctation.
- 28** Which of the following is the most probable etiology?
(a) Eisenmenger
 Late cyanosis with clubbing, a loud P2 and central dilation with peripheral pruning of pulmonary arteries plus RV hypertrophy mean a right-to-left shunt in response to raised pulmonary vascular resistance: Eisenmenger. In TOF the pulmonary artery is hypoplastic, not enlarged.
- 29** What is the best treatment?
(a) Medical treatment
 Eisenmenger's syndrome makes the patient inoperable, and large VSDs with irreversible pulmonary vascular obstructive disease are not candidates for surgery; symptomatic treatment is given. Closing the defect, a shunt or prostaglandin E1 are therefore not the answer.
- 30** A female 16 years old, with shortness of breath. She has normal pulse, RR, BP, no murmurs and S2 wide fixed...
(c) Infective endocarditis
 Wide fixed splitting of S2 with dilated right heart means ASD. Infective endocarditis is rare in ASD due to low pressure gradient between the two atria, while heart failure and supra-ventricular arrhythmias are recognised.
- 31** Which is the most probable diagnosis?
(e) PDA
 A machinery murmur at the left upper sternal border with bounding pulses and no cyanosis means PDA. A big VSD gives a holosystolic murmur at the left lower sternal border.
- 32** * A full-term 4-day-old sick male baby comes with severe heart failure, metabolic acidosis and shock. On...
(d) Critical aortic coarctation
 Femoral pulses are palpated to detect coarctation, and early onset COA may reveal cardiomegaly, pulmonary edema and other signs of congestive heart failure. The answer is inferred from these lines; a large PDA gives bounding pulses instead.
- 33** Central cyanosis is not found in:
(c) Left-to-right shunt
 Central cyanosis is caused by congenital heart diseases with right to left shunt and is seen in acute pulmonary oedema. In a left-to-right shunt the oxygenated blood is shunted from left to right, so the lesion is acyanotic.
- 34** Which of the following congenital heart defects causes fixed splitting of the second heart sound?
(a) Atrial septal defect
 Fixed splitting of S2 is the most characteristic sign of ASD. In Ebstein's anomaly the second heart sound is usually normal.
- 35** * Pedal pulse is 'absent' in all except:
(c) Coarctation of aorta
 In coarctation collateral vessels connect the upper arteries to the vessels below the coarctation, and the femoral pulse shows radio-femoral delay. Embolism can make pulsations absent, and Leriche syndrome narrows the terminal abdominal aorta; the answer is inferred from these lines.

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

- 1** 9 months old baby with small muscular VSD, which of the following is not true:
(a) Heart failure is common
 In small VSDs the patients are usually asymptomatic and the only abnormality is a loud murmur at the left lower sternal border; heart failure belongs to medium and large defects. Endocarditis is more common in small than in big VSD.

2 9 months old infant with small VSD, which of the following is true?

(a) Loud pansystolic murmur over the left parasternal border

In small VSDs the patients are usually asymptomatic and the only abnormality is a loud murmur at the left lower sternal border. Heart failure and chest infection belong to medium and large defects, and pulmonary vascular obstructive disease follows high pressure and high flow.

3 3 years old boy with aortic coarctation, which of the following is not true:

(d) Recurrent lower respiratory infections

Radio-femoral delay, upper limb hypertension and rib notching are features of coarctation. Respiratory infections belong to medium and large left-to-right shunts such as VSD.

4 * 10 months old baby with large muscular VSD, which of the following is false:

(b) Loud harsh pansystolic murmur

Respiratory infection, heart failure and a loud single S2 when PAP is elevated are expected in medium and large VSDs. The answer is inferred from 'The intensity of the murmur may vary between grades II-V/VI', so the murmur of a large defect is not always loud.

5 All of the following are true about pulmonary stenosis except:

(d) Accentuated second heart sound

A loud P2 is a sign of pulmonary hypertension, not of pulmonary stenosis, where S2 is soft. Pulmonary stenosis is a pressure overload that causes right ventricular hypertrophy.

Model Answers | Essay Questions

1 Enumerate four complications of ventricular septal defect.

- ① **Congestive heart failure:** patients with medium and large defects may present with symptoms of congestive heart failure (dyspnea, tachypnea, sweating and failure to gain weight) or with symptoms related to bronchial obstruction and/or respiratory infection.
- ② **Pulmonary vascular obstructive disease**
 - May develop in **10%** of VSDs.
 - This is probably related to the exposure of the pulmonary vascular bed to high pressure and high flow.
- ③ **Development of infundibular stenosis "Gasul's transformation of the VSD"**
 - May occur in **8%** of the defects.
 - It is more with right aortic arch and increased angle of the right ventricular outflow tract.
 - While development of infundibular stenosis eventually requires the patient to have surgery, it indeed protects the pulmonary vascular bed and prevents development of pulmonary vascular obstruction disease.
- ④ **Aortic insufficiency**
 - Develops in approximately **5%** of patients.
 - This may either be related to prolapse of an aortic valve cusp into the VSD or lack of support to the aortic root.
 - More with supracristal VSDs.
 - Surgical correction is indicated if moderate to severe aortic insufficiency is present.
- ⑤ **Eisenmenger's complex:** flow reversal across a VSD when pulmonary vascular resistance exceeds systemic levels.
- ⑥ **Infective endocarditis:** congenital anomalies, esp. VSD, PDA and coarctation.
 - Narrow orifice: more common in small than in big VSD.

2 Describe the complications of Fallot's tetralogy.

- ① The mortality rate in untreated patients reaches **50%** by age 6 years.
- ② **Syncope.**
 - Hypoxic syncope: Fallot's tetralogy and other cyanotic diseases.
- ③ **Right-sided heart failure:** elevated jugular venous pressure [JVP] with large A wave, ascites, peripheral edema, hepatomegaly.
- ④ **Pulmonary valve insufficiency "PR":** low-pitched, short diastolic murmur.
- ⑤ A mild **anemia** may be present; polycythemia is rare in the absence of cyanosis.
- ⑥ Coagulation profile may be abnormal in patients with cyanosis and **bleeding**.
- ⑦ **Endocarditis or sepsis:** obtain blood cultures in febrile patients to rule out endocarditis or sepsis.
- ⑧ **Cyanotic spells** (adults with acute cyanosis).
- ⑨ **Hyperviscosity** in a nondehydrated state, most commonly when hematocrit levels > **65-70**; if symptoms occur with hematocrits < 65, evidence of iron deficiency should be sought.
- ⑩ **Cerebral arterial thrombosis:** prophylactic phlebotomy has no place in the prevention of cerebral arterial thrombosis.

Note The handout gives no separate list of Fallot complications; the points are gathered from its pages.

3 Mention five complications of patent ductus arteriosus (PDA) and PDA diagnosis in pediatrics.

Complications

- ① **Congestive heart failure:** moderate to large PDA may either present with symptoms of easy fatigability, symptoms associated congestive heart failure.
- ② **Bacterial endocarditis:** isolated PDA is an indication for trans-catheter device closure by cardiologists, mainly to prevent bacterial endocarditis.
 - Infective endocarditis, underlying cardiac lesion: congenital anomalies, esp. VSD, PDA and coarctation.
- ③ **Hyperdynamic pulmonary hypertension:** it is due to increased pulmonary arterial blood flow; it occurs in congenital heart disease with left to right shunt e.g. ASD, VSD & PDA.
- ④ **Eisenmenger's physiology:** a right-to-left shunt occurs in response to an elevation in pulmonary vascular resistance.
- ⑤ **Differential clubbing:** occasionally, clubbing may occur in lower limbs, sparing the upper limbs; it may occur in a patient with patent ductus arteriosus associated with pulmonary artery hypertension and right to left shunt.

Diagnosis

Clinical presentation depends upon the size of the ductus.

- * If the PDA is small, there are no symptoms, and it is usually detected because of a murmur detected on a routine examination.
- * Moderate to large PDA may either present with symptoms of easy fatigability, symptoms associated **congestive heart failure**.

Physical findings

- * Left ventricular impulse is normal in small PDA and may be hyperdynamic with large shunts.
- * A thrill may be felt at the left upper sternal border and in the suprasternal notch.
- * S1 is usually normal and S2 may be buried within the murmur.
- * In most cases, a **continuous murmur** is heard best at the **left upper sternal border**. The murmur begins in systole and continues through S2 into the diastole. The systolic component of the murmur crescendos up to S2 while the diastolic part decrescendo to a varying distance (time) into the diastole → **machinery murmur**.

- * In big PDA, a mid-diastolic murmur may be heard at the apex because of increased flow across the mitral valve; such a mid-diastolic murmur suggests a Qp:Qs greater than **2:1** (Pulmonary to systemic flow = Qp:Qs).
- * Arterial pulses are **bounding** in all but patients with very small ductus.

Investigations

	Findings
Chest x-ray	If moderate to large PDA, cardiomegaly, increased pulmonary blood flow and left atrial enlargement may be seen.
Electrocardiogram	The ECG may be normal or may show left atrial and left ventricular enlargement, depending upon the size of the ductus.
Echocardiogram	Doppler echocardiography shows characteristic diastolic flow pattern in the pulmonary artery, indicative of PDA. Also determine the size of PDA.
Cardiac catheterization and selective cine angiography	Integral part of transcatheter closure. Selective aortic arch injection demonstrates the size, shape, and location of the ductus.

Note The handout gives no separate list of PDA complications; the points are gathered from several pages.

4 Enumerate 3 complications of patent ductus arteriosus.

- 1 **Congestive heart failure:** moderate to large PDA may either present with symptoms of easy fatigability, symptoms associated congestive heart failure.
- 2 **Bacterial endocarditis:** isolated PDA is an indication for trans-catheter device closure by cardiologists, mainly to prevent bacterial endocarditis.
 - Infective endocarditis, underlying cardiac lesion: congenital anomalies, esp. VSD, PDA and coarctation.
- 3 **Hyperdynamic pulmonary hypertension:** it is due to increased pulmonary arterial blood flow; it occurs in congenital heart disease with left to right shunt e.g. ASD, VSD & PDA.
- 4 **Eisenmenger's physiology:** a right-to-left shunt occurs in response to an elevation in pulmonary vascular resistance.
- 5 **Differential clubbing:** occasionally, clubbing may occur in lower limbs, sparing the upper limbs; it may occur in a patient with patent ductus arteriosus associated with pulmonary artery hypertension and right to left shunt.

Note The handout gives no separate list of PDA complications; the points are gathered from several pages.

5 Discuss treatment of patent ductus arteriosus (PDA).

- * Isolated PDA is an indication for **trans-catheter device closure** by cardiologists, mainly to prevent bacterial endocarditis.
- * If the patient is asymptomatic, waiting until **6-12 months** of age.
- * For large-sized PDA, **surgical closure** is available option.
- * If so small PDA, but no continuous murmur on auscultation → called "**silent ductus**" may no need for intervention.

6 Mention clinical picture (CP) and treatment of PDA.

Clinical picture

Symptoms

Clinical presentation depends upon the size of the ductus.

- * If the PDA is small, there are no symptoms, and it is usually detected because of a murmur detected on a routine examination.
- * Moderate to large PDA may either present with symptoms of easy fatigability, symptoms associated **congestive heart failure**.

Physical findings

- * Left ventricular impulse is normal in small PDA and may be hyperdynamic with large shunts.
- * A thrill may be felt at the left upper sternal border and in the suprasternal notch.
- * S1 is usually normal and S2 may be buried within the murmur.
- * In most cases, a **continuous murmur** is heard best at the **left upper sternal border**. The murmur begins in systole and continues through S2 into the diastole. The systolic component of the murmur crescendos up to S2 while the diastolic part decrescendo to a varying distance (time) into the diastole → **machinery murmur**.
- * In big PDA, a mid-diastolic murmur may be heard at the apex because of increased flow across the mitral valve; such a mid-diastolic murmur suggests a Qp:Qs greater than **2:1** (Pulmonary to systemic flow = Qp:Qs).
- * Arterial pulses are **bounding** in all but patients with very small ductus.

Treatment (Management)

- * Isolated PDA is an indication for **trans-catheter device closure** by cardiologists, mainly to prevent bacterial endocarditis.
- * If the patient is asymptomatic, waiting until **6-12 months** of age.
- * For large-sized PDA, **surgical closure** is available option.
- * If so small PDA, but no continuous murmur on auscultation → called "**silent ductus**" may no need for intervention.

7

Clarify hemodynamics, clinical picture (CP) and complications of TOF.

Hemodynamics

TOF is one of the most common CHDs, comprises:

- ① Right ventricular (RV) outflow tract obstruction (RVOTO) (infundibular stenosis).
- ② Ventricular septal defect (VSD).
- ③ Aorta dextroposition (overriding aorta).
- ④ And RV hypertrophy.

Cyanotic CHD with **reduced pulmonary blood flow**.

Clinical picture

Signs and symptoms

Signs and symptoms are directly related to the severity of the anatomic defects and may include the following:

- ① Lack of exercise endurance.
- ② Syncope.
- ③ Palpitations.
- ④ Exertional dyspnea, usually worsening with age.

Physical findings in adults

- * Evidence of **right-sided heart failure** (elevated jugular venous pressure [JVP] with large A wave, ascites, peripheral edema, hepatomegaly).
- * In individuals with pulmonary valve insufficiency "PR": low-pitched, short diastolic murmur.

Complications

- ① The mortality rate in untreated patients reaches **50%** by age 6 years.
- ② **Syncope.**
 - Hypoxic syncope: Fallot's tetralogy and other cyanotic diseases.
- ③ **Right-sided heart failure:** elevated jugular venous pressure [JVP] with large A wave, ascites, peripheral edema, hepatomegaly.
- ④ **Pulmonary valve insufficiency "PR":** low-pitched, short diastolic murmur.
- ⑤ A mild **anemia** may be present; polycythemia is rare in the absence of cyanosis.
- ⑥ Coagulation profile may be abnormal in patients with cyanosis and **bleeding**.
- ⑦ **Endocarditis or sepsis:** obtain blood cultures in febrile patients to rule out endocarditis or sepsis.
- ⑧ **Cyanotic spells** (adults with acute cyanosis).
- ⑨ **Hyperviscosity** in a nondehydrated state, most commonly when hematocrit levels > **65-70**; if symptoms occur with hematocrits < 65, evidence of iron deficiency should be sought.
- ⑩ **Cerebral arterial thrombosis:** prophylactic phlebotomy has no place in the prevention of cerebral arterial thrombosis.

Note The handout does not explain the haemodynamics of Fallot or list its complications; points are gathered from its pages.

- 8** Mention the clinical picture (CP), investigations and complications of the following: TOF, aortic coarctation.

(a) Tetralogy of Fallot

Clinical picture

Signs and symptoms

Signs and symptoms are directly related to the severity of the anatomic defects and may include the following:

- ① Lack of exercise endurance.
- ② Syncope.
- ③ Palpitations.
- ④ Exertional dyspnea, usually worsening with age.

Physical findings in adults

- * Evidence of **right-sided heart failure** (elevated jugular venous pressure [JVP] with large A wave, ascites, peripheral edema, hepatomegaly).
- * In individuals with pulmonary valve insufficiency "PR": low-pitched, short diastolic murmur.

Investigations

In adult patients, laboratory studies may be helpful:

	Finding / use
Complete blood cell count	A mild anemia may be present; polycythemia is rare in the absence of cyanosis.
Coagulation profile	May be abnormal in patients with cyanosis and bleeding.
Arterial blood gas (including serum lactate, base excess, oxygen partial pressure [PO₂])	The patient's oxygenation status, serum lactate levels, and base excess value appear to be prognostic markers for mortality in repaired TOF.
Blood cultures	Obtain blood cultures in febrile patients to rule out endocarditis or sepsis.

	Findings
CXR	May be normal or depict cardiomegaly and prominent RV shadowing. Although the classic boot-shaped heart (coeur en sabot) is the hallmark of the disorder in infants, this shape of the heart may not be seen in adult patients.
Echocardiography	Color-flow Doppler echocardiography: assesses overall cardiac function and the status of the valves, and for the presence of any residual VSD, ductus arteriosus, or atrial septal defect. Reveals the grade and severity of any RVOT obstruction.
MRI	Gold standard for assessing RV function and size, and for quantifying the pulmonary regurgitant volume; provides good delineation of the aorta size and assessment of the pulmonary arteries and its branches, the status of the RVOT, and the presence of VSDs, and/or RV hypertrophy.
Cardiac catheterization	Allows: assessment of the pulmonary annulus size and pulmonary arteries; assessment of the severity of RVOTO; location of the position and size of the VSD; ruling out possible coronary artery anomalies.

Complications

- ① The mortality rate in untreated patients reaches **50%** by age 6 years.
- ② **Syncope.**
 - Hypoxic syncope: Fallot's tetralogy and other cyanotic diseases.
- ③ **Right-sided heart failure:** elevated jugular venous pressure [JVP] with large A wave, ascites, peripheral edema, hepatomegaly.
- ④ **Pulmonary valve insufficiency "PR":** low-pitched, short diastolic murmur.
- ⑤ A mild **anemia** may be present; polycythemia is rare in the absence of cyanosis.
- ⑥ Coagulation profile may be abnormal in patients with cyanosis and **bleeding.**
- ⑦ **Endocarditis or sepsis:** obtain blood cultures in febrile patients to rule out endocarditis or sepsis.
- ⑧ **Cyanotic spells** (adults with acute cyanosis).
- ⑨ **Hyperviscosity** in a nondehydrated state, most commonly when hematocrit levels > **65-70**; if symptoms occur with hematocrits < 65, evidence of iron deficiency should be sought.
- ⑩ **Cerebral arterial thrombosis:** prophylactic phlebotomy has no place in the prevention of cerebral arterial thrombosis.

(b) Aortic coarctation

Clinical picture

In adolescents and adults, the diagnosis should always be sought in the event of unexplained hypertension of the upper extremities.

Symptoms

- * Presenting symptoms may include headaches, chest pain, fatigue, or even life-threatening intracranial hemorrhage.
- * True claudication is rare.
- * Many patients are asymptomatic except for the incidentally noted hypertension.
- * Frequently, COA is not recognized by the primary care physician.

Pulse & blood pressure

- * Palpation of femoral pulses (to detect **radio-femoral delay**), and measurement of blood pressure during routine examination is necessary to avoid a delay in the diagnosis.
- * A pressure difference of more than **20 mm Hg** in favor of the arms may be considered evidence of coarctation of the aorta.

Auscultation

- * A murmur in the left infraclavicular area and under the left scapula may be systolic, but the murmur may also sound continuous in the presence of multiple collateral vessels or, occasionally, severe coarctation.
- * An ejection click may be audible when an associated bicuspid aortic valve and a murmur of aortic stenosis or insufficiency are present.
- * Similarly, a murmur of mitral stenosis or LV outflow tract obstruction may also occur.
- * A gallop rhythm may occur in the presence of a hypertrophic noncompliant LV.

Other findings on physical examination

- * Abnormalities of blood vessels in the retina and a prominent suprasternal notch pulsation.
- * A thrill may be present in the suprasternal notch or on the precordium in the presence of significant aortic valve stenosis.
- * In the rare case of abdominal coarctation, an abdominal bruit may be noted.

Investigations

	Findings
ECG	May be normal or may reveal LV hypertrophy and may show signs of LV ischemia or strain. Sometimes, LV hypertrophy may manifest as increased S waves in leads V5 and V6, the so-called posterobasal LV hypertrophy.
CXR	With early onset of COA may reveal cardiomegaly, pulmonary edema, and other signs of congestive heart failure (CHF); with late onset COA may reveal cardiomegaly; an inverted "3" sign of the barium-filled esophagus or a "3" sign on a highly penetrated CXR (frontal view) may be visualized; rib notching secondary to collateral vessels may also be seen.
Echocardiography	The suprasternal notch 2-D view allows evaluation of the aortic arch to assess the transverse aortic arch, isthmus, and severity of coarctation and to measure the pressure gradient "PG" at the site of COA.
MRI and CT	Are useful in older or postoperative patients to assess residual arch obstruction, arch hypoplasia, or formation of aneurysms.

Complications

- ① Complications of COA include: secondary **LVH**, systemic **HTN**, **HF**, **aortic dissection**, premature **CAD**, and **cerebrovascular complications**.
- ② Some patients with COA may have **cerebral aneurysms**, predisposing them to cerebrovascular accidents with severe hypertension later in life.
- ③ With untreated coarctation a mean survival of **35 years** with a 75% mortality by age 50 years can be expected.

Note The handout gives no separate list of Fallot complications; that part is gathered from its pages.

9 Describe treatment of the following congenital cardiac defects: TOF, PDA, VSD, TGA.

(a) Tetralogy of Fallot

Cyanotic spells

For adults with acute cyanosis:

- * Place them in a **knee-chest position**.
- * In addition, provide/administer **Oxygen**, **IV morphine** and **IV propranolol** for severe cases.

Surgery

- * Most symptomatic adults with TOF require some type of surgical intervention.
- * **Pulmonary valve replacement** is the most common procedure performed.

Phlebotomy

N.B. Phlebotomy is indicated under two sets of circumstances: patients symptomatic of hyperviscosity in a nondehydrated state, most commonly when hematocrit levels > 65-70; in asymptomatic patients with hematocrit levels > 65 before surgery to minimize postoperative bleeding.

- * If symptoms occur with hematocrits < 65, evidence of iron deficiency should be sought.
- * When indicated, removal of **500 mL** of blood over 30-45 minutes followed by quantitative volume replacement with normal saline or dextran can be performed.
- * The procedure may be repeated every 24 hours until improvement occurs.
- * An attempt should be made to achieve symptom control with the smallest quantity of blood removal to avoid iron deficiency.
- * Prophylactic phlebotomy has no place in the prevention of cerebral arterial thrombosis.

(b) PDA

- * Isolated PDA is an indication for **trans-catheter device closure** by cardiologists, mainly to prevent bacterial endocarditis.
- * If the patient is asymptomatic, waiting until **6-12 months** of age.
- * For large-sized PDA, **surgical closure** is available option.
- * If so small PDA, but no continuous murmur on auscultation → called "**silent ductus**" may no need for intervention.

(c) VSD

Strategies depend, to a large degree, on the size of the VSD.

Defect size	What to do
In small VSDs	Reassurance and follow up.
In moderate-sized defects	Treatment of heart failure if present, should be undertaken. Markedly enlarged left ventricle are probably indications for surgical closure.
In very large defects	The heart failure should be treated aggressively. If the congestive heart failure is difficult to control with medics, surgical closure should be undertaken.

Resistance	What to do
When the pulmonary vascular resistance is elevated	Its response to oxygen and other vasodilator agents (NO) may be necessary to determine the suitability for surgical closure; patients with calculated pulmonary vascular resistance < 8 wood units with a Qp: Qs > 1.5 are generally considered suitable candidates for surgery; if the resistance drops < 8 units after administering oxygen or other vasodilator agents, the patient becomes a candidate for surgery.
Large VSDs with severe elevation of pulmonary resistance	(Irreversible pulmonary vascular obstructive disease) are not candidates for surgery. Symptomatic treatment and erythropheresis for symptoms of polycythemia should be undertaken. These patients may eventually become candidates for lung transplantation.

- * When surgery is indicated, **open heart surgical technique** is the treatment of choice.
- * **Transcatheter device occlusion** of VSD is amenable now for some types as muscular and peri-membranous types with sufficient septum in the subaortic region so that the device can be implanted without interfering with aortic valve function.
- * Aortic insufficiency: surgical correction is indicated if moderate to severe aortic insufficiency is present.

Note The handout does not cover transposition of the great arteries, so that part is not answered.

10 Classification of congenital heart diseases.

The simplest way is to classify them as **Cyanotic** and **Acyanotic** CHDs.

- * **Cyanotic CHD:** our scope in Cyanotic CHD is on Ebstein anomaly and Fallot Tetralogy only, both with reduced pulmonary blood flow.
 - Ebstein's Anomaly.
 - Tetralogy of Fallot "TOF".
- * **Acyanotic CHDs:** further classified to Left to right shunting and obstructive lesions.
 - Acyanotic heart defects: Left-to-right shunts
 - Atrial Septal Defect (ASD).
 - Ventricular Septal Defect (VSD).
 - Patent Ductus Arteriosus "PDA".
 - Acyanotic Obstructive lesions
 - Pulmonary Stenosis (RVOTO).
 - Aortic Stenosis "AS" (LVOTO).
 - Aortic Coarctation "COA".

11 Using the stethoscope only, mention the diagnostic findings of the following: ASD, VSD, PDA.

(a) ASD

- * S2 is **widely split and fixed** (splitting does not vary with respiration) and is the most characteristic sign of ASD.
- * A soft ejection systolic murmur of grade I-II/VI intensity (due to increased flow across the pulmonary valve and is heard best at the left upper sternal border).
- * A grade I-II/VI mid-diastolic flow rumble is heard (with the bell of the stethoscope) best at the left lower sternal border. This is due to large volume flow across the tricuspid valve.
- * There is no audible murmur because of flow across the ASD.

(b) VSD

- ① In very small defects, murmur, though begins with first heart sound, may not last through the entire systole; the shorter the murmur, the smaller is the defect.
- ② In small defects the only abnormality is a loud Ejection systolic murmur heard best at the left lower sternal border.
- ③ In medium and large defects:
 - S2 is split unless elevated PAP "pulmonary artery pressure", in which case a loud single S2.
 - A **holosystolic murmur** is best heard at the left lower sternal border and does not usually radiate although it may be heard widely over the precordium.
 - The intensity of the murmur may vary between grades II-V/VI.
 - There is no significant variation of this murmur with respiration.
- ④ A grade I-II/VI mid-diastolic flow rumble may be heard at the apex in patients with medium to large-sized VSD and large left-to-right shunts; this murmur is heard best with the bell of the stethoscope. The mid diastolic murmur is due to increased flow across the mitral valve and usually indicates a Qp: Qs greater than **2:1**.

(c) PDA

- * S1 is usually normal and S2 may be buried within the murmur.
- * In most cases, a **continuous murmur** is heard best at the left upper sternal border. The murmur begins in systole and continues through S2 into the diastole. The systolic component of the murmur crescendos up to S2 while the diastolic part decrescendo to a varying distance (time) into the diastole → **machinery murmur**.
- * In big PDA, a mid-diastolic murmur may be heard at the apex because of increased flow across the mitral valve, such a mid-diastolic murmur suggests a Qp:Qs greater than 2:1.

Valvular Heart Disease

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	7	8	9	10	11	12
Answer	b	b	d	a	c	b	b	a	a	b	b	a

Question no.	13	14	15	16	17	18	19	20	21	22	23	24
Answer	b	c	b	c	d	b	b	a	d	e	b	a

Question no.	25	26	27	28	29	30	31	32	33	34	35	36
Answer	c	a+c+d	b	c	a	c	c	b	c	d	b	b

Question no.	37	38	39	40	41	42	43	44	45
Answer	a	b	b	a	d	b	a	d	b

► Second | Questions with a Note

Question no.	1	2	3	4
Answer	d	b	d	c

► Why this answer?

1 The following investigations are used in diagnosis of valvular heart lesions except:

(b) CT chest

Trans-thoracic and trans-esophageal echo and coronary angiography before open heart surgery are the investigations used. CT chest is not one of them; only a CT aortogram is used, in patients with aortic aneurysm.

2 Apical systolic murmur is not due to:

(b) Aortic regurgitation

The murmur of aortic regurgitation is a soft blowing decrescendo (diastolic) murmur best heard at the second aortic area. MR gives a holosystolic murmur on the apex, and the aortic stenosis murmur is propagated to the neck and to the apex.

- 3 *Heaving apex is found in the following lesion:*
(d) Aortic stenosis.
 The apex is sustained (heaving) in left ventricular hypertrophy and hyperdynamic in dilatation; aortic stenosis gives signs of left ventricular hypertrophy with a sustained apex. Aortic regurgitation gives a hyperdynamic apex.
- 4 *Diastolic thrill over the apex is usually due to:*
(a) Mitral stenosis
 A palpable diastolic thrill at the apex is a sign of mitral stenosis. MR gives a palpable systolic thrill at the apex, and aortic stenosis a systolic thrill at the first aortic area.
- 5 *Which of the following is an indication of aortic valve replacement:*
(c) Severe aortic stenosis and severe aortic regurgitation.
 Surgical aortic valve replacement is indicated in severe aortic stenosis and in severe aortic regurgitation. Mild and moderate aortic lesions are not indications.
- 6 *Aortic valve surgery is indicated in:*
(b) Severe aortic stenosis
 Aortic valve replacement (SAVR or TAVR) is indicated in severe aortic stenosis. The other options are mitral lesions.
- 7 *Mitral valve surgery is indicated in:*
(b) Severe mitral stenosis
 Mitral valve intervention (PBMV or surgical replacement) is indicated when the mitral valve area is $< 1.5 \text{ cm}^2$, and severe MS is an area $< 1 \text{ cm}^2$. The other options are aortic lesions.
- 8 *All are true about mechanical valve except:*
(a) Long-term anticoagulant and warfarin is not needed
 A metallic (mechanical) prosthesis needs warfarin for life with INR 2.5-3.5, so (a) is false. Lifelong warfarin brings a bleeding risk, and the valve carries a clot risk, while its durability is long.
- 9 *All are false about bioprosthetic valve except:*
(a) Long-term anticoagulant and warfarin is not needed
 A bio prosthesis needs warfarin for 3-6 months, then antiplatelet therapy, so long-term warfarin is not needed. Its durability is short and its risks of thromboembolism and bleeding are low.
- 10 *Aortic valve surgery is indicated in:*
(b) Severe aortic regurgitation
 Surgical aortic valve replacement is indicated in severe aortic regurgitation. Mild or moderate aortic stenosis and mitral lesions are not indications.
- 11 ** All are true about mechanical valve except:*
(b) Decreased durability
 A mechanical valve is durable, so 'decreased durability' is false. It needs warfarin for life, which brings its clot and bleeding risks; durability is inferred from the line 'If metallic prosthesis: warfarin for life', as the handout does not state it.
- 12 *All are true regarding bioprosthetic valves except:*
(a) Long-term anticoagulant is needed
 A bio prosthesis needs warfarin for 3-6 months, then antiplatelet therapy, not long-term anticoagulation. It is less durable and has less risk of thromboembolism and bleeding.
- 13 *Mitral valve surgery is indicated in:*
(b) Severe mitral regurgitation
 Intervention or surgery is indicated in severe MR, symptomatic or asymptomatic with EF $< 60\%$ and/or a dilated LV. Moderate MR is not an indication, and aortic stenosis is treated on the aortic valve.
- 14 *Which of the following is indicated for mitral valve surgery:*
(c) Severe mitral stenosis and severe mitral regurgitation.
 Mitral valve intervention is indicated in MS with a valve area $< 1.5 \text{ cm}^2$ and in severe MR. Mild lesions and moderate MR are not indications.

- 15 *The worst prognosis in aortic stenosis, when the patient presents with:*
(b) Heart failure
 Average survival in symptomatic aortic stenosis is 2 years with heart failure, 3 years with syncope and 5 years with chest pain, so heart failure has the worst prognosis.
- 16 *All of the following are not indications for aortic valve surgery, except:*
(c) Severe aortic stenosis and severe aortic regurgitation.
 Aortic valve replacement is indicated in severe aortic stenosis and severe aortic regurgitation. In every other option the aortic lesion is mild.
- 17 *As regards surgical options of severe mitral regurgitation, all the following are true except:*
(d) Percutaneous balloon mitral valvuloplasty
 Percutaneous balloon valvuloplasty is the intervention for mitral stenosis, not regurgitation. MR is treated by surgical repair or by replacement with a bio or metallic prosthesis.
- 18 *As regards surgical options of mitral stenosis, all of the following are true except:*
(b) Transcatheter aortic valve implantation (TAVI).
 Transcatheter aortic valve replacement is a treatment of severe aortic stenosis, not of the mitral valve. Mitral stenosis is treated by PBMV or surgical mitral valve replacement.
- 19 *A 59-year-old M, with breathlessness, auscultation demonstrates systolic murmur at base of heart. Which one...*
(b) Left ventricular enlargement
 A systolic murmur at the base (first aortic area) is the murmur of aortic stenosis. The ECG in aortic stenosis shows left ventricular hypertrophy.
- 20 *A 55-year-old F presented with exertional dyspnea. Chest X-ray showed large pulmonary arteries & left atrial...*
(a) MS
 Prominent main pulmonary arteries and an enlarged left atrium on chest X-ray are findings of mitral stenosis. Mitral stenosis leads to pulmonary hypertension with right ventricular strain, which fits the right-sided ECG signs; AS, AR and low EF load the left ventricle.
- 21 ** TR can be due to:*
(d) All of above
 Congenital heart disease and infective endocarditis are listed causes of tricuspid regurgitation. Inferior wall infarction is not named; it is inferred from the cause 'Functional (RV dilatation...)', as an infarction reaching the right ventricle dilates it.
- 22 *A 5-year-old boy, asymptomatic. On a routine health check, his doctor accidentally auscultated a systolic...*
(e) AR
 The murmur of aortic regurgitation is diastolic. MR gives a holosystolic murmur and AS a systolic murmur; VSD and PS also give systolic murmurs.
- 23 *If the patient has mitral stenosis as a cause of his heart failure symptoms, which of the following drugs...*
(b) Isosorbide dinitrate
 Pulmonary congestion in mitral stenosis is controlled with diuretics and beta-blockers, which slow the heart rate and increase the diastolic time; diltiazem also slows the rate. A nitrate lowers the filling pressure while the narrow valve fixes the output, so it can cause marked hypotension.
- 24 *If the patient presented with acute mitral regurge, which of the following is typically present:*
(a) Coarse crepitations over his back
 Acute MR presents with bilateral coarse crepitations of acute pulmonary edema. Bedside echo shows no left ventricular dilatation, so the apex is not shifted; marked lower limb edema belongs to right sided heart failure in chronic disease.
- 25 ** Tricuspid stenosis is a rare cause of systemic congestion. Which of the following supports its diagnosis?*
(c) Diastolic hepatic pulsations
 Tricuspid stenosis gives a giant a wave and slow y descent in the JVP; the a wave comes with atrial contraction at the end of diastole, so the liver pulsation is diastolic. This is inferred from the line 'Giant a wave and slow y descent in JVP'; systolic expansion of neck veins and a pulsatile liver belong to tricuspid regurgitation.

- 26** Aortic stenosis is a cause of increased afterload on the left ventricle. Before failure of compensation, it...
(a) Sustained apex + (c) Systolic murmur on the apex + (d) Systolic thrill on the neck
 In compensated aortic stenosis the left ventricle hypertrophies without dilating, so the apex is sustained but localized and not shifted. The handout says the aortic stenosis murmur is propagated to the neck and to the apex, and a carotid (neck) thrill could be felt. A shifted apex down and out means dilatation, which comes only after failure of compensation.
- 27** Acute severe mitral regurg could manifest as:
(b) Coarse crepitations over his back.
 Acute severe MR presents with tachycardia, hypotension and bilateral coarse crepitations of acute pulmonary edema. There is no left ventricular dilatation, so no cardiomegaly, and marked lower limb edema is a sign of right sided heart failure.
- 28** What is the most probable diagnosis:
(c) Congestive heart failure on top of mitral valve disease.
 Thickening, fibrosis and restricted mobility of the leaflets mean primary MR (structural damage of the leaflets), not secondary MR, which has no structural problem with the valve. Pulmonary and systemic congestion together mean left plus right sided failure, 'Lt. to Rt. = CHF'.
- 29** What is the most probable cause of the patient's condition?
(a) Rheumatic heart disease
 Rheumatic fever causes primary MR mostly combined with mitral stenosis, and rheumatic mitral stenosis is more common in females. Dilated and ischemic cardiomyopathy give secondary MR with no structural problem of the valve.
- 30** Which of the following could predict higher risk of stroke in this patient?
(c) Dilated left atrium.
 As left atrial pressure remains elevated the left atrium increases in size, with a greater chance of atrial fibrillation. A dilated left atrium > 50 mm is itself an indication for oral anticoagulation.
- 31** The patient has mild mitral stenosis. This means that the mitral valve area is:
(c) 1.5 - 4 cm²
 Mild MS is a mitral valve area > 1.5 cm², moderate 1-1.5 cm² and severe < 1 cm²; the normal area is 4-6 cm².
- 32** The most common cause of the patient presentation is:
(b) Rheumatic heart disease.
 A diastolic rumbling murmur on the apex with an accentuated P2 means mitral stenosis with pulmonary hypertension. Rheumatic fever is the most common cause of mitral stenosis.
- 33** What is the expected character of the patient's apex?
(c) Hyperdynamic.
 An apex shifted down and outward with a palpable systolic thrill and an apical systolic murmur means MR with signs of LV dilatation, and the apex is hyperdynamic. A sustained apex is the sign of left ventricular hypertrophy in aortic stenosis.
- 34** Which of the following is not an option of management of such patient?
(d) Aortic valve replacement.
 The patient has mitral and tricuspid regurgitation with no aortic lesion, so aortic valve replacement has no place. Coronary angiography before open heart surgery, mitral valve replacement and tricuspid valve repair are all options.
- 35** Which of the following clinical signs is most commonly associated with mitral stenosis?
(b) Loud S1 with an opening snap and mid-diastolic murmur
 Mitral stenosis gives an accentuated 1st heart sound and a diastolic rumbling murmur on the apex. A systolic murmur at the aortic area is aortic stenosis and a continuous machinery murmur is PDA.
- 36** What is the most likely cause of the patient's stroke?
(b) Cardioembolism from atrial fibrillation
 In mitral stenosis the dilated left atrium gives a greater chance of AF, and embolic stroke could occur secondary to AF, resulting in hemiplegia. She was not taking the anticoagulation that AF requires.

- 37 Which of the following echocardiographic findings is most characteristic of severe mitral stenosis?
(a) Mitral valve area <1 cm²
 Severe MS is a mitral valve area < 1 cm² on echocardiography.
- 38 The least common complication of MS is:
(b) Subacute bacterial endocarditis
 Infective endocarditis is not common in MS because of marked fibrosis and low pressure. Pulmonary hypertension, atrial fibrillation and stroke are the usual complications.
- 39 * All of the following clinical findings are consistent with severe mitral stenosis except:
(b) Opening snap late after S2
 In severe mitral stenosis the left atrial pressure is high, so the valve opens early and the opening snap comes soon after S2, not late; this is inferred from that line, as the handout does not give the timing of the snap. AF and pulmonary congestion and a pulsatile liver from associated tricuspid regurgitation all fit severe MS.
- 40 Which of the following parameters is typically reduced in chronic severe aortic regurgitation?
(a) Diastolic blood pressure
 Chronic AR gives a wide pulse pressure with increased systolic and decreased diastolic pressure. The left ventricular preload increases and the ventricle dilates.
- 41 What is the most common cause of obstruction to left ventricular inflow?
(d) Rheumatic mitral disease
 Obstruction to left ventricular inflow is mitral stenosis, and rheumatic fever is its most common cause. Degenerative annular calcification and congenital causes are uncommon causes.
- 42 In cases of severe mitral stenosis, which of the following parameters is typically increased?
(b) Left atrial pressure
 Mitral stenosis causes an increase in left atrial pressure, while the cardiac output decreases as it progresses.
- 43 Which cardiac valvular lesion is most common in patients with ankylosing spondylitis?
(a) Aortic regurgitation
 Ankylosing spondylitis is an inflammatory cause of primary aortic root disease (annular dilatation), which causes aortic regurgitation.
- 44 * Mitral stenosis is frequently complicated by pulmonary hypertension. Which of the following is a cause of...
(d) All of the above
 The raised left atrial pressure is transmitted to the pulmonary vasculature, and long standing passive pulmonary hypertension leads to pulmonary arteriolar vasoconstriction. Edema in the walls of the small vessels is inferred from the pulmonary congestion of mitral stenosis, as the handout does not state it.
- 45 Most common cardiac lesion in carcinoid syndrome is:
(b) Tricuspid incompetence
 Carcinoid syndrome is listed among the causes of tricuspid regurgitation; it is not named as a cause of the other lesions.

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

- 1 Which of the following investigations are needed for valvular heart diseases:
(d) All the above.
 Echocardiography, trans-thoracic and trans-esophageal, assesses the valve lesion, and coronary angiography is done in patients with suspected ischemic heart disease before open heart surgery. So all the above are used.
- 2 Which of the following diseases is characterized by localized and shifted down apex?
(b) Aortic stenosis
 A localized apex directed outwards and downwards is a sign of left ventricular enlargement, while right ventricular enlargement gives a diffuse apex, so aortic stenosis is the only left-sided lesion offered. In aortic stenosis itself the handout gives the apex as localized and not shifted.

- 3 A 49-year-old F with aortic root dilatation & AR. Which one of the following is the least likely indication...

(d) Dilated LV dimensions

In severe AR, symptoms, LVEF < 50% and open heart surgery for other cause (CABG) are each an indication for valve replacement. LV dilatation counts only when the systolic diameter is > 50 mm, so 'dilated LV dimensions' with no size given is the weakest choice.

- 4 * All of the following are potential causes of tricuspid regurgitation except:

(c) Inferior wall myocardial infarction

Congenital heart disease, infective endocarditis and rheumatic fever are listed causes of tricuspid regurgitation. Inferior wall MI is the only option not named in that list, so it is the expected answer.

Model Answers | Essay Questions

- 1 List the different surgical options for surgery in case of mitral stenosis.

Intervention treatment

- * **Percutaneous Balloon Mitral Valvuloplasty (PBMV):** indicated when there is **suitable anatomy** and mitral valve area < **1.5 cm²** with:
 - Symptomatic.
 - Or asymptomatic with:
 - High risk of thromboembolism.
 - Pulmonary hypertension.
 - Desire of pregnancy.
 - Undergoing major non cardiac surgery.

Surgical treatment

- * **Surgical mitral valve replacement**
 - Indications: the same indications of PBMV when:
 - Anatomy is not suitable.
 - Or the patient needs open heart surgery for other reason.
 - Post-operative medications:
 - If **metallic prosthesis**: warfarin for life with INR adjustment between **2.5 and 3.5**.
 - If **bio prosthesis**: warfarin for **3-6 months**, then antiplatelet therapy.

Note The handout gives only these two options for mitral stenosis; other operations are not covered.

- 2 List the different surgical options for mitral valve surgery.

Mitral stenosis

- * **Percutaneous Balloon Mitral Valvuloplasty (PBMV):** indicated when there is **suitable anatomy** and mitral valve area < **1.5 cm²** with:
 - Symptomatic.
 - Or asymptomatic with:
 - High risk of thromboembolism.
 - Pulmonary hypertension.
 - Desire of pregnancy.
 - Undergoing major non cardiac surgery.
- * **Surgical mitral valve replacement**
 - Indications: the same indications of PBMV when:
 - Anatomy is not suitable.
 - Or the patient needs open heart surgery for other reason.

Mitral regurgitation: modes of intervention

- * **Surgical mitral valve repair:** when leaflets are **not damaged** as in most cases of MV prolapse and secondary MR (due to annular dilatation).
- * **Surgical mitral valve replacement:** when leaflets are **damaged** as in most cases of rheumatic valvular disease.
- * **Transcatheter end-to-end repair:** a new technique that aims to fix both leaflets at certain scallops using clips applied trans-catheter without need to surgical intervention.

Post-operative medications

- * If **metallic prosthesis:** warfarin for life with INR adjustment between **2.5 and 3.5**.
- * If **bio prosthesis:** warfarin for **3-6 months**, then antiplatelet therapy.
- * After **surgical valve repair:** warfarin for **3 months**.

- 3** List the indications of aortic valve replacement in a patient with aortic regurgitation.

Chronic aortic regurgitation: surgical aortic valve replacement (SAVR)

- * Indications: **severe aortic regurgitation:**
 - If symptomatic.
 - Or asymptomatic with one of the following:
 - LVEF **< 50%** and/or LV dilatation (systolic diameter **> 50 mm**).
 - Significantly dilated aortic root or ascending aorta.
 - Patient is undergoing open heart surgery for other cause.

Management of acute aortic regurge

- * **Emergency surgical intervention** for aortic valve replacement ± aortic root replacement.
- * Surgery may be **deferred** in patients with acute AR secondary to active infective endocarditis who are **hemodynamically stable** for **5 to 7 days** of antibiotic therapy.

Coronary Heart Disease

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	7	8	9	10	11	12
Answer	c	c	c	a	d	c	a	a	b	d	a	c

Question no.	13	14	15	16	17	18	19	20	21	22	23	24
Answer	d	c	c	a	b	d	d	a	b	b	d	c

Question no.	25	26	27	28	29	30	31	32	33	34	35	36
Answer	b	c	d	c	a	c	b	d	a	a	c	d

Question no.	37	38	39	40	41	42	43	44	45	46	47	48
Answer	c	b	b	d	a	d	c	c	d	b	a	c

Question no.	49	50	51	52	53	54	55	56	57	58	59	60
Answer	c	b	a	b	d	c	b	a+b	b+d	a	a	a+b+c+d

Question no.	61	62	63	64	65	66	67	68	69	70	71	72
Answer	b+c	d	c	b	a	c	a	c	d	b	c	d

Question no.	73	74	75	76	77	78
Answer	c	a	a	c	b	a

► Second | Questions with a Note

Question no.	1	2	3	4
Answer	a	a	b	a+b+c+d

► Why this answer?

- 1 *After assessment of cardiovascular risk in an asymptomatic patient with coronary heart disease, if the...*
(c) Referral for invasive testing.
Invasive coronary angiography is recommended in patients with positive stress test, while CT coronary angiography is a good negative test in patients less likely to have coronary artery disease. So a high-risk patient goes on to invasive testing; retesting later and non-invasive testing suit lower risk, and folic acid has no role.
- 2 *Which of the following is NOT one of the criteria of typical chest pain?*
(c) Pain provocation by deep inspiration
Typical angina is constricting chest discomfort, brought on by exertion or emotion, and relieved within minutes by rest or nitroglycerin. Pain made worse by deep breathing is pleuritic pain and points to a non-ischemic cause. A duration of about 15 minutes is still consistent with angina, which is short-lived (minutes, not seconds or hours), so it does not exclude typical pain.
- 3 *Nitrates are anti-anginal drugs. Which of the following is a common side effect?*
(c) Headache.
Headache is a common side effect of nitrates that could be managed with paracetamol. Nitrates are vasodilators, so they lower rather than raise blood pressure.
- 4 *Which of the following is an absolute contraindication of thrombolytic therapy in patients with acute...*
(a) Aortic dissection
Aortic dissection is an absolute contraindication for fibrinolysis. Refractory hypertension is only relative, menses are excluded from bleeding disorders, and ischaemic stroke counts only within the preceding 6 months.
- 5 *How much exercise each week is advised to help prevent heart disease?*
(d) 150 minutes on average
Physical exercise is one of the lifestyle measures. The advised amount is at least 150 minutes of moderate-intensity exercise every week.
- 6 *Which is not considered a life-threatening cause of chest pain?*
(c) Acute myocarditis
The chest pains that can kill within hours are acute coronary syndrome (unstable angina included), pulmonary embolism, aortic dissection and tension pneumothorax. Myocarditis is a cause of acute chest pain but is not one of these emergencies.
- 7 *Which of the following should NOT be used routinely in primary prevention of IHD?*
(a) Daily Aspirin.
Aspirin for life is given to patients who already have chronic stable angina, not routinely to people without heart disease, because of its bleeding risk. Exercise, smoking cessation and weight reduction are the routine lifestyle measures.
- 8 *Which of the following drugs is contraindicated in patients with acute myocardial infarction?*
(a) Verapamil.
Non DHP CCBs such as verapamil decrease contractility and must be avoided in heart failure with reduced systolic function as they worsen heart failure; acute infarction often damages the pump, so verapamil is avoided. Ticagrelor and rosuvastatin are emergency drugs of ACS and beta-blockers are continued for life after it.
- 9 *In a patient with acute chest pain that lasts for 30 minutes, with ECG showing ST depression in lateral...*
(b) Non ST elevation MI
No ST segment elevation with positive cardiac enzymes is NSTEMI. STEMI needs ST elevation, and unstable angina has negative enzymes.
- 10 *In a patient with chronic coronary stable [sic] who is still symptomatic on metoprolol and short-acting...*
(d) Isosorbide mononitrate
He is already on 1st line therapy (short acting nitrate and beta blocker), so the next step is 2nd line therapy: long-acting nitrates such as isosorbide mononitrate. His heart rate of 55 is already within the target 50-60, so verapamil and ivabradine add little, and sildenafil must not be given with nitrates.

- 11** Which of the following is an indication of immediate coronary intervention in a patient with acute coronary...
(a) Cardiogenic shock.
 Cardiogenic shock is hemodynamic instability, which puts the patient in the very high-risk group: PCI within less than 2 hours (immediate). Fixed ST depression is not a dynamic change, and diabetes or hypertension are not criteria for immediate PCI.
- 12** In a 75-year-old male patient with passed-time acute inferior myocardial infarction, after 48 hours of the...
(c) Alteplase
 Fibrinolytic therapy such as alteplase can be given only in the first 12 hours of chest pain, so at 48 hours it must be avoided. After 48 hours PCI should still be done if the patient presents with electrical complications such as complete heart block, and a temporary pacemaker treats the block.
- 13** In a 58-year-old male patient presented with non-ST elevation myocardial infarction with recurrent attacks...
(d) Coronary intervention
 Recurrent ventricular tachycardia is electrical instability, so this NSTEMI patient is very high risk and needs PCI within less than 2 hours. Streptokinase is fibrinolytic therapy for STEMI only, and short self-limiting runs do not need DC shock.
- 14** A 65-year-old male patient has persistent anginal attacks despite taking bisoprolol and long-acting...
(c) Trimetazidine
 Avoid combining nicorandil with nitrates, and with a heart rate of 55 the rate-lowering drugs verapamil and ivabradine are not suitable. Trimetazidine changes the heart fuel from fatty acids to glucose without slowing the heart, so it is the right add-on.
- 15** What is the next step of management if you suspect a patient of having coronary artery disease after...
(c) Stress ECG
 Once life-threatening causes are excluded, suspected coronary disease needs a stress test to unmask exercise induced ischemic heart disease, and stress ECG is one of them. Chewed aspirin, morphine and emergency referral belong to acute coronary syndrome.
- 16** Ticagrelor is one of the emergency treatments of acute myocardial infarction. Which of the following is a...
(a) Patients on oral anti-coagulation
 Ticagrelor is contraindicated in patients on oral anticoagulants, advanced liver disease, and previous intracranial hemorrhage. Stroke in general, hypertension and diabetes are not in this list.
- 17** In patients with suspected chronic stable angina and normal resting ECG and echocardiography, which of the...
(b) Stress myocardial perfusion imaging
 Resting ECG and echocardiography are usually normal in between attacks, so a stress test is needed, and stress myocardial perfusion imaging is one of them. A resting cardiac MRI does not unmask ischemia, and cardiac enzymes belong to acute coronary syndrome.
- 18** Which of the following should NOT be used routinely in primary prevention of IHD?
(d) Aspirin (81 mg) daily.
 Aspirin for life is given to patients who already have chronic stable angina, not routinely to people without heart disease, because of its bleeding risk. Smoking cessation, exercise and healthy diet are routine lifestyle measures.
- 19** Which of the following clinical characters is not suggestive of acute coronary syndrome and does not...
(d) Chest pain lasting for 5 minutes
 Anginal pain usually lasts for less than 15 minutes, while acute coronary syndrome pain lasts for more than 20 minutes, so a 5-minute pain alone is not suggestive. Fast breathing, tachycardia above 120 and systolic pressure below 90 are signs of an unstable patient who must be referred.
- 20** Ticagrelor is one of the emergency treatments of acute myocardial infarction. Which of the following is a...
(a) History of intracranial hemorrhage
 Previous intracranial hemorrhage is a contraindication for ticagrelor. Stroke in general, hypertension and diabetes are not in the list.

- 21** *Diltiazem is a first line anti-anginal drug. Which of the following is a contraindication?*
(b) Ejection fraction 35%.
 Diltiazem is a non DHP CCB; avoid non DHP CCBs in heart failure with reduced systolic function as they worsen heart failure, so an ejection fraction of 35% is a contraindication. In coronary spasm it is the beta blocker that is avoided, and Ca-channel blockers improve diastolic filling in HOCM.
- 22** *In patients with suspected chronic stable angina and normal resting ECG and echocardiography, which of the...*
(b) Dobutamine echocardiography.
 Resting ECG and echocardiography are usually normal in between attacks, so a stress test is needed, and dobutamine echocardiography is a pharmacological stress test.
- 23** *Which of the following is less likely to be anginal pain?*
(d) Non-exertional fleeting stitching chest pain
 Anginal pain could be only in the referred areas, so exertional jaw, back or epigastric pain relieved by rest can be angina. Pain lasting for seconds is less likely related to ischemic origin, so fleeting non-exertional pain is the least likely.
- 24** *The following drug could increase the heart rate in patients with chronic angina:*
(c) Amlodipine
 Beta blockers (bisoprolol), non DHP CCBs (diltiazem) and ivabradine all lower the heart rate. Amlodipine is a dihydropyridine CCB, a pure vasodilator, and can cause reflex tachycardia.
- 25** *Which of the following is not an indication of ACE inhibitors in a patient with unstable angina?*
(b) Recurrent chest pain
 After ACS, the ACE inhibitor or ARB is given for life to those with hypertension, diabetes mellitus, anterior wall STEMI and reduced left ventricular systolic function. Recurrent chest pain is not one of them.
- 26** *A 54-year-old male is presented with unstable angina. He is indicated for emergent coronary intervention if:*
(c) Cardiogenic shock
 Cardiogenic shock is hemodynamic instability, which puts the patient in the very high-risk group: PCI within less than 2 hours. A fixed T inversion is not a dynamic change, and extrasystoles are not electrical instability.
- 27** *A patient was prescribed sildenafil. Which of the following drugs must be avoided in his case?*
(d) Isosorbide dinitrate
 It is not recommended to give nitrates such as isosorbide dinitrate to a patient on sildenafil, to avoid profound hypotension and shock.
- 28** *A 54-year-old male is presented with unstable angina. He is indicated for coronary intervention if:*
(c) Ventricular tachycardia
 Ventricular tachycardia is electrical instability, which puts the patient in the very high-risk group: immediate PCI. Isolated ventricular extrasystoles and a fixed T inversion are not criteria.
- 29** *Bisoprolol is first line anti-anginal treatment in chronic coronary syndrome but must be avoided in which of...*
(a) Suspected coronary spasm.
 Avoid betablocker in variant angina as it produces coronary spasm. Heart failure with reduced systolic function is a reason to avoid non DHP CCBs, not beta blockers, and bisoprolol can be used in stable HF.
- 30** *Chest x ray could help in a patient with suspected acute coronary syndrome to rule out all the following...*
(c) Esophageal spasm
 Chest x-ray can give clues about pneumothorax, aortic dissection and pulmonary embolism and could pick up a dilated aortic root. Esophageal spasm gives no X-ray sign.
- 31** *Which of the following is a relative contraindication of thrombolytic therapy in patients with acute...*
(b) Severe hypertension
 Refractory hypertension (systolic blood pressure >180 mmHg) is a relative contraindication for fibrinolysis. Aortic dissection, haemorrhagic stroke and ischaemic stroke in the preceding 6 months are absolute contraindications.

- 32 Regarding lifestyle recommendations of ischemic heart disease patients, all of the following are recommended...
(d) Hormone replacement is recommended for all menopausal women.
 Hormone replacement therapy does not protect the heart and is not given for that purpose. Exercise, weight loss in obese patients and aspirin in selected patients are sound advice; the handout lists physical exercise and weight reduction.
- 33 Fibrinolytic therapy is indicated in:
(a) ST elevation myocardial infarction
 Fibrinolytic therapy is only for STEMI. It has no role in unstable angina, NSTEMI or coronary spasm.
- 34 A 57-year-old man has increasing chest pain for the past 3 days. The pain is progressively persistent (even...
(a) Emergency transfer to hospital.
 Pain of increasing intensity, frequency and duration that occurs at rest and is not relieved by sublingual nitrates is acute coronary syndrome. He needs emergency transfer to hospital, not more tablets or an X-ray.
- 35 Which is the first-line treatment for primary prevention of ASCVD in diabetics?
(c) Statin therapy.
 In diabetic patients the first drug to prevent atherosclerotic disease is a statin, which inhibits cholesterol synthesis. Aspirin is not routine in patients without established disease.
- 36 In patients with ischemic heart disease receiving the maximal doses of atorvastatin and ezetimibe, what is...
(d) Alirocumab
 After the maximal dose of statin with ezetimibe, the next step is a PCSK9 inhibitor such as alirocumab. Rosuvastatin is just another statin and adds little to maximal atorvastatin.
- 37 Ticagrelor should be avoided in acute coronary syndrome patients who have history of:
(c) Intracranial hemorrhage
 Previous intracranial hemorrhage is a contraindication for ticagrelor. Venous thrombosis, ischemic stroke and hemoptysis are not in the list.
- 38 Patients with functional mitral regurg, acute chest pain and normal troponin mostly have the following ECG:
(b) ST depression
 Acute chest pain with normal troponin is unstable angina, which has no ST segment elevation. Its ischemic ECG change is ST segment depression or T wave inversion.
- 39 The modifiable risk factor that is associated with coronary artery disease is:
(b) Obesity
 Central obesity is a risk factor of coronary atherosclerosis that the patient can change. Age, gender and heredity cannot be changed.
- 40 Cardiogenic shock is characterized by:
(d) a and b
 Cardiogenic shock presents with tachycardia and hypotension. It is pump failure, so filling pressures and the central venous pressure rise; its pulmonary edema is not hypertensive.
- 41 The following drug should be avoided in patients on nitrates therapy:
(a) Sildenafil
 It is not recommended to give nitrates to a patient on sildenafil, to avoid profound hypotension and shock.
- 42 Which of the following anticoagulants should be avoided in acute coronary syndrome patients undergoing...
(d) Fondaparinux
 Fondaparinux is not used if the patient is to undergo PCI (risk of catheter thrombosis).
- 43 Which of the following should NOT be used routinely in primary prevention of ischemic heart disease?
(c) Aspirin (81 mg) daily
 Aspirin for life is given to patients who already have chronic stable angina, not routinely to people without heart disease, because of its bleeding risk. Smoking cessation, exercise and diet with lipid lowering in dyslipidemia are routine.

- 44 *Right ventricular infarction could occur in setting of acute inferior wall myocardial infarction. Which of...*
(c) Nitroglycerine
Nitrates decrease preload by venodilatation; the infarcted right ventricle depends on its filling, so nitroglycerine can cause severe hypotension and must be avoided, while IV fluids support it.
- 45 *The best time window for thrombolytic therapy in patients with acute myocardial infarction is:*
(d) Within 12 hours from the onset of chest pain
Fibrinolytic therapy can be given in the first 12 hours of chest pain. The 24 and 48 hour figures belong to primary PCI.
- 46 *Which of the following is characteristic of STEMI (ST-Elevation Myocardial Infarction)?*
(b) Prolonged chest pain with ST-segment elevation on ECG
STEMI has ST segment elevation with positive cardiac enzymes, and ACS pain lasts for more than 20 minutes. Normal ECG, normal troponin and quick relief do not fit.
- 47 *Which is a characteristic feature of unstable angina in contrast to NSTEMI?*
(a) Normal troponin levels
Unstable angina and NSTEMI both have no ST segment elevation; the difference is the cardiac enzymes: negative in unstable angina, positive in NSTEMI.
- 48 *What is the recommended target for LDL cholesterol in patients with Acute Coronary Syndrome (ACS)?*
(c) <55 mg/dL
Target LDL for patients with acute coronary syndrome must be below 55 mg/dl with the maximum tolerated dose of statins with or without ezetimibe and PCSK9 inhibitors.
- 49 *Which of the following is a common symptom in patients with Chronic Coronary Syndrome?*
(c) Exertional chest pain (angina)
Anginal chest pain is brought on by exertion and relieved by rest. Pain at rest points to acute coronary syndrome.
- 50 *In patients with STEMI, which of the following is the preferred method of reperfusion therapy?*
(b) Primary percutaneous coronary intervention (PCI)
Primary PCI is the preferred line of treatment in the 1st 24 hours after STEMI. Fibrinolytic therapy is given only if Cath lab is not available.
- 51 *In patients with ACS and elevated LDL cholesterol, which of the following medications would most likely be...*
(a) Statins
A loading dose of statin is given in ACS to reach the target LDL below 55 mg/dl. Aspirin, ACE inhibitors and CCBs do not lower LDL.
- 52 *Which of the following clinical manifestations is common in patients with Chronic Coronary Syndrome?*
(b) Chronic exertional angina
Chronic coronary syndrome presents with exertional anginal pain that usually lasts for less than 15 minutes. Hours-long pain, rest dyspnea and sudden heart attack belong to acute coronary syndrome.
- 53 *Which of the following could be a manifestation of chronic stable angina?*
(d) Exertional syncope
Faintness is one of the angina equivalents, so exertional syncope can be a manifestation of stable angina. Exertional leg pain points to peripheral artery disease, and ankle edema or headache are not angina equivalents.
- 54 *The following anti-ischemic drug must be avoided in heart failure with reduced systolic function:*
(c) Diltiazem
Avoid non DHP CCBs such as diltiazem in heart failure with reduced systolic function as they worsen heart failure.
- 55 *If the patient is presented to a PCI non-capable center, the time window of thrombolytic therapy is:*
(b) 12 hours
Fibrinolytic therapy can be given in the first 12 hours of chest pain if Cath lab is not available, then immediate transfer to a PCI capable center.

- 56** The following is/are absolute contraindications of thrombolytic therapy (choose all that apply):
(a) History of previous intracranial hemorrhage + (b) Brain malignancy
 The handout lists as absolute contraindications haemorrhagic stroke at any time and central nervous system neoplasms. Severe (refractory) hypertension above 180 mmHg systolic and pregnancy (within 1 week post-partum) are only relative contraindications.
- 57** The following anti-ischemic drug/s must be continued for life after acute myocardial infarction (choose all...)
(b) Aspirin + (d) Atorvastatin
 The handout says aspirin is continued at 75 mg daily for life and atorvastatin 80 mg daily for life after an acute coronary syndrome. Clopidogrel (or ticagrelor) is given with aspirin for one year only, then stopped. Long-acting nitrates such as isosorbide dinitrate are for symptoms only and are not a lifelong secondary prevention drug.
- 58** The following is the most common pathophysiology of acute coronary syndrome:
(a) Plaque rupture and coronary thrombosis
 ACS occurs on top of acute coronary ischemic event, mostly thrombosis, with plaque instability (erosion or rupture). Spasm, dissection and embolism are less common causes.
- 59** What is the best next step for such patient?
(a) Stress echocardiography
 Exertional chest pain with normal resting investigations needs a stress test to unmask exercise induced ischemic heart disease, such as stress echocardiography. Cardiac enzymes belong to acute coronary syndrome.
- 60** The following is/are symptoms of typical angina chest pain (choose all that apply):
(a) Exertional + (b) Retrosternal compressing + (c) Relief by rest + (d) Relief by sublingual nitrates
 The handout defines typical angina by three features: constricting discomfort in the chest, brought on by exertion, and relieved within 5 minutes by rest or nitroglycerin. All four options describe these features, so all apply.
- 61** Which of the following is/are first-line anti-anginal drugs (choose all that apply)?
(b) verapamil + (c) Short acting sublingual nitrates
 In the handout, first-line anti-anginal therapy is short-acting sublingual nitrates plus a beta blocker or a non-dihydropyridine calcium channel blocker such as verapamil. Long-acting nitrates are second line, and ranolazine is a later-line drug.
- 62** Despite being first line anti-anginal drug, metoprolol should be avoided in:
(d) Variant angina
 Avoid betablocker in variant angina as it produces coronary spasm. Beta-blockers are given for life after ACS.
- 63** Which of the following anti-anginal drugs acts by improving the myocardial metabolic profile?
(c) Trimetazidine
 Trimetazidine inhibits β -oxidation of free fatty acid and changes the heart fuel from fatty acids to glucose, reducing lactic acid production and angina. Ivabradine is a funny Na channel blocker, ranolazine blocks IKr, and nicorandil relaxes coronary smooth muscle.
- 64** The following medication/s could decrease the probability of future myocardial infarction in this patient:
(b) Rosuvastatin.
 Lipid lowering drugs such as rosuvastatin prevent the progression of disease and the development of acute coronary syndrome. Nitrates and beta blockers are anti-anginal drugs for symptom relief.
- 65** You strongly suspect chronic angina in this patient. What is the best test to confirm the diagnosis?
(a) Dobutamine echocardiography
 Resting ECG and echocardiography are usually normal in between attacks, so a stress test is needed to unmask ischemia, and dobutamine stress echocardiography is one of them. Chest X-ray, CT chest and resting cardiac MRI do not unmask ischemia.
- 66** The following medication/s could decrease the frequency of angina attacks in this patient:
(c) Trimetazidine
 Only anti-anginal drugs for symptom relief reduce the attacks, and trimetazidine is a 3rd line one. Clopidogrel, atorvastatin and ACEI-type drugs prevent progression of disease and acute coronary syndrome.

- 67** The following could be a non-coronary cause of anginal pain:
(a) Severe anemia
 Severe anaemia is a functional cause that decreases oxygen supply, so it gives true anginal pain without coronary disease. Pleuritic pain is mostly non-ischemic, and herpes zoster and rib fracture are other causes of chest pain.
- 68** The following is/are emergency medications in the ER:
(c) 180 mg ticagrelor
 Only the ticagrelor dose is right: loading dose 180 mg. The loading doses are aspirin 300 mg, clopidogrel 300 mg (600 mg if undergoing PCI) and atorvastatin 80 mg.
- 69** After successful primary percutaneous coronary intervention, the following drug/s must continue for life:
(d) All the above
 Aspirin and the statin are continued for life after ACS. This patient is diabetic and hypertensive, so the ACE inhibitor ramipril is also for life.
- 70** If the patient developed acute pulmonary edema after acute myocardial infarction, the following is a...
(b) Acute severe mitral regurgitation
 Acute severe mitral regurgitation due to infarction of the papillary muscles is a complication of MI and causes acute pulmonary edema. Pulmonary embolism gives a clear back, and tricuspid regurgitation and tamponade do not cause pulmonary edema.
- 71** If the patient did not get his LDL levels reduced with the maximal doses of rosuvastatin and ezetimibe, what...
(c) Alirocumab
 After the maximal dose of statin with ezetimibe, the next step is a PCSK9 inhibitor, and alirocumab is one. Fibrates and omega 3 mainly lower triglycerides, and olive oil is diet only.
- 72** The most specific marker for diagnosis of acute myocardial infarction is:
(d) Cardiac troponins
 The handout names cardiac troponin and CK-MB as the cardiac biomarkers. Troponins are the most specific, because they come only from heart muscle.
- 73** Acute subendocardial infarction will have the ECG finding:
(c) Deep symmetrical T wave inversion
 A subendocardial infarction does not involve the full wall, so it is a non-ST elevation infarction with ST segment depression or T wave inversion, without ST elevation or pathological Q waves.
- 74** All of the following are common arrhythmias developing from AMI except:
(a) Sinus arrhythmia
 Respiratory sinus arrhythmia is a normal variation found in healthy young adults, not a complication of infarction. Ventricular tachycardia and atrioventricular block are electrical complications of ACS, and accelerated idioventricular rhythm is common after reperfusion.
- 75** Angina due to an imbalance between O₂ supply and demand without atherosclerosis would most likely be seen in...
(a) Aortic regurgitation
 In aortic regurgitation the chest pain is due to drop of diastolic pressure and impaired coronary filling, so angina occurs with normal coronaries. Tamponade, pulmonary regurgitation and right heart failure do not cause this mismatch.
- 76** A 55-year-old man is admitted to the emergency ward with acute myocardial infarction. Which of the following...
(c) Streptokinase
 Streptokinase is a fibrinolytic used for reperfusion in STEMI. Clopidogrel is an antiplatelet, and heparin and warfarin are anticoagulants; none of them opens the artery.
- 77** Which of the following findings is the most specific for a diagnosis of myocardial infarction?
(b) Evolution of Q waves on ECG
 Evolution of pathological Q waves means the myocardium has actually died, so it is the most specific of these findings for infarction. Cardiac enzymes such as CK, LDH and AST also rise in muscle injury, liver disease and other conditions, and even troponin rises in many non-infarct causes of myocardial injury. Chest pain and ST elevation also occur in pericarditis, early repolarisation and other conditions.

78 A 54-year-old male redevelops chest pain 72 hours after treatment for an anterior myocardial infarction....

(a) CK-MB

Troponins stay high for 1-2 weeks after an infarction, so at 72 hours they cannot show a new one. CK-MB, the other cardiac biomarker, returns to normal in 2-3 days, so a fresh rise means reinfarction.

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

1 A -- years old patient with intracranial hemorrhage; the following drug is contraindicated:

(a) Ticagrelor

Previous intracranial hemorrhage is a contraindication for ticagrelor, so (a) is the drug the handout names.

2 A 45-year-old patient presented with angina. He has liver cirrhosis and hematemesis. The following drug is...

(a) Ticagrelor

Advanced liver disease is a contraindication for ticagrelor, so (a) is the drug the handout names in this cirrhotic patient.

3 Which of the following medications is used in the management of Chronic Coronary Syndrome to relieve chest...

(b) Nitrates

Nitrates work by coronary vasodilation and increasing coronary blood supply, and by decreasing preload and afterload. Beta blockers and non DHP CCBs act by decreasing contractility and heart rate.

4 The following is/are emergency anti-ischemic medications in the emergency room (choose all that apply):

(a) 300 mg aspirin + (b) 180 mg ticagrelor + (c) 80 mg atorvastatin + (d) 5 mg sublingual nitrates

This is an anterior STEMI, and the handout's emergency treatment gives loading doses of aspirin 300 mg, ticagrelor 180 mg and atorvastatin 80 mg. Nitrates relieve ischemic pain; a 5 mg sublingual isosorbide dinitrate tablet is the usual first nitrate dose, and the blood pressure (150/90) allows it. So all four are given in the emergency room.

Model Answers | Essay Questions

1 Discuss the diagnostic work up of a patient with chronic stable angina.

1. To check for etiology / risk factors

- * Fasting blood sugar / 2 hours post prandial / HbA1C.
- * Lipid profile.
- * Kidney function tests.
- * CBC.
- * Chest X ray could pick up rib fracture or dilated aortic root.

2. To confirm the diagnosis of chronic coronary syndrome

Test	Findings / use
ECG	Usually is normal in between attacks; if performed during the attack: you could pick up ischemic ECG changes.
Echocardiography	Usually normal in between attacks; if performed during the attack: you could pick up ischemic wall motion abnormality with or without diastolic dysfunction or systolic dysfunction; could pick up other potential causes of chest pain: pericardial effusion could refer to pericarditis, aortic stenosis, aortic aneurysm.
Stress test	To unmask exercise induced ischemic heart disease (stress could be exercise or pharmacological stress using dobutamine or adenosine); stress ECG; stress echocardiography; stress myocardial perfusion imaging; stress cardiac MRI.
Non-invasive coronary imaging	CT coronary angiography is recommended as a good negative test in patients less likely to have coronary artery disease.
Invasive coronary angiography	Is recommended in patients with positive stress test .

2 Outline the indications of revascularization of non-ST elevation acute coronary syndromes.

For NSTEMI-ACS patients (NSTEMI and UA):

Risk group	Timing of PCI	Criteria
Very high-risk patients	PCI should be done within less than 2 hours (immediate)	Hemodynamic instability; electrical instability; refractory chest pain.
High-risk patients	PCI should be done within less than 24 hours (early invasive)	Elevated cardiac biomarkers (troponin); dynamic changes in ST segment or T wave in ECG.
Low-risk patients (other patients not having criteria of very high or high risk)	Selective PCI may be considered in patients who have one or more of the following	Prior PCI within 6 months; prior CABG; DM; CKD; LVEF <40%; early post-infarction angina.

3 Is this pain life threatening? Justify your answer.

Anginal chest pain

- * Constricting / heavy discomfort to the chest, jaw, neck, shoulders, or arms.
- * Symptoms brought on by **exertion**, emotion, cold weather and heavy meals.
- * Symptoms relieved within **5 min** by **rest or nitro-glycerine (GTN)**.
- * Anginal pain usually lasts for **less than 15 minutes**.

Acute coronary syndrome: typical presentation

The typical presentation is continuous severe retrosternal compressing anginal pain which occurs **at rest**, lasts for **more than 20 minutes**, and **not relieved** by rest or sublingual nitrates.

Note The handout does not state which chest pain is life-threatening, answer incomplete.

4 Classify this chest pain. Justify your answer.

- ① Typical angina.

Anginal chest pain

- ① Constricting / heavy discomfort to the chest, jaw, neck, shoulders, or arms.
② Symptoms brought on by **exertion**, emotion, cold weather and heavy meals.
③ Symptoms relieved within **5 min** by **rest or nitro-glycerine (GTN)**.

Number of features	Type of chest pain
All the 3 features	Typical angina
Only 2 features	Atypical angina
0-1 feature	Non-anginal chest pain

5 What is the best next step?

To confirm the diagnosis of chronic coronary syndrome:

Test	Findings / use
ECG	Usually is normal in between attacks; if performed during the attack: you could pick up ischemic ECG changes.
Echocardiography	Usually normal in between attacks; if performed during the attack: you could pick up ischemic wall motion abnormality with or without diastolic dysfunction or systolic dysfunction; could pick up other potential causes of chest pain: pericardial effusion could refer to pericarditis, aortic stenosis, aortic aneurysm.
Stress test	To unmask exercise induced ischemic heart disease (stress could be exercise or pharmacological stress using dobutamine or adenosine): stress ECG; stress echocardiography; stress myocardial perfusion imaging; stress cardiac MRI.
Non-invasive coronary imaging	CT coronary angiography is recommended as a good negative test in patients less likely to have coronary artery disease.
Invasive coronary angiography	Is recommended in patients with positive stress test .

Note The handout does not name one best next step, answer incomplete.

6 Is this pain life threatening? Justify your answer.

Typical presentation of acute coronary syndrome

The typical presentation is continuous severe retrosternal compressing anginal pain which occurs **at rest**, lasts for **more than 20 minutes**, and **not relieved** by rest or sublingual nitrates.

* Association: typically associated with nausea, vomiting, and sweating.

Other presentations of acute coronary syndromes

- * Attacks of anginal pain of increasing intensity, frequency, and duration (crescendo angina).
- * New onset angina (de novo angina).
- * Post infarction angina.
- * Syncopal attacks as a complication.
- * Acute dyspnea (acute heart failure).
- * Cardiac arrest as a complication.

Prognosis

The outcomes of patients with ACS showed that the 30-day mortality rate is: **1.7%** for patients with unstable angina (UA); **7.4%** for patients with NSTEMI; **11.1%** for those with STEMI.

Note The handout does not state which chest pain is life-threatening, answer incomplete.

7 What's your best next step as a primary care physician?

Surface 12-lead Electrocardiogram (ECG): should be done **as early as possible** for the suspected ACS.

Emergency treatment

- ① Opioids only in severe intolerable pain: e.g., morphine IV.
- ② Immediate loading doses of antiplatelet drugs:
 - Aspirin: an initial daily dose of **300 mg**, followed by a daily dose of 75 mg for life.
 - P2Y12 inhibitors: ticagrelor, loading dose of **180 mg** followed by 90 mg twice daily for one year; or clopidogrel, 300 mg (600 mg if undergoing percutaneous coronary intervention (PCI)) loading dose, then 75 mg daily for one year.
 - Ticagrelor is preferred unless unavailable or contraindicated (patients in oral anticoagulants, advanced liver disease, and previous intracranial hemorrhage).

- ③ Loading dose of statin therapy:
- 80 mg atorvastatin followed by daily dose of 80 mg for life;
 - OR 40 mg rosuvastatin followed by daily dose of 40 mg for life.
 - Target LDL for patients with acute coronary syndrome must be below **55 mg/dl** with the maximum tolerated dose of statins with or without ezetimibe and PCSK9 inhibitors.
- ④ Intravenous nitroglycerin at 5-20 µg/min may help to control ischemic pain with close monitoring of blood pressure. Dose can be increased according to the blood pressure.
- ⑤ Anticoagulants: one of the following:
- Unfractionated heparin.
 - Low molecular weight heparin.
 - Fondaparinux (fondaparinux is **not used if the patient is to undergo PCI** (risk of catheter thrombosis)).

Note The handout has no primary-care plan or referral step, answer incomplete.

8 Enumerate causes of life-threatening chest pain.

Differential diagnosis of acute chest pain:

	Cause	Clue in ABCDE approach
Cardiac causes	Pericarditis	Pericardial rub
	Myocarditis	Pericardial rub
	Pulmonary embolism	Tachycardia, desaturation, and clear back
Chest causes	Tension pneumothorax	Diminished air entry on one side
	Pleuritis	Diminished air entry on one side if with effusion
Vascular causes	Aortic dissection	Unequal pulse and BP

	Cause	Clue in ABCDE approach
GIT causes	Cholecystitis	Epigastric tenderness
	Pancreatitis	Epigastric tenderness
	Esophageal spasm	
Chest wall causes	Myositis	Tenderness
	Chest wall trauma	Tenderness and visible injury
	Herpes zoster infection	Rash on the skin

Note The handout does not mark which causes are life-threatening, answer incomplete.

9 Outline the risk factors of chronic stable angina.

Risk factors of coronary atherosclerosis:

- * Old age.
- * Male gender.
- * Sedentary life.
- * Diabetes mellitus.
- * Hypertension.
- * Dyslipidemia.
- * Smoking.
- * Central obesity.

10 Enumerate the three lines of treatment of anginal pain for chest pain relief.

Anti-anginal drugs for symptom relief:

1st line therapy

① Short acting nitrates

- Sublingual nitroglycerine or nitroglycerine spray.
- Used during the attack of angina or shortly before exertion.
- It is not recommended to give nitrates to patient on sildenafil to avoid profound hypotension and shock.
- Nitrates work by: coronary vasodilation and increasing coronary blood supply; decreasing the O₂ demand by decreasing preload (venodilatation) and afterload (vasodilatation).

② Beta blockers or calcium channel blockers (non-dihydropyridines)

- Both groups decrease contractility and heart rate with subsequent reduction in myocardial oxygen demand.
- Target heart rate: **50-60 beat/min.**
- Selective B₁ blockers are useful: carvedilol, bisoprolol, metoprolol.
- Non DHP CCBs: verapamil and diltiazem.
- Avoid betablocker in variant angina as it produces coronary spasm.
- Avoid non DHP CCBs in heart failure with reduced systolic function as they worsen heart failure.

2nd line therapy: Long-acting nitrates

- * Oral and transdermal patches are available.
- * It is important to leave **8 hours** nitrates free period to avoid nitrates tolerance.
- * Headache is a common side effect that could be managed with paracetamol.

3rd line therapy

Drug	Mechanism & notes
Nicorandil	It relaxes coronary vascular smooth muscle by stimulating guanylyl cyclase and increasing cyclic GMP (cGMP) levels; also, it results in activation of K ⁺ channels and hyperpolarization; avoid combining nicorandil with nitrates.
Trimetazidine	It is a cytoprotective drug that inhibits β -oxidation of free fatty acid and increases the metabolic rate of glucose; therefore, it changes the heart fuel from fatty acids to glucose with subsequent reduction in lactic acid production and angina.
Ranolazine	It blocks IK _r , the rapid portion of the delayed rectifier potassium current, and prolongs the QT _c interval in a dose-dependent fashion; ranolazine exerts its therapeutic effects without negative chronotropic, dromotropic, or inotropic actions neither at rest, nor during exercise.
Ivabradine	Funny Na channel blocker; possible alternative to beta-blockers (or rate-limiting calcium antagonists) for rate control if contra-indicated or not tolerated; may cause visual disturbance (phosphenes) due to retinal side-effects; it has cytochrome P450 3A4 drug interactions.

11 Enumerate the indications of revascularization in patients with chronic angina.

Revascularization with PCI (percutaneous coronary intervention) or CABG (coronary artery bypass graft) in the following indications:

- ① Coronary stenosis **> 90 %** in one or more coronary arteries.
- ② Coronary stenosis **(50-90%)** with one of the following:
 - Refractory angina.
 - Systolic dysfunction.
 - Positive noninvasive stress test.
 - Significant ischemia with invasive hemodynamic assessment.

12 Classify acute coronary syndrome and mention the characteristics of each group.

DEFINITION

Acute coronary syndrome (ACS) refers to a spectrum of clinical presentations that occur on top of acute coronary ischemic event (mostly thrombosis).

Type	ST segment in ECG	Cardiac enzymes
ST-segment elevation myocardial infarction (STEMI)	ST segment elevation	Positive
Non-ST segment elevation myocardial infarction (NSTEMI)	No ST segment elevation	Positive
Unstable angina	No ST segment elevation	Negative

13 List the investigations of a patient with suspected acute coronary syndromes and possible key findings.

1. Surface 12-lead Electrocardiogram (ECG)

Should be done as early as possible for the suspected ACS. It may show any of the following (changes must be present in **2 or more** contiguous leads):

- * ST elevation (≥ 1 mm) - pathological Q waves (if STEMI).
- * ST segment depression.
- * T wave inverted or biphasic.
- * The presence of normal ECG does **NOT exclude ACS** if typical clinical picture is present and at this time follow up ECG, full investigation and monitoring should be done.
- * Electrical complications e.g., atrioventricular block, supraventricular arrhythmias (including atrial fibrillation), ventricular tachyarrhythmias (ventricular tachycardia and ventricular fibrillation).

2. Echocardiography

It should not delay revascularization in STEMI in acute setting. However, in acute setting, it is helpful in uncertain diagnosis and in patients with hemodynamic compromise. You could pick up:

- * Evidence of new regional wall motion abnormality.
- * Mechanical complications: mitral regurgitation - ventricular septal rupture.
- * Other causes of chest pain e.g., aortic dissection, cardiac tamponade, pulmonary embolism.

3. Chest x-ray

It should not delay revascularization in STEMI in acute setting. However, in acute setting, it is helpful in uncertain diagnosis. You could pick up:

- * Ischemic-related left-sided heart failure (pulmonary congestion).
- * Clues about other causes of chest pain e.g., pneumothorax, aortic dissection, and pulmonary embolism.

4. Laboratory tests (should not delay revascularization in patients with STEMI)

- * Elevated cardiac biomarkers (only in STEMI & NSTEMI, not in unstable angina) e.g., cardiac troponin, or CK-MB.
- * Lipid profile should be obtained within **24 hours** of the onset of ACS.
- * Complete blood count: to exclude anemia and check for platelets.
- * Kidney function test: to adjust medication doses.
- * Glycated hemoglobin (HbA1C): to check for DM and if controlled.

14 Discuss the treatment strategy of patients presenting with ST elevation myocardial infarction.

Emergency treatment

- ① Opioids only in severe intolerable pain: e.g., morphine IV.

- ② Immediate loading doses of antiplatelet drugs:
 - Aspirin: an initial daily dose of **300 mg**, followed by a daily dose of 75 mg for life.
 - P2Y12 inhibitors: ticagrelor, loading dose of **180 mg** followed by 90 mg twice daily for one year; or clopidogrel, 300 mg (600 mg if undergoing percutaneous coronary intervention (PCI)) loading dose, then 75 mg daily for one year.
 - Ticagrelor is preferred unless unavailable or contraindicated (patients in oral anticoagulants, advanced liver disease, and previous intracranial hemorrhage).
- ③ Loading dose of statin therapy:
 - 80 mg atorvastatin followed by daily dose of 80 mg for life;
 - OR 40 mg rosuvastatin followed by daily dose of 40 mg for life.
 - Target LDL for patients with acute coronary syndrome must be below **55 mg/dl** with the maximum tolerated dose of statins with or without ezetimibe and PCSK9 inhibitors.
- ④ Intravenous nitroglycerin at 5-20 µg/min may help to control ischemic pain with close monitoring of blood pressure. Dose can be increased according to the blood pressure.
- ⑤ Anticoagulants: one of the following:
 - Unfractionated heparin.
 - Low molecular weight heparin.
 - Fondaparinux (fondaparinux is **not used if the patient is to undergo PCI** (risk of catheter thrombosis)).

Reperfusion therapy: for STEMI patients

Time is muscle (reperfusion should start as early as possible).

Fibrinolytic therapy (only for STEMI)

Can be given in the first **12 hours** of chest pain if Cath lab is not available. Then immediate transfer to a PCI capable center (streptokinase or better alteplase).

Contraindications for fibrinolysis

*** Absolute contraindications**

- Haemorrhagic stroke or stroke of unknown origin at any time.
- Ischaemic stroke in the preceding 6 months.
- Central nervous system damage, neoplasms or structural vascular lesions (e.g. arteriovenous malformation).
- Recent major trauma/surgery/head injury (within the preceding 3 weeks).
- Gastro-intestinal bleeding within the last month.
- Known bleeding disorder (excluding menses).
- Aortic dissection.

*** Relative contraindications**

- Transient ischaemic attack in preceding 6 months, dementia.
- Oral anticoagulant therapy.
- Pregnancy within 1-week post-partum.
- Non-compressible punctures.
- Traumatic resuscitation.
- Refractory hypertension (systolic blood pressure >180 mmHg).
- Advanced liver disease.
- Infective endocarditis.
- Active peptic ulcer.

Primary PCI (percutaneous coronary intervention)

- * Is the **preferred line of treatment** in the 1st 24 hours after STEMI (could be done even within 48 hours after chest pain onset).
- * Should be done if late presentation after 48 hours ONLY IF presented by electrical (arrhythmias) or mechanical (pulmonary edema or cardiogenic shock) complications.

Surgery

- * Coronary artery bypass surgery (CABG) if:
 - Multi-vessel coronary artery disease after opening of infarct-related artery with balloon dilatation.
 - Surgical repair of a mechanical complication (e.g., ventricular septal rupture).

Other medications started in hospital after revascularization and continued for long term after discharge

- * Angiotensin-converting enzyme (ACE) inhibitor or angiotensin receptor blockers (ARBs): for life for those with:
 - Hypertension.
 - Diabetes mellitus.
 - Anterior wall STEMI.
 - Reduced left ventricular systolic function.
- * Beta-blockers for life with target heart rate in the ischemic patients (**50-60 bpm** at rest).
- * Proton pump inhibitors if:
 - The patient on dual antiplatelet drugs in the first year after ACS.
 - Gastroesophageal reflux disease (GERD).
 - High risk for GIT bleeding.
 - History of peptic ulcer.
- * Lifestyle modifications: the same as chronic angina.

- 15** Outline the emergency treatment and revascularization strategy of a patient presented with ventricular tachycardia on top of acute ST elevation myocardial infarction within the first 3 hours of the onset of chest pain.

Ventricular tachycardia: acute management

Restoring sinus rhythm with **DC shock** regardless hemodynamics status.

Emergency treatment

- ① Opioids only in severe intolerable pain: e.g., morphine IV.
- ② Immediate loading doses of antiplatelet drugs:
 - Aspirin: an initial daily dose of **300 mg**, followed by a daily dose of 75 mg for life.
 - P2Y12 inhibitors: ticagrelor, loading dose of **180 mg** followed by 90 mg twice daily for one year; or clopidogrel, 300 mg (600 mg if undergoing percutaneous coronary intervention (PCI)) loading dose, then 75 mg daily for one year.
 - Ticagrelor is preferred unless unavailable or contraindicated (patients in oral anticoagulants, advanced liver disease, and previous intracranial hemorrhage).
- ③ Loading dose of statin therapy:
 - 80 mg atorvastatin followed by daily dose of 80 mg for life;
 - OR 40 mg rosuvastatin followed by daily dose of 40 mg for life.
 - Target LDL for patients with acute coronary syndrome must be below **55 mg/dl** with the maximum tolerated dose of statins with or without ezetimibe and PCSK9 inhibitors.

- ④ Intravenous nitroglycerin at 5-20 µg/min may help to control ischemic pain with close monitoring of blood pressure. Dose can be increased according to the blood pressure.
- ⑤ Anticoagulants: one of the following:
 - Unfractionated heparin.
 - Low molecular weight heparin.
 - Fondaparinux (fondaparinux is **not used if the patient is to undergo PCI** (risk of catheter thrombosis)).

Reperfusion therapy: for STEMI patients

Time is muscle (reperfusion should start as early as possible).

Fibrinolytic therapy (only for STEMI)

Can be given in the first **12 hours** of chest pain if Cath lab is not available. Then immediate transfer to a PCI capable center (streptokinase or better alteplase).

Primary PCI (percutaneous coronary intervention)

- * Is the **preferred line of treatment** in the 1st 24 hours after STEMI (could be done even within 48 hours after chest pain onset).
- * Should be done if late presentation after 48 hours **ONLY IF** presented by electrical (arrhythmias) or mechanical (pulmonary edema or cardiogenic shock) complications.

Surgery

- * Coronary artery bypass surgery (CABG) if:
 - Multi-vessel coronary artery disease after opening of infarct-related artery with balloon dilatation.
 - Surgical repair of a mechanical complication (e.g., ventricular septal rupture).

16 Enumerate the differential diagnosis of cases with chest pain.

Differential diagnosis of acute chest pain

	Cause	Clue in ABCDE approach
Cardiac causes	Pericarditis	Pericardial rub
	Myocarditis	Pericardial rub
	Pulmonary embolism	Tachycardia, desaturation, and clear back
Chest causes	Tension pneumothorax	Diminished air entry on one side
	Pleuritis	Diminished air entry on one side if with effusion
Vascular causes	Aortic dissection	Unequal pulse and BP

	Cause	Clue in ABCDE approach
GIT causes	Cholecystitis	Epigastric tenderness
	Pancreatitis	Epigastric tenderness
	Esophageal spasm	
Chest wall causes	Myositis	Tenderness
	Chest wall trauma	Tenderness and visible injury
	Herpes zoster infection	Rash on the skin

Cardiac pain

- * Ischaemic heart disease: M.I.; angina pectoris.
- * Pericardial disease: dry pericarditis; massive pericardial effusion.
- * Massive pulmonary embolism and pulmonary infarction.
- * Aortic dissection and aortic aneurysm.

- * Cardiac neurosis:
 - Occurs in neurotic individuals.
 - Site: left inframammary and localized.
 - Radiation: no specific radiation (may be to left arm).
 - Character: stitching or stabbing.
 - No relation to exertion and no relief by rest.
 - Variable duration (seconds or hours).
 - Associated with local tenderness and may be other features of neurosis.
 - Normal cardiac exam.
- * Huge cardiomegaly: may rarely cause retrosternal heaviness.
 - N.B. Huge left atrium: sawing pain (erosion of spine).
- * Mitral valve prolapse, AS, AR.

17 How will you deal with cases presenting with chest pain?

Physical examination

- * Examination is normal in most of cases.
- * With **ABCDE approach**, you could pick up the cause of pain (cardiac or non-cardiac) or catastrophic complications for urgent management:

Step	What you could pick up
A - Airway	
B - Breathing	Coarse crepitations and desaturation could refer to acute pulmonary edema which is a common complication of ACS.
C - Circulation	Pulse: you could pick up tachy-arrhythmias (ventricular tachycardia) or brady-arrhythmias (complete heart block); BP: you could pick up hypertensive emergency as a cause or cardiogenic shock as a complication; ABG: you could find respiratory acidosis in acute heart failure or metabolic acidosis in cardiogenic shock.
D - Disability	Check for disturbed conscious level which could occur as complication (stroke or cardiogenic shock); check random blood sugar as acute MI could precipitate diabetic ketoacidosis.
E - Exposure and complete examination	Systolic murmur of acute mitral regurge; systolic murmur of ventricular septal rupture.

Investigations

Surface 12-lead Electrocardiogram (ECG): should be done **as early as possible** for the suspected ACS.

Note The handout has no stepwise plan for chest pain, answer incomplete.

18 What is the most likely diagnosis?

- 1 Musculoskeletal (chest wall) pain.

Differential diagnosis of acute chest pain: chest wall causes

	Cause	Clue in ABCDE approach
Chest wall causes	Myositis	Tenderness
	Chest wall trauma	Tenderness and visible injury
	Herpes zoster infection	Rash on the skin

Features that make angina less likely

- * Pleuritic localized chest pain or that worse with swallowing is mostly non-ischemic pain.

19 What is the most likely diagnosis? And what is the next step?

- ① Panic attacks (anxiety disorder).

Note The handout does not cover panic attacks or the next step, answer incomplete.

20 Enumerate non-cardiac causes of chest pain.

Differential diagnosis of acute chest pain:

	Cause	Clue in ABCDE approach
Chest causes	Tension pneumothorax	Diminished air entry on one side
	Pleuritis	Diminished air entry on one side if with effusion
Vascular causes	Aortic dissection	Unequal pulse and BP
GIT causes	Cholecystitis	Epigastric tenderness
	Pancreatitis	Epigastric tenderness
	Esophageal spasm	

	Cause	Clue in ABCDE approach
Chest wall causes	Myositis	Tenderness
	Chest wall trauma	Tenderness and visible injury
	Herpes zoster infection	Rash on the skin

Infective Endocarditis

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	7	8	9	10	11	12
Answer	c	a	c	c	c	c	c	a	d	a	c	b

Question no.	13	14	15	16
Answer	a	c	d	d

► Second | Questions with a Note

Question no.	1
Answer	b

► Why this answer?

- Which of the following is the least cardiac lesion that can develop infective endocarditis?
(c) ASD
Infective endocarditis is rare in ASD due to the low pressure gradient between the two atria. VSD and PDA are among the congenital lesions where it is common, and pulmonary stenosis is not named as a rare site.
- The causative organism of infective endocarditis when the route of entry is dental procedures is:
(a) Streptococcus viridans
After dental procedures and oral or upper respiratory surgery the organism is usually Strept. viridans. Staph. aureus follows cardiac surgery and catheterization, and Strept. faecalis follows gastrointestinal and genitourinary procedures.
- The least cardiac lesion that can develop infective endocarditis is:
(c) ASD
The disease is rare in ASD due to the low pressure gradient between the two atria. MR, VSD and PDA are common sites, and a narrow orifice (small defect) makes it more common, not less.
- Incidence of infective endocarditis is least in:
(c) ASD
Infective endocarditis is rare in ASD due to the low pressure gradient between the two atria. MR, VSD and PDA are high pressure lesions where it is common.

- 5 Populations not indicated for prophylaxis against infective endocarditis include:
(c) Mitral stenosis
 Infective endocarditis is not common in MS because of marked fibrosis and low pressure, and in MS prophylaxis is given only if there is a history of infective endocarditis or a mitral valve prosthesis. Prosthetic valves and a previous attack need prophylaxis, and so does the early period after device closure of a shunt.
- 6 * High risk groups of infective endocarditis include the following except:
(c) Mitral valve prolapse
 Prosthetic valves and I.V. drug addiction are named as settings of infective endocarditis, and a past history of infective endocarditis is an indication for prophylaxis. Mitral valve prolapse is not named; the answer is inferred from the list of underlying cardiac lesions.
- 7 A major criterion for the diagnosis of infective endocarditis is:
(c) Vegetations on echocardiography
 Echo showing vegetations, new regurg or valve ring abscess is a major Duke criterion. A predisposing heart condition and embolic or immune manifestations such as splinter haemorrhages and Osler's nodes are minor criteria.
- 8 Suspect a perivalvular abscess in patients with IE who develop:
(a) A new heart block
 A valve ring abscess lies next to the conduction tissue, so a new heart block in endocarditis should raise suspicion of it; the handout lists both valve ring abscess and aortic valve endocarditis with 2nd degree or complete heart block as indications for surgery. Petechiae, splinter haemorrhages and clubbing are general manifestations.
- 9 Which heart valve is most likely to be involved in infective endocarditis in intravenous drug users?
(d) Tricuspid valve
 In I.V. drug addicts the right valves are affected, esp. the tricuspid, with Staph and Candida as the infecting organisms.
- 10 A 38-year-old male IV drug abuser presents with malaise, fatigue and rigors. His temperature on admission...
(a) Systolic murmur at left sternal border
 In I.V. drug addicts the right valves are affected, esp. the tricuspid. Tricuspid regurgitation gives a pansystolic murmur heard best at the lower sternal edge in inspiration, that is a systolic murmur at the left sternal border.
- 11 The following organism is a cause of culture-negative infective endocarditis:
(c) Fungal organism
 Culture -ve endocarditis may be due to prior use of antibiotics or infection with other organisms e.g. fungi, rickettsia. Strept. viridans, Staph. aureus and Strept. faecalis are the usual culture +ve organisms.
- 12 Which of the following is an echocardiographic indication of surgical management in infective endocarditis?
(b) Valve ring abscess
 Valve ring abscess is one of the conditions that require surgical treatment. Valve replacement is for severe valvular damage, so moderate MR is not enough.
- 13 Which of the following should be avoided in the treatment of infective endocarditis?
(a) Bacteriostatic antibiotics
 Bactericidal drugs in large doses and parenterally should be used, so bacteriostatic antibiotics are avoided. Combinations are used, e.g. penicillin or ampicillin plus streptomycin or gentamycin.
- 14 Commonest organism producing acute bacterial endocarditis is:
(c) Staphylococcus aureus
 Acute bacterial endocarditis is caused by highly virulent organisms, and its regimen is aimed at Staph. aureus and Gram -ve bacilli. Strept. viridans and Strept. faecalis are the organisms of SBE.
- 15 * All of the following may produce hemiplegia by cerebral embolism except:
(d) Right atrial myxoma
 Emboli from the right side of the heart go to the lungs, as the handout shows for right sided endocarditis, so a right atrial myxoma does not cause cerebral embolism. Cerebral embolism with hemiplegia occurs in infective endocarditis and embolic stroke secondary to AF; mitral valve prolapse is a left-sided valve lesion.

16 Which of the following is not a clinical finding associated with infective endocarditis?

(d) Erythema nodosum

Osler's nodules, Roth spots (retinal haemorrhagic infarctions) and splinter haemorrhages are all manifestations of infective endocarditis. Erythema nodosum is not one of them.

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

1 Osler's nodules in patients with infective endocarditis are typically seen in:

(b) Palm & sole

Osler's nodules are tender intracutaneous nodules in the pulps of the fingers and toes and may be on the hands and feet. Palm and sole is the only option on the hands and feet.

Model Answers | Essay Questions

1 Enumerate indications of surgical management of infective endocarditis.

Surgical treatment may be required in the following conditions:

- * **Valve replacement** in: severe valvular damage e.g. MR, AR, and resistant cases.
- * Replacement of **infected prosthesis**.
- * Excision of infected **ductus arteriosus or coarctation**.
- * **Fungal endocarditis**.
- * **Valve ring abscess**.
- * **Aortic valve endocarditis** with 2nd degree or complete heart block (CHB).

2 Summarize the laboratory & radiological investigations needed for diagnosis of infective endocarditis in pediatrics. What are Duke criteria for diagnosis?

(I) Laboratory investigations

① **Blood culture**

- It is the **most important** investigation (+ve in **80%** of cases).
- Samples are cultured under aerobic and anaerobic conditions [and results are looked for after **2 days up to 21 days**].
- Bone marrow culture if blood culture is repeatedly -ve.
- Culture -ve endocarditis may be due to:
 - Prior use of antibiotics.
 - Infection with other organisms e.g. fungi, rickettsia.

② **Blood picture**

- Normocytic anaemia.
- Leucopenia with monocytosis in SBE.
- Polymorphonuclear leucocytosis in ABE.

③ **ESR and C-reactive protein:** increased but not as high as in rheumatic fever.

④ **Urine analysis**

- Microscopic or macroscopic haematuria.
- Proteinuria and casts.

⑤ **Immunological tests**

- Increased gamma globulins.
- False +ve rheumatoid factor.
- False +ve serological tests for syphilis.

Modified Duke's criteria

Diagnosis: **2 major**, or **1 major + 3 minor**, or **5 minor**.

* **Major**

- +ve blood culture:
 - 3 of 3 samples (if more, the majority +ve) taken over **> 1 h**, or
 - typical organism from 2 cultures, or
 - persistently +ve cultures taken **> 12 hs** apart.
- Echo: vegetations, new regurg or valve ring abscess.

* **Minor**

- Predisposing heart condition.
- Fever **$\geq 38^{\circ}\text{C}$** .
- I.V. drug misuse.
- Embolic manifestation.
- Immune manifestation.
- Echo.
- Blood culture if suggestive but not definitive.

N.B. Possible diagnosis [not definitive] if one major & one minor, or 3 minor.

(II) Cardiac investigations

① **Echocardiography**, esp. trans-oesophageal (TOE) echo

- May detect vegetations.
- Detects the underlying cardiac lesion.
- Detects complications of endocarditis e.g. mitral and aortic incompetence.

② **CXR and ECG**

- May help in diagnosis of the underlying cardiac disease.

Pericardial Diseases

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	7	8	9	10	11	12
Answer	d	d	c	d	c	c	b	b	a	b	c	c

Question no.	13	14	15	16	17
Answer	a	d	d	b	b

► Second | Questions with a Note

Question no.	1
Answer	c

► Why this answer?

- All are causes of hemorrhagic pericardial effusion except:*

(d) Heart failure

Haemorrhagic pericarditis occurs in malignant infiltration, uraemia, myocardial infarction and T.B.. Right-sided heart failure causes hydropericardium, an accumulation of transudate.
- All are signs of pericardial tamponade except:*

(d) Pulmonary crepitations

Signs of tamponade include signs of low COP, pulsus paradoxus, congested neck veins and weak distant heart sounds. Pulmonary crepitations are not among them; the chest x-ray shows pulmonary oligoemia.
- Beck's triad is seen in:*

(c) Cardiac tamponade

Cardiac tamponade is the severest form of pericardial effusion, with decreased Bl. Pr. + elevated JVP + muffled heart sounds = Beck's triad.
- All are true about pain of pericarditis except:*

(d) Relieved by lying down, inspiration, cough, swallowing, or...

Pericarditis pain is increased by breathing, rotation of the trunk and lying flat, and decreased by sitting forwards, so (d) is false. Its character is severe, stitching, and it may be referred to the shoulders.
- Which of the following is the most important physical finding when diagnosing acute pericarditis?*

(c) A precordial friction rub.

The pericardial rub is the sign of dry pericarditis: gritty, usually over the bare area and base, and not affected by respiration. Decreased Bl. Pr. belongs to cardiac tamponade.

- 6** *Ewart's sign is seen in:*
(c) Pericardial effusion.
 Ewart's sign is dullness over the left subscapular region due to compression of the base of the left lung by pericardial fluid, a sign of pericardial effusion.
- 7** *The hallmark of benign idiopathic pericardial effusion in ECHO is:*
(b) A clear ECHO-free space.
 Simple fluid shows as a clear echo-free space around the heart; masses, strands, solid parts or a thick pericardium suggest a malignant, purulent or tuberculous effusion. Echo is the most sensitive test for diagnosis of pericardial effusion.
- 8** *Beck's triad consists of the following except:*
(b) Prominent apical pulsation
 Beck's triad is decreased Bl. Pr. + elevated JVP + muffled heart sounds. In effusion the apical pulsations are weak or absent, not prominent.
- 9** *Constrictive pericarditis is characterized by all of the following except:*
(a) Big pulse volume - Pulsus alternans
 Constrictive pericarditis gives signs of low COP and pulsus paradoxus, so a big pulse with pulsus alternans is false. Systemic venous congestion (raised JVP, enlarged liver, ascites praecox) and the pericardial knock are present.
- 10** *Beck's triad consists of the following except:*
(b) Basal back crepitations
 Beck's triad is decreased Bl. Pr. + elevated JVP + muffled heart sounds. Basal crepitations are not part of it; the chest x-ray of effusion shows pulmonary oligoemia.
- 11** *Which of the following drugs is not indicated in treatment of idiopathic acute pericarditis?*
(c) Phenytoin
 Treatment of pericarditis is treatment of the cause and anti-inflammatory drugs e.g. indomethacin, aspirin, ibuprofen; intractable causes may require steroids or colchicine. Phenytoin is not one of them.
- 12** *In cardiac tamponade Beck's triad consists of all the following except:*
(c) Pulsus paradoxus
 Beck's triad is decreased Bl. Pr. + elevated JVP + muffled heart sounds. Pulsus paradoxus is a general sign of tamponade but not part of the triad.
- 13** *The commonest cause of pericarditis is:*
(a) Post-viral
 The commonest causes of pericarditis are idiopathic (mostly viral), tuberculous, rheumatic and uraemic, and idiopathic is probably viral.
- 14** *A 60-year-old man presented with dyspnea, anxiety and retrosternal heaviness. His blood pressure is 80/50...*
(d) Pericardiocentesis
 Decreased Bl. Pr., congested neck veins and distant heart sounds with pericardial fluid are cardiac tamponade. Pericardial aspiration is used for treatment of cardiac tamponade, and there is no place for digoxin.
- 15** * *Pulsus paradoxus is seen in all except:*
(d) Dilated cardiomyopathy
 Pulsus paradoxus is a sign of tamponade and constrictive pericarditis, and it also occurs in acute severe asthma. Dilated cardiomyopathy is the exception, inferred from the line "Rt. sided heart failure: no pulsus paradoxus".
- 16** * *Kussmaul's sign is present in:*
(b) Right ventricular infarction
 Kussmaul's sign is due to failure of the right side of the heart to accept the increased venous return during inspiration. Right ventricular infarction is inferred from this line, as the infarcted right ventricle fails in the same way.

17 * Dressler's syndrome results from:

(b) Autoimmune reaction

Dressler's (post-infarction) syndrome is listed among the causes of pericarditis after myocardial infarction and, being intractable, may require steroids or colchicine. Its autoimmune nature is inferred from this need for anti-inflammatory treatment.

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

1 Constrictive pericarditis is characterized by all of the following except:

(c) JVP is normal

In constrictive pericarditis the neck veins are congested and pulsating and rise further on inspiration, so a normal JVP is the false statement. The small pulse volume, the large liver with early ascites, and the pericardial knock at the left sternal border are all features of constriction.

Model Answers | Essay Questions

1 Enumerate Light criteria for diagnosis of exudative pericardial effusion.

Features of exudate and transudate effusions

	Transudate	Exudate
Proteins	< 3 gm%	> 3 gm%
Sp. gravity	< 1016	> 1016
LDH	< 200 IU/L	> 200 IU/L
Cells (WBCs)	< 1000/cmm	> 1000/cmm

* Stains & cultures including that for T.B. & fungi should be done & examine for malignant cells.

Note The handout does not name Light's criteria; it gives these as the features of an exudate.

2 Mention clinical picture (symptoms and signs) of 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.

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 Bl. 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.

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.

3 Classify the type of this patient's chest pain.

Non-anginal chest pain.

Anginal chest pain

- * Constricting / heavy discomfort to the chest, jaw, neck, shoulders, or arms.
- * Symptoms brought on by exertion, emotion, cold weather and heavy meals.
- * Symptoms relieved within 5 min by rest or nitro-glycerine (GTN).

Number of features	Type of chest pain
All the 3 features	Typical angina
Only 2 features	Atypical angina
0-1 feature	Non-anginal chest pain

Features that make angina less likely

- * Pain that is continuous or lasting for seconds is less likely relates to ischemic origin.
- * **Pleuritic localized chest pain** or that worse with swallowing is mostly non-ischemic pain.

4 What is the most likely diagnosis?

Dry pericarditis.

Pain

Feature	Description
Site and radiation	Retrosternal or precordial pain "may be referred to the upper abd. (along the intercostal nerves) and shoulders (along the phrenic nerves)".
Character	Severe, stitching.
Decreased by	Sitting forwards.
Increased by	Breathing , rotation of the trunk, lying flat (but not related to exertion).
Pleuritic pain	May occur due to associated pleurisy.

ECG

- * **Raised S-T segment (concave upwards) in all ECG leads.**
- * May be inverted or flat T wave.

5 Describe causes of this type of cardiac chest pain.

Cardiac pain

- * **Pericardial disease:** dry pericarditis; massive pericardial effusion.
- * Massive pulmonary embolism and pulmonary infarction.
- * Aortic dissection and aortic aneurysm.
- * Cardiac neurosis:
 - Occurs in neurotic individuals.
 - Site: left inframammary and localized.
 - Radiation: no specific radiation (may be to left arm).
 - Character: stitching or stabbing.
 - No relation to exertion and no relief by rest.
 - Variable duration (seconds or hours).
 - Associated with local tenderness and may be other features of neurosis.
 - Normal cardiac exam.
- * Huge cardiomegaly: may rarely cause retrosternal heaviness.
 - N.B. Huge left atrium: sawing pain "erosion of spine".
- * Mitral valve prolapse, AS, AR.

Note The handout gives one list of causes of cardiac pain; it does not separate non-ischaemic causes (ischaemic heart disease left out here).

Myocardial Disease (Cardiomyopathy)

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

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Answer Key | Multiple Choice Questions

Question no.	1	2	3	4	5	6	7	8	9	10	11	12
Answer	a	e	d	d	c	d	c	g	b	a	c	d

Question no.	13	14
Answer	d	c

► Second | Questions with a Note

Question no.	1
Answer	e

► Why this answer?

- 1** *Drugs to be avoided in HOCM are:*
(a) Vaso-Dilators / Diuretics / Digoxin
 HOCM is treated with avoidance of drugs that increase obstruction: +ve inotropic drugs (e.g. digoxin), and drugs that decrease ventricular volume as diuretics or afterload as vasodilators. B-blockers are part of the treatment.
- 2** *Infiltrative restrictive cardiomyopathy with increased LV wall thickness includes:*
(e) Glycogen storage diseases
 Glycogen storage disease fills the heart muscle cells, so the left ventricular wall becomes thick. Haemochromatosis (iron overload) is a myocardial cause of restrictive cardiomyopathy but usually leaves the wall thickness normal, and endomyocardial fibrosis, radiation and the idiopathic type stiffen the heart without thickening it.
- 3** *As regard treatment of restrictive cardiomyopathy which is wrong:*
(d) Digoxin is the preferred drug in RCM and AF
 Digoxin is not the preferred drug in restrictive cardiomyopathy, as toxicity occurs easily, especially in amyloid heart. Treatment is of heart failure, so diuretics, risk-factor control, anticoagulation in AF and SGLT2i, the only drug class with mortality benefit in HFpEF, are correct.
- 4** *The following is incorrect as regard HOCM*
(d) Autosomal recessive inherited disorder
 Hypertrophic cardiomyopathy is familial: autosomal dominant, not recessive. Sudden death is a complication, HOCM is a cause of cardiac syncope, and many patients have no symptoms early in life.

- 5 *The prognosis is poor for dilated cardiomyopathy patients with:*
(c) S3 Gallop
A persistent S3 gallop reflects a high filling pressure and a severely failing ventricle, and it is a recognised sign of poor prognosis in dilated cardiomyopathy. A diffuse weak apex and soft heart sounds are usual findings of the disease and do not by themselves predict outcome. Functional MR and TR are also usual in dilated cardiomyopathy; marked MR adds risk, but the classic answer for poor prognosis is the S3 gallop.
- 6 *The least likely etiology of dilated cardiomyopathies is:*
(d) Hemochromatosis
Viral infection and peripartum are listed causes of dilated cardiomyopathy, and dilated or ischemic cardiomyopathy causes left ventricular dilatation. Haemochromatosis is a cause of restrictive cardiomyopathy, so it is the least likely.
- 7 *The least likely diagnosis of restrictive cardiomyopathy is:*
(c) Systemic Lupus
Amyloidosis, haemochromatosis and sarcoidosis are myocardial causes of restrictive cardiomyopathy. SLE is listed under collagen diseases causing dilated cardiomyopathy.
- 8 *Which of the following is the most probable diagnosis?*
(g) Obstructive cardiomyopathy
Echo showing asymmetrical septal hypertrophy (ASH) is diagnostic of hypertrophic cardiomyopathy. Jerky pulse, giant A wave and a midsystolic murmur along the left sternal border also fit, with sudden death as a complication.
- 9 *The following is absent in patient with restrictive cardiomyopathy:*
(b) Left ventricular dilatation.
Restrictive cardiomyopathy is a non-dilated, non-obstructive cardiomyopathy: the ventricles are stiff with a normal or small cavity, while both atria dilate. The wall can be thickened, for example in amyloidosis, so hypertrophy is not always absent. Left ventricular dilatation is the feature that is absent.
- 10 *Hypertrophic obstructive cardiomyopathy is the most common cause of sudden cardiac death in young. Its...*
(a) Autosomal dominant
Hypertrophic cardiomyopathy is familial: autosomal dominant, thus screening of first degree relatives and genetic counselling are used.
- 11 *Which of the following drug/s should be avoided in patients with hypertrophic obstructive cardiomyopathy:*
(c) Amlodipine.
Drugs that decrease afterload as vasodilators increase the obstruction and are avoided; amlodipine acts mainly as a vasodilator. B-blockers (metoprolol) and rate-slowing Ca-channel blockers like verapamil (diltiazem) are used in treatment.
- 12 *The following could be a reversible cause of dilated cardiomyopathy:*
(d) All could be reversible.
In peripartum cardiomyopathy half of patients recover completely and most of the rest improve. Stress cardiomyopathy and tachycardia-induced cardiomyopathy also recover when the stress or the fast rate is controlled, so all could be reversible.
- 13 *Which of the following infectious agents have been associated with the development of inflammatory...*
(d) All of the above
Diphtheritic and viral myocarditis are listed among specific fevers affecting the heart, and coxsackie B virus and Chagas disease (caused by *Trypanosoma cruzi*) are infective causes of dilated cardiomyopathy. So all are correct.
- 14 *Deficiency of which one of the following trace elements is implicated as a cause of cardiomyopathy?*
(c) Selenium
Nutritional causes of dilated cardiomyopathy are Beri Beri and selenium deficiency [Keshan disease].

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

- 1 Which of the following is the most probable diagnosis?

(e) Dilated cardiomyopathy

A woman with a past mastectomy has had breast cancer, and adriamycin is a toxic cause of dilated cardiomyopathy. The displaced apex fits biventricular enlargement, the irregular pulse fits AF, and the pansystolic murmurs fit functional MR and TR.

Model Answers | Essay Questions

- 1 Enumerate causes and categories of dilated cardiomyopathy.

Aetiology of dilated cardiomyopathy

- ① Idiopathic.
- ② **Infective:**
 - Viral esp. coxsackie B virus.
 - Chagas disease.
- ③ **Collagen diseases** e.g. SLE.
- ④ **Endocrinal:**
 - Myxoedema.
 - D.M.
 - Acromegaly.
- ⑤ **Peripartum cardiomyopathy:**
 - In the last trimester or within **6 months** of delivery.
 - Half of patients recover completely & most of the rest improving.
 - Avoid further pregnancy to decrease risk of recurrence.
 - ACE inhibitors are contraindicated in pregnancy.
- ⑥ **Toxic:**
 - Alcohol.
 - Adriamycin.
- ⑦ **Neurologic:**
 - Duchenne's myopathy.
 - Myotonia dystrophica.
 - Friedreich's ataxia.
- ⑧ **Familial.**
- ⑨ **Nutritional:** Beri Beri, selenium deficiency [Keshan disease].

- 2 Enumerate the investigations of dilated cardiomyopathy.

Investigation	Findings
Chest x-ray	Cardiac enlargement involving all the chambers; pulmonary congestion.
ECG	Low voltage; depressed ST segment; flat or inverted T-wave; may be A.F.
Echocardiography and Doppler U/S	Dilated ventricles with no hypertrophy; decreased contractility; MR and TR.

3 Demonstrate investigations of hypertrophic cardiomyopathy.

Investigation	Findings
Chest x-ray	Left ventricular hypertrophy; pulmonary congestion.
ECG	Left ventricular enlargement; right ventricular hypertrophy if there is associated R.V. outflow tract obstruction.
Echocardiography and Doppler U/S	Hypertrophy of the left ventricle with the septal wall thickness exceeds the free wall thickness (asymmetrical septal hypertrophy, ASH): diagnostic .
Microscopic examination	Myocardial fibre disarray "malalignment" [whorls of cells].

4 What is the management?

Treatment

- ① Treatment of H.F. with **avoidance of drugs that increase obstruction**, i.e. +ve inotropic drugs and drugs that decrease ventricular volume as diuretics or afterload as vasodilators.
- ② **B-blockers**: e.g. propranolol decrease tachycardia: "better filling of left ventricle" and prevent syncopal attacks.
- ③ **Ca-channel blockers** e.g. verapamil improve diastolic filling.
- ④ **Surgical treatment**: resection of the septum (myotomy or myomectomy) or laser myoplasty.

Preventive measures

Familial: autosomal dominant, thus screening of first degree relatives and genetic counselling are used as preventive measures.

Systemic 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	7	8	9	10	11	12
Answer	a	c	c	b	d	a	d	d	b	b	b	a

Question no.	13	14	15	16	17	18	19	20	21	22	23	24
Answer	c	a	d	d	d	d	b	c	c	c	c	d

Question no.	25	26	27	28	29	30	31
Answer	c	d	c	d	d	c	d

► Second | Questions with a Note

Question no.	1	2	3
Answer	c	d	a

► Why this answer?

- From the following scenarios, who is NOT suspected to have secondary hypertension?
(a) A 33-year-old obese woman presenting with BP 160/90 and a...
Hereditary and obesity are predisposing factors of essential HPN, so the young obese woman with a family history fits essential HPN. Sudden development, HPN resistant to treatment and onset after 50 years are clues to renal artery stenosis, a secondary cause.
- Which of the following statements regarding the treatment of hypertensive emergencies and urgencies is FALSE?
(c) All patients with marked blood pressure elevation should...
Only patients with acute target organ damage (emergencies) need hospitalization, preferably in ICU. Urgencies have no acute end organ damage and blood pressure is lowered over 1-2 days.
- Regarding severe elevation of blood pressure, which is incorrect?
(c) Needs only reassurance of the patient and relatives.
Severe elevation of blood pressure always needs treatment, never reassurance alone. Without acute end organ damage it is an urgency, lowered over 1-2 days; with acute Lt.v. failure and pulmonary oedema it is an emergency needing hospital and rapid reduction.

- 4 All of the following antihypertensive drugs are safe in pregnancy except:
(b) ACEI
 ACE inhibitors are on the 'Avoid' list of HPN with pregnancy. Alpha methyl dopa and CCBs (not at labour) are on the 'Use' list, and labetalol is a safe combined alpha and beta blocker.
- 5 Which of the following patients can be typically managed as an outpatient?
(d) Patient with blood pressure 200/110 and has no signs of...
 No acute end organ damage means a hypertensive urgency, where blood pressure can be lowered over 1-2 days, so the patient can be treated as an outpatient. Pulmonary oedema and acute chest pain are emergencies that need hospitalization, preferably in ICU.
- 6 Presence of acute chest pain in hypertensive patient is consistent with:
(a) Hypertensive emergency.
 Acute chest pain in a hypertensive patient points to unstable angina, myocardial infarction or a dissecting aortic aneurysm, and these are counted as hypertensive emergencies.
- 7 Paroxysmal hypertension is classically found in:
(d) Pheochromocytoma
 Pheochromocytoma gives paroxysmal HPN.
- 8 A 25-year-old male presented with episodic rise in heart rate and blood pressure with sweating, flushing and...
(d) Pheochromocytoma.
 Attacks of high blood pressure with fast pulse, sweating, flushing and tremors fit pheochromocytoma, which gives paroxysmal HPN and is an endocrinal cause of secondary HPN.
- 9 A 30-year-old pregnant female patient is diagnosed with hypertension. Which of the following is safe for her?
(b) Labetalol.
 ACE inhibitors are on the 'Avoid' list of HPN with pregnancy, while alpha methyl dopa, hydralazine and CCBs are on the 'Use' list; ARBs act on the same angiotensin system. Labetalol, a combined alpha and beta blocker, is a standard safe drug in pregnancy.
- 10 What is the target of blood pressure control for most patients with hypertension according to 2024 ESC...
(b) Systolic BP 120–129 mmHg and diastolic BP 70–79 mmHg.
 For most treated hypertensive patients the target is a systolic pressure of 120-129 mmHg with a diastolic pressure of 70-79 mmHg, as long as the patient tolerates it.
- 11 * The following facts are true regarding hypertension except:
(b) Treatment of hypertension can be stopped once controlled.
 This is inferred from the handout: sudden withdrawal of a drug such as clonidine causes a hypertensive emergency, and clonidine gives rebound hypertension, so treatment is not stopped once controlled. HPN may be asymptomatic, and every drug group has side effects.
- 12 Presence of acute chest pain in hypertensive patient is consistent with:
(a) Hypertensive emergency
 Acute chest pain points to unstable angina, myocardial infarction or a dissecting aortic aneurysm, which are hypertensive emergencies. Hypertensive urgency has no acute end organ damage.
- 13 The antihypertensive drug contraindicated in diabetic patients is:
(c) Thiazide and beta-blocker combination
 In HPN with DM, non-selective B-blockers and thiazides are on the 'Avoid' list, and a thiazide is used only added to ACE inhibitors or ARBs. ACE inhibitors and ARBs are on the 'Use' list, and metformin is not an antihypertensive.
- 14 The antihypertensive drug used in diabetic patients is:
(a) ACEI
 ACE inhibitors ± thiazide come first on the 'Use' list of HPN with DM. Non-selective B-blockers and thiazides are on the 'Avoid' list, and metformin is not an antihypertensive.

- 15 Antihypertensive drug not preferred with diabetes mellitus is:
(d) Thiazides and beta-blocker combination
 Non-selective B-blockers and thiazides are on the 'Avoid' list of HPN with DM, while ACE inhibitors, ARBs and non-DHP CCBs are on the 'Use' list.
- 16 Common causes of resistant hypertension are:
(d) All the above
 Salt intake is a predisposing factor of HPN and corticosteroids are a drug cause of secondary HPN, so both keep the blood pressure high in spite of treatment. Not taking the drugs regularly is the commonest cause of apparent resistance.
- 17 Common causes of resistant hypertension are all the following except:
(d) Smoking
 Non-adherence, excess salt and long steroid therapy are common causes of resistant HPN. Smoking is a cardiovascular risk factor, not a usual cause of resistance to treatment.
- 18 Which of the following is considered a referral criterion for a hypertensive patient?
(d) Hypertensive crisis
 A hypertensive crisis needs hospitalization and preferably admission to ICU, so it must be referred. Good response, controlled blood pressure and a family history are managed in the clinic.
- 19 Hypertension increases the risk of the following except:
(b) Asthma
 HPN leads to heart failure from pressure overload on the Lt.v., stroke from cerebral thrombosis or hge, and renal failure from nephrosclerosis. Asthma is not a complication of HPN.
- 20 A 60-year-old woman has persistently elevated blood pressure, with a reading of 155/84 mmHg. Which of the...
(c) Thiazide diuretic
 Persistent blood pressure of 155/84 at age 60 is confirmed hypertension, mainly systolic, and it needs drug treatment together with lifestyle change. The handout recommends diuretics for hypertension in older patients, and a thiazide is a standard first-line drug. Lifestyle change is always advised, but it is not 'enough' on its own here. Alpha blockers and hydralazine are not first-line drugs.
- 21 A 60-year-old man has untreated hypertension (168/106 mmHg; blood pressure has been elevated on at least 3...
(c) Initiate with a 2-agent combination pill.
 168/106 is stage 2, which needs drug therapy in every risk group. A diastolic of 105-114 is moderate HPN, and moderate HPN needs 2 drugs, so treatment starts with a 2-drug combination.
- 22 A woman with chronic hypertension has blood pressure that remains above target despite a daily regimen of...
(c) Resistant hypertension
 Blood pressure above target in spite of three drugs of different classes in full doses, one of them a diuretic, is resistant hypertension. 'HPN resistant to tt' is also a clue to renal artery stenosis.
- 23 Which of the following is an indication for referral of a hypertensive patient?
(c) Continuous abdominal bruit.
 A bruit in the loin or lateral to the umbilicus is a clue to renal artery stenosis, a secondary cause. Gradual onset, good response and onset in middle age fit essential HPN.
- 24 Which of the following is a recommended lifestyle modification?
(d) Maintain normal weight (BMI 18.5-24.9 kg/m²)
 Weight reduction in obese patients is a dietary measure in the treatment of HPN, so keeping a normal weight is recommended. Salt restriction is advised and 10 g/day is not a real restriction; more coffee, tea or meat is not advised.
- 25 All of the following suggest secondary hypertension except:
(c) Presence of end organ damage
 Target organ damage is a complication of any HPN, essential or secondary. Young age at onset and HPN resistant to treatment and long corticosteroid use point to a secondary cause.

26 Secondary hypertension is suspected when:

(d) All of the above.

Onset at a young or old age, sudden development and HPN resistant to treatment are all clues to renal artery stenosis, a secondary cause. The handout gives the ages as below 25 or above 50 years.

27 * To diagnose hypertension, repeated measures are required except if:

(c) a and b

This is inferred from the handout: a diastolic above 130 mmHg with retinal hges is accelerated or malignant HPN, a severe form treated at once. Otherwise the measurement should be done at least twice on at least two visits.

28 To diagnose hypertension-induced end organ damage, we need all the following investigations except:

(d) Lipid profile.

A lipid profile looks for an associated risk factor of atherosclerosis, not for target organ damage. Echo shows Lt.v. hypertrophy and fundus examination shows retinopathy, and carotid Doppler shows vascular damage.

29 * When discussing facts regarding hypertension during patient education, which of the following is true?

(d) Hypertension requires life-long monitoring at 3 to 6...

This is inferred from the handout: sudden withdrawal of a drug such as clonidine causes a hypertensive emergency, so treatment is continuous with life-long follow-up. HPN may be asymptomatic, so symptoms cannot be relied on.

30 All of the following produce systemic hypertension except:

(c) Addison's disease

Polycystic kidney, pheochromocytoma and Conn's syndrome are listed causes of secondary HPN. Addison's disease is adrenal failure with loss of cortisol and aldosterone, so it lowers the blood pressure.

31 Accelerated hypertension should not have:

(d) Papilledema

Papilloedema is grade IV retinopathy and the mark of malignant HPN. Accelerated HPN has retinal hges and exudates (grade III), which include silver wiring and kinking of veins at AV crossing.

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

1 All of the following are correct regarding management of severe elevation of blood pressure except:

(c) Sublingual captopril is used in hypertensive urgency

Urgency has no acute end organ damage and blood pressure is lowered over 1-2 days without admission, emergencies need hospitalization, and B-blockers act by decreasing the cardiac output. A quick sublingual drop is not needed in urgency, so (c) is the expected false statement.

2 * A 49-year-old postmenopausal obese woman has type 2 diabetes mellitus. Her blood pressure readings average...

(d) Recommend lifestyle changes and revisit in 3 months

This is inferred from the risk table: 136/86 is pre-hypertension and DM puts her in Risk Group C, where life style modification is the base of treatment. Both drugs offered are unsuitable, because thiazides and non-selective B-blockers are on the 'Avoid' list of HPN with DM.

3 Which of the following features does NOT suggest secondary hypertension?

(a) Age <30 years and >55 years

Sudden development of HPN and HPN resistant to treatment suggest a secondary cause, and so does a sudden severe rise in a controlled patient. Option (a) fits as the key only if read as onset between 30 and 55 years, the middle age of essential HPN.

1 List the risk factors of essential hypertension and outline the causes of secondary hypertension.

Essential (primary) hypertension (more than 90% of cases)

- * It is of unknown aetiology and occurs in middle and old ages.
- * **Predisposing factors:** heredity; obesity; salt intake; stress.

Factors that may play a role in the etiology:

- ① **Sympathetic overactivity:** excessive stimulation of sympathetic nervous system produces V.C. and increases peripheral vascular resistance.
- ② **Renin secretion:** increased renal secretion of renin, due to unknown cause, which converts angiotensinogen into angiotensin I, which is then converted to angiotensin II, leading to V.C. and secretion of aldosterone, which leads in turn to salt and water retention.
- ③ **Hormonal factors:** increased secretion of a mineralocorticoid from the supra-renal cortex causing salt and water retention.
- ④ **Multifactorial theory:** combination of the above.

Secondary HPN (5-10% of patients)

① Renal causes

- Renal parenchymal (75% of renal causes)
 - Acute and chronic GN.
 - Chronic pyelonephritis.
 - Hydronephrosis.
 - Polycystic kidney.
 - Diabetic nephropathy.
 - Collagen diseases e.g. SLE, PAN, scleroderma.
 - Gout nephropathy, nephrocalcinosis, amyloidosis.
- Reno-vascular (renal artery stenosis) (25% of renal causes)
 - Atherosclerosis of renal artery.
 - Fibromuscular dysplasia.
 - Vasculitis e.g. PAN.
 - Renal artery thrombosis and embolism.

② Endocrinal causes

- Cushing syndrome, Conn's syndrome and CAH.
- Pheochromocytoma.
- Myxoedema.
- Hyperparathyroidism.
- Acromegaly.

③ Neurological causes

- Increased I.C.T.
- Lesions of the medulla e.g. bulbar poliomyelitis.
- Lesions of hypothalamus.
- Polyneuritis: acute porphyria, chronic lead poisoning.

④ Drugs

- Contraceptive pills.
- Corticosteroids and ACTH.
- Carbenoxolone.
- Liquorice.
- MAOI.

⑤ Miscellaneous causes

- Coarctation of aorta.
- Toxaemia of pregnancy.
- PRV.
- Hypercalcaemia.
- PAN.

2 Define the term hypertensive emergency and explain how this differs from hypertensive urgency.

Hypertensive emergencies

- * Hypertensive encephalopathy.
- * Subarachnoid and cerebral hge.
- * Acute Lt.v. failure and pulmonary oedema.
- * Dissecting aortic aneurysm.
- * Acute renal failure.

N.B. Unstable angina, myocardial infarction and toxaemia of pregnancy can be considered in the item of hypertensive emergencies.

Treatment of hypertensive emergencies

- ① Hospitalization and preferably admission to ICU.
- ② Rapid reduction of blood pressure to approximately **100 mmHg** diastolic within **one hour** using rapidly acting drugs.

Hypertensive urgencies

- * No acute end organ damage.
- * Blood pressure can be lowered over **1-2 days**.

Time frames in treatment of HPN

Situation	What is done	Time
Hypertensive emergencies	Rapid reduction of blood pressure to approximately 100 mmHg diastolic	Within one hour
Hypertensive urgencies	No acute end organ damage; blood pressure can be lowered	Over 1-2 days

Note The handout lists the hypertensive emergencies but gives no worded definition, answer incomplete.

3 Define hypertensive emergency, mention "4" causes of secondary hypertension in young adults, and treatment of hypertensive crises.

Hypertensive emergencies

- * Hypertensive encephalopathy.
- * Subarachnoid and cerebral hge.
- * Acute Lt.v. failure and pulmonary oedema.
- * Dissecting aortic aneurysm.
- * Acute renal failure.

N.B. Unstable angina, myocardial infarction and toxæmia of pregnancy can be considered in the item of hypertensive emergencies.

N.B. Hypertensive urgencies: no acute end organ damage; blood pressure can be lowered over **1-2 days**.

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 - Diabetic nephropathy.
 - Collagen diseases e.g. SLE, PAN, scleroderma.
 - Gout nephropathy, nephrocalcinosis, amyloidosis.
- Reno-vascular (renal artery stenosis) (25% of renal causes)
 - Atherosclerosis of renal artery.
 - Fibromuscular dysplasia.
 - Vasculitis e.g. PAN.
 - Renal artery thrombosis and embolism.

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- Lesions of hypothalamus.
- Polyneuritis: acute porphyria, chronic lead poisoning.

④ Drugs

- Contraceptive pills.
- Corticosteroids and ACTH.
- Carbenoxolone.
- Liquorice.
- MAOI.

⑤ Miscellaneous causes

- Coarctation of aorta.
- Toxaemia of pregnancy.
- PRV.
- Hypercalcaemia.
- PAN.

Treatment of hypertensive emergencies

① Hospitalization and preferably admission to ICU.

② Rapid reduction of blood pressure to approximately **100 mmHg** diastolic within **one hour** using rapidly acting drugs e.g.:

Drug	Dose
Tridil	0.6 mg/h.
Na nitroprusside	0.5-10 ug/kg/min I.V.
Nifedipine	10-20 mg sublingually.
Diazoxide	50-100 mg/5-10 min I.V.
Hydralazine	10-20 mg repeated every 30 min I.V.
Alpha methyl dopa	250-500 mg/6 h. I.V.

- ① I.V. frusemide: is better given with these drugs to assist in control of blood pressure and prevent fluid retention.
- ② Venesection is rarely needed in resistant cases.
- ③ Following control of blood pressure, oral antihypertensive treatment should be started usually with a diuretic, B-blocker and vasodilator.
- ④ Further treatment according to the emergency:
 - In hypertensive encephalopathy:
 - Cerebral dehydrating measures: I.V. mannitol or conc. glucose; Mg. sulphate per rectum.
 - Anticonvulsants: I.V. diazepam and phenytoin.
 - Lumbar puncture is rarely required.
 - In subarachnoid and cerebral hge: see neurology.
 - In acute Lt.v. failure: see treatment of acute pulmonary oedema.
 - In dissecting aortic aneurysm: see later.
 - In renal failure: refer to nephrology.

N.B. Urgent enquiry into the cause of hypertensive emergency e.g. acute GN, pheochromocytoma, malignant HPN, sudden withdrawal of drug e.g. clonidine, and eclampsia.

Note The handout gives no worded definition of hypertensive emergency and does not separate the causes of secondary HPN in young adults, answer incomplete.

4 What is the definition and clinical presentation of hypertensive emergencies?

Hypertensive emergencies

- * Hypertensive encephalopathy.
- * Subarachnoid and cerebral hge.
- * Acute Lt.v. failure and pulmonary oedema.
- * Dissecting aortic aneurysm.
- * Acute renal failure.

N.B. Unstable angina, myocardial infarction and toxemia of pregnancy can be considered in the item of hypertensive emergencies.

N.B. Hypertensive urgencies: no acute end organ damage; blood pressure can be lowered over **1-2 days**.

Hypertensive encephalopathy

Pathogenesis: sudden severe rise of blood pressure leading to rush of blood into the cerebral circulation due to failure of autoregulation and causing cerebral oedema and petechial hge.

Clinical picture:

- * Severe headache.
- * Vomiting, blurring of vision.
- * Drowsiness, convulsions and lastly coma (usually without signs of lateralization).

Note The handout gives no worded definition and describes the clinical picture of hypertensive encephalopathy only, answer incomplete.

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:

- 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.

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- 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.

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- 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.

(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.

Pulmonary Embolism & DVT

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

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Answer Key | Multiple Choice Questions

Question no.	1	2	3	4	5	6	7	8	9	10
Answer	b	c	c	d	d	a	a	e	c	d

► Why this answer?

- Which of the following is considered an indirect marker of DVT?

(b) D-dimer

D-dimer is raised in thromboembolism but is not specific; it is a breakdown product of fibrin, so it points to a clot indirectly.
- In suspected cases of deep vein thrombosis, which of the following is the most appropriate investigation?

(c) Duplex ultrasound scan

Colored Doppler and venography are both listed for diagnosis of DVT; duplex ultrasound is preferred because it is accurate and non-invasive. D-dimer is not specific, and CT pulmonary angiography looks for a lung embolus.
- Treatment of Deep venous thrombosis includes all of the following except:

(c) Systemic vasodilator medications

Thrombolytic therapy and heparin iv, and inferior vena cava interruption by filter or umbrella, are all part of treatment. Vasodilators are not listed in the treatment of thromboembolism.
- A blood clot can result from:

(d) All of the above.

The predisposing factors of DVT are Virchow's triad: slow circulation, hyper-coagulability and injury of vascular endothelium, so all three are correct.
- What is the definitive diagnosis of pulmonary embolism?

(d) CT Pulmonary angiography

Pulmonary angiography is the most specific test for pulmonary embolism; CT pulmonary angiography shows the clot directly. The ventilation perfusion lung scan is only more accurate than the perfusion scan alone.
- If pulmonary embolism is unlikely with Wells score < 4, we will order the following test, which is a good...

(a) D-dimer

D-dimer is raised in embolism but not specific: it is also raised in infection, pregnancy, malignancy and post operative. So a normal result rules embolism out when the probability is low.
- The diagnostic tool that is used in DVT:

(a) Duplex US

Investigations for diagnosis of DVT are e.g. colored Doppler & venography or plethysmography; duplex US is the colored Doppler study of the veins.

8 The following is not an indication for prophylaxis against pulmonary embolism:

(e) Dental procedures

Prolonged rest in bed (fracture, surgery) and malignancies are predisposing factors, and hip or knee surgery and malignancy are candidates for prophylaxis. A dental procedure is not among them.

9 Treatment of Deep venous thrombosis include all of the following except:

(c) Systemic vasodilator medication

Thrombolytic therapy, heparin iv and oral anticoagulant (e.g. warfarin) are all part of treatment. Vasodilators are not listed in the treatment of thromboembolism.

10 A patient with DVT and active hematemesis, what is the appropriate treatment?

(d) IVC filter

Inferior vena cava interruption (by filter or umbrella) is performed when anticoagulation is contraindicated or ineffective. Active bleeding contraindicates thrombolysis, heparin and warfarin.

Model Answers | Essay Questions

1 Mention the predisposing factors and risk factors of deep vein thrombosis (DVT).

Predisposing factors (Virchow's triad)

① Slow circulation:

- Prolonged rest in bed (e.g. fracture, surgery, or paralyzed limb).
- Heart failure.
- Varicose veins.
- Obesity, pregnancy.

② Hyper-coagulability:

- Polycythemia, thrombocytosis.
- Dehydration.
- C.C. pills (combined contraceptive pills), pregnancy.
- Malignancies: leukaemia; myeloma; paraneoplastic.
- Nephrotic syndrome.
- Decrease of protein C, S, & decrease of factor V Leiden & decrease of antithrombin III, increase of PAI-1.
- Behcet, vasculitis & APA, PNH, SCA, homocystinuria, DIC and TTP.

③ Injury of vascular endothelium:

- Inflammation.
- Trauma.

2 List the predisposing factors and high-risk conditions of deep venous thrombosis.

Predisposing factors (Virchow's triad)

① Slow circulation:

- Prolonged rest in bed (e.g. fracture, surgery, or paralyzed limb).
- Heart failure.
- Varicose veins.
- Obesity, pregnancy.

② Hyper-coagulability:

- Polycythemia, thrombocytosis.
- Dehydration.
- C.C. pills (combined contraceptive pills), pregnancy.
- Malignancies: leukaemia; myeloma; paraneoplastic.
- Nephrotic syndrome.
- Decrease of protein C, S, & decrease of factor V Leiden & decrease of antithrombin III, increase of PAI-1.
- Behcet, vasculitis & APA, PNH, SCA, homocystinuria, DIC and TTP.

③ Injury of vascular endothelium:

- Inflammation.
- Trauma.

Candidates for venous thrombosis prophylaxis

Risk	Candidates
Moderate risk of DVT	Major surgery in patients > 40 years or with other risk factor; major medical illness e.g. HF, chest infection, malignancy, IBD.
High risk of DVT	Hip or knee surgery; major abdominal or pelvic surgery for malignancy or with history of DVT or known thrombophilia.

3 Enumerate the risk factors and outline the clinical presentations of acute pulmonary embolism.

Types & source of emboli

I - Thrombo-emboli

* A. Peripheral veins (DVT): predisposing factors (Virchow's triad):

- Slow circulation: prolonged rest in bed (e.g. fracture, surgery, or paralyzed limb); heart failure; varicose veins; obesity, pregnancy.
- Hyper-coagulability: polycythemia, thrombocytosis; dehydration; C.C. pills, pregnancy; malignancies: leukaemia, myeloma, paraneoplastic; nephrotic syndrome; decrease of protein C, S, & decrease of factor V Leiden & decrease of antithrombin III, increase of PAI-1; Behcet, vasculitis & APA, PNH, SCA, homocystinuria, DIC and TTP.
- Injury of vascular endothelium: inflammation; trauma.

* B. Right side of the heart:

- Vegetations of infective endocarditis.
- Mural thrombi over myocardial infarction.
- Arrhythmia esp. AF.

* C. Paradoxical emboli: emboli passing from the left side of the heart through a defect - e.g. VSD or ASD - to the right side of the heart.

II - Other types of emboli

- * Amniotic fluid.
- * Fat.
- * Parasitic or malignant cells emboli.
- * Air.
- * Foreign material.

Clinical picture

Presentations of pulmonary embolism are variable depending on: size of embolus; infected or not; number of emboli; underlying lung disease.

A. Minute embolus

- * Asymptomatic.
 - * Recurrent minute emboli may cause thromboembolic pulmonary hypertension & subacute cor pulmonale.
- B. Medium sized embolus:** causes pulmonary infarction (if occlusion involves **< 65%** of pulmonary vascular bed).
- * Sudden pleuritic stitching chest pain.
 - * Dyspnea, cough, hemoptysis.
 - * In the 2nd day, fever & jaundice may occur.
 - * Pleural rub or hemorrhagic pleural effusion may be present.
 - * There may be features of DVT: pain, tenderness along the course of the thrombosed vein, edema, slight fever + Homans' sign.
 - **Homans' sign:** pain in the calf on dorsiflexion of the foot.
 - **Loewenberg's sign:** pain in the calf on elevating pressure by sphygmomanometer.

N.B. Platypnea in embolism of upper part of lung.

C. Big emboli: occlusion involves **65 - 85%** of the pulmonary vascular bed: massive pulmonary embolism; occlusion involves **> 85%** of the vascular bed: sudden death.

- * Chest pain: sudden severe retrosternal pain due to sudden decrease of coronary blood flow or sudden pericardial distension due to sudden RV dilatation.
- * Dyspnea, manifestations of low CO & may be central cyanosis.
- * Rapid appearance of manifestations of pulmonary hypertension & right ventricular failure (**acute cor pulmonale**).
- * Cardiogenic shock may occur.
- * There may be features of DVT.

N.B. Infected embolus may lead to pneumonia & lung abscess.

Note The handout has no heading "risk factors"; the answer is its Types & source of emboli.

4 How to manage a patient with confirmed acute pulmonary embolism with hemodynamic compromise (high risk)?

Therapeutic

- ① **Hospitalization, preferably in ICU.**
- ② **If the patient is hemodynamically unstable**, i.e. in the presence of shock or heart failure:
 - a) **Thrombolytic therapy:**
 - Using streptokinase, urokinase, or tissue plasminogen activator given intravenously or intrapulmonary.
 - Streptokinase is given as follows: **250,000 - 500,000 I.U.** iv bolus followed by 100,000 I.U./hour for 24 - 72 hours continuously by iv infusion.
 - This is followed by:
 - Heparin (HMWH) iv for 5 days or LMWH SC [fractionated heparin has a longer half life than SH "standard heparin"] (PTT [1.5 - 2.5]).
 - Heparin & oral anticoagulants for 5 days, INR [2 - 3].
 - Oral anticoagulant (e.g. warfarin) for **3 months** at least [up to 6 months].
 - b) **Pulmonary embolectomy** (Trendelenburg's operation): performed in some cases when thrombolytic therapy is contraindicated.

③ Symptomatic treatment:

- Analgesics e.g. pethidine 50 mg IV or IM. Morphine should be avoided because of its respiratory depressant action.
- Oxygen inhalation.
- Mechanical ventilation may be required in severe cases.

④ Treatment of complications:

- Shock.
- Right ventricular failure.

⑤ Correction of predisposing factors of thromboembolism.

5 Answer the following about this patient:

(a) Risk factors for DVT

- * Slow circulation: prolonged rest in bed (e.g. surgery).
- * Hyper-coagulability: malignancies.
- * Moderate risk of DVT: major surgery in patients > 40 years or with other risk factor.
- * High risk of DVT: major abdominal or pelvic surgery for malignancy or with history of DVT.

(b) Prophylactic measures

- * Prophylaxis & treatment of predisposing factors of embolism.
- * Early postoperative & postpartum ambulation.
- * Prophylactic anticoagulation in susceptible patients using heparin **5000 units / 8 hours** S.C. "minidose".
- * Fondaparinux & ximelagatran.
- * Intermittent pneumatic compression stockings: prevent post-operative thrombosis.

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	7	8	9	10	11	12
Answer	d	d	c	a	b	a	a	c	d	a	a	c

Question no.	13	14	15	16	17	18	19	20	21	22	23	24
Answer	a	d	c	a	a	b	a	b	b	a	d	d

Question no.	25	26	27	28	29	30	31	32
Answer	d	d	b	b	c	d	d	a

► Second | Questions with a Note

Question no.	1
Answer	a

► Why this answer?

- Which of the following is the best emergency treatment of patient with complete heart block?
(d) Temporary pacemaker
The emergency treatment of an unstable bradyarrhythmia is temporary pacing, transcutaneous or transvenous. Atropine is usually temporary, for patients with no temporary pacing; DC shock is for tachyarrhythmias and amiodarone slows conduction further.
- In a patient with daily rapid palpitations of sudden onset and offset and normal resting ECG in the...
(d) Ambulatory ECG monitoring.
The resting ECG is normal between attacks, so the rhythm must be recorded during an attack; with daily attacks, short term ECG recording by Holter monitoring will catch one.
- Which of the following arrhythmia is characterized with AV dissociation (variable PR interval)?
(c) 3rd degree heart block
In complete (third degree) heart block no atrial impulse reaches the ventricles, so P waves and QRS complexes are independent and the PR interval varies (AV dissociation). In Mobitz type II the conducted beats keep the same PR interval; the handout lists both under AV nodal blocks.

- 4 In a 34 years old male patient with atrial fibrillation and uncontrolled ventricular rate for 4 days with no...
(a) Propafenone
 AF for 4 days is more than 48 hours, and without transesophageal echo restoring sinus rhythm carries a high risk of thromboembolism, so rate control is chosen. Propafenone restores sinus rhythm, so it is avoided, while diltiazem (rate control) and rivaroxaban (anticoagulation) fit the plan.
- 5 Which of the following is a differential diagnosis of narrow complex arrhythmia?
(b) Atrial flutter.
 Tachycardia is classified by QRS width and site of origin. Atrial flutter is supraventricular, so the QRS is narrow (< 120 msec); ventricular tachycardia, ventricular flutter and pre-excited AF give wide QRS complexes.
- 6 Which of the following is not a line of treatment of supraventricular tachycardia.
(a) IV heparin
 IV adenosine, IV verapamil and IV beta blockers such as metoprolol are AV node blocking strategies for supraventricular tachycardia. Heparin is a parenteral anticoagulant, given in atrial flutter and AF, not in supraventricular tachycardia.
- 7 The following drug is recommended for rhythm control of a patient with recent onset atrial fibrillation with...
(a) Propafenone
 With no structural heart disease, rhythm control is by class IC drugs, propafenone or flecainide. Sotalol is for structural heart disease, bisoprolol is for rate control and adenosine is for supraventricular tachycardia.
- 8 A 75 years old patient with recurrent syncopal attacks and complete heart block, what is the emergency...
(c) Temporary pacemaker
 The emergency treatment of an unstable bradyarrhythmia is temporary pacing. A permanent pacemaker is the later definitive treatment, atropine is for patients with no temporary pacing, and DC shock is for tachyarrhythmias.
- 9 A patient with supraventricular tachycardia and acute pulmonary edema, which is the emergency treatment?
(d) DC shock
 Acute pulmonary edema means heart failure, so the patient is unstable and needs emergency DC shock. Carotid massage, adenosine and amiodarone are for stable patients.
- 10 A patient has rapid onset and offset of chest pain. Which of the following tests can the patient do out of...
(a) Holter monitoring
 Holter monitoring is short term ambulatory ECG recording, done while the patient goes about his day out of the clinic. Stress ECG, MRI and echo are done inside the hospital.
- 11 A 75 years old patient with recurrent syncopal attacks with sinus bradycardia, what is the emergency...
(a) IV atropine
 For symptomatic sinus bradycardia the first emergency step is IV atropine (0.5 to 1 mg), which blocks vagal tone on the sinus node. Temporary pacing is used if atropine fails, and it is the first emergency step in complete heart block, where atropine usually does not work. A permanent pacemaker is the later definitive treatment, and DC shock has no role.
- 12 A patient with stable supraventricular tachycardia, which is the first line treatment?
(c) Carotid massage
 In stable supraventricular tachycardia the first AV node blocking strategy listed is vagal maneuvers such as carotid massage, before IV adenosine and the other drugs.
- 13 * A 26 years old female patient presented with atrial flutter and anterior wall MI which of the following...
(a) Ivabradine
 This is inferred from the handout: ivabradine is used for inappropriate sinus tachycardia, and it is a funny Na channel blocker of the sinus node, so it does not control the rate in atrial flutter. Rivaroxaban is used for anticoagulation, and bisoprolol and non-DHP CCBs such as verapamil control the rate.
- 14 The following is not an indication of pacemaker implantation.
(d) Right bundle branch block
 Heart block can be associated with bradycardia or not; complete heart block, Mobitz type II and sinus node disease with chronotropic incompetence give a slow rate needing pacing. Isolated right bundle branch block does not slow the heart and needs no pacemaker.

- 15 * The following statement is False regarding supra ventricular arrhythmia:
(c) Pregnant women with cardiogenic shock and with...
 This is inferred from the handout: shock makes the patient unstable, and unstable arrhythmia is treated by rapid restoration of sinus rhythm with DC shock, so (c) is false. Anticoagulation is part of AF treatment, and its extension depends on the thromboembolic risk profile.
- 16 For a patient admitted with acute atrial fibrillation lasting for more than 48 hours and hemodynamically...
(a) Immediate intravenous amiodarone.
 After 48 hours, restoring sinus rhythm carries a high risk of thromboembolism, so rate control is chosen. Immediate IV amiodarone restores sinus rhythm and is avoided; rate control, parenteral anticoagulation and transesophageal echo are correct steps.
- 17 The following is the favorable intra-venous drug to rapidly control heart rate in patient with decompensated...
(a) IV digoxin
 In AF with structural heart disease, rate control is by IV digoxin, and oral digoxin is used if AF is associated with heart failure. IV metoprolol and verapamil are for patients with no structural heart disease.
- 18 The best emergency treatment of second-degree AV block Mobitz type II (6:1 block) is:
(b) Temporary pacemaker
 The emergency treatment of an unstable bradyarrhythmia is temporary pacing. Drugs such as atropine are usually temporary, for patients with no temporary pacing, and DC shock is for tachyarrhythmias.
- 19 Which of the following is the treatment of choice for hemodynamically unstable patients with atrial...
(a) Direct current (DC) cardioversion
 An unstable patient with AF needs DC cardioversion. Rate control and amiodarone are for stable patients, and a pacemaker treats bradyarrhythmia.
- 20 Which of the following is the definition of bradyarrhythmia?
(b) Heart rate less than 60 beats per minute
 Bradyarrhythmia is any heart rate of 60 beats per minute or less.
- 21 Which of the following is defined as a tachyarrhythmia?
(b) Heart rate >100 beats per minute
 Tachyarrhythmia is a heart rate of 100 beats per minute or more, with or without abnormal rhythm.
- 22 Which of the following is the treatment of choice for symptomatic bradycardia due to sinus node dysfunction?
(a) Atropine
 Atropine and atropine derivatives are the drugs listed for bradyarrhythmia, and atropine is the only rate-raising choice here. Beta blockers slow the heart more, and defibrillation and cardioversion are for tachyarrhythmias.
- 23 Which of the following can cause supraventricular arrhythmias:
(d) All of the above
 Ischemic heart disease and myocarditis are listed causes of tachyarrhythmia, and arrhythmias, especially AF, are complications of dilated and hypertrophic cardiomyopathy. So all three can cause supraventricular arrhythmias.
- 24 Urgent treatment in hemodynamically unstable patient with Atrial fibrillation is:
(d) DC- cardioversion
 An unstable patient with AF needs DC cardioversion to restore sinus rhythm. Beta blockers and digoxin only slow the rate in stable patients, and ACE inhibitors do not treat AF.
- 25 COVID-19 can cause the following arrhythmia:
(d) All of the above
 COVID-19 causes fever, myocarditis and electrolyte disturbance, which are physiologic, inflammatory and electrolyte causes of tachyarrhythmia. So it can cause sinus tachycardia, AF and ventricular tachycardia.
- 26 AV- block associating acute myocardial infarction:
(d) Usually patients with AV block and anterior wall MI will...
 AV block in MI is more serious in anterior wall MI and more frequent and benign in inferior wall MI, so (a), (b) and (c) are wrong. The serious block of anterior wall MI usually needs a pacemaker.

27 * Which of the following is not a cause of sinus bradycardia?

(b) Complete heart block

This is inferred from the handout: complete heart block is an AV nodal block, apart from sinus bradycardia, which is an SA nodal disorder. Myxoedema (endocrine), hypothermia and obstructive jaundice slow the sinus node itself.

28 Atrial fibrillation leads to:

(b) Irregular pulse with pulse deficit

AF makes the pulse irregular, and some weak early beats do not reach the wrist, so the apical rate is higher than the pulse rate (pulse deficit).

29 Cardiac arrest may be due to:

(c) Pulseless ventricular tachycardia

Ventricular tachycardia may cause sudden cardiac arrest, and pulseless ventricular tachycardia is an arrest rhythm. Ectopics, atrial flutter and Wenckebach block keep a cardiac output.

30 Drug of choice in acute management of PSVT is:

(d) Adenosine

After vagal maneuvers, IV adenosine, a short acting AV node blocking agent, is the first drug listed for stable supraventricular tachycardia.

31 Hyperthyroid heart disease is manifested by:

(d) Paroxysmal atrial fibrillation

Hyperthyroidism is an endocrine cause of tachyarrhythmia, and AF, often paroxysmal, is its typical arrhythmia. It raises the cardiac output (high COP state), so low output, prolonged circulation time and pericardial effusion do not fit.

32 Which of the following ECG findings is associated with sudden cardiac death?

(a) Prolonged QT interval

Long QT syndrome commonly leads to ventricular tachycardia, and ventricular tachycardia carries a risk of sudden cardiac death. First-degree AV block, RBBB and ventricular ectopics are usually benign.

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

1 Which of the following is the best emergency treatment of ventricular tachycardia.

(a) DC shock

Ventricular tachycardia is treated by restoring sinus rhythm with DC shock regardless of hemodynamic status. Verapamil and adenosine are for supraventricular tachycardia, and beta blockers are for long term prevention.

Model Answers | Essay Questions

1 Outline the treatment lines of supraventricular tachycardia.

Treatment

- ① **Emergency treatment** (unstable patients with severe hypotension or shock, heart failure, chest pain):
 - Circulatory support (IV line fluids and circulatory support).
 - Monitoring.
 - Rapid restoration of sinus rhythm, using electric cardioversion or defibrillation (DC shock).
- ② Removal of the cause, and treatment of the primary condition.
- ③ Life-style modification (avoiding certain foods and drinks, modifying physical activity, improving the quality of sleep, relieving stresses).
- ④ Pharmacological / interventional treatment: according to the type of arrhythmia.

Supraventricular tachycardia: acute treatment

- ① In hemodynamically unstable patients: **DC shock**.

- ② In stable patients: you aim to abort the tachycardia with one of the following AV node blocking strategies:
- Vagal maneuvers: carotid massage or induced vomiting.
 - IV adenosine: short acting AV node blocking agent.
 - IV non-dihydropyridine CCB (verapamil).
 - IV beta blockers.
 - IV digoxin.
 - IV amiodarone.

Supraventricular tachycardia: long term treatment

- ① Medications to prevent recurrence:
- Beta blockers.
 - Non-dihydropyridine CCB (verapamil).
 - Amiodarone.
- ② **EP study and catheter ablation** to cure the patient and stop the need for long term anti-arrhythmic drugs.

- 2 Discuss the treatment strategy of a patient with stable atrial fibrillation presenting after 48 hours. (Also asked as: Treatment lines of a patient with late onset (> 48 hours) uncontrolled atrial fibrillation with normal blood pressure.)

Treatment

- ① Removal of the cause, and treatment of the primary condition.
- ② Life-style modification (avoiding certain foods and drinks, modifying physical activity, improving the quality of sleep, relieving stresses).
- ③ Pharmacological / interventional treatment: according to the type of arrhythmia.

Atrial fibrillation: acute treatment

- ① **Anticoagulation:** IV loading of parenteral anticoagulation.
- ② If the onset of arrhythmia occurred **more than 48 hours**, the risk of thromboembolism will be high if you restored sinus rhythm unless you checked the patient with transesophageal echo. Therefore, **rate control** (slowing AV nodal conduction) will be appropriate:
- No structural heart disease: IV BB or non-dihydropyridine CCB.
 - Structural heart disease: IV digoxin.

Atrial fibrillation: long term treatment

- ① **Anticoagulation:**
- If valvular (mitral stenosis) or prosthetic valve: VKA anticoagulation (warfarin) after bridging therapy with parenteral anticoagulation till reaching INR (2-3).
 - If not: VKA or DOAC (rivaroxaban or apixaban) anticoagulation for one month. Extension of the anticoagulation treatment after one month will depend on the thromboembolic risk profile of the patient.
- ② Maintenance of sinus rhythm for patients who underwent rhythm control strategy:
- No structural heart disease: class IC (propafenone, flecainide).
 - Structural heart disease: class III (amiodarone, sotalol).
 - EP study and catheter or surgical ablation (maze procedure): curative.
- ③ Maintenance of rate control for patients who underwent rate control strategy:
- Selective beta blockers: oral bisoprolol, metoprolol, nebivolol.
 - If BB contraindicated: oral non-dihydropyridine CCB (diltiazem).
 - If associated with heart failure: oral digoxin.

After **one month** of adequate anticoagulation and rate control for patients with AF presented more than 48 hours, restoring sinus rhythm can be safely done as in rhythm control:

- * If hemodynamically unstable: DC cardioversion.
- * If stable: pharmacological cardioversion by antiarrhythmic drugs:
 - If no structural heart disease: class IC (propafenone, flecainide).
 - If structural heart disease: class III (amiodarone).

3 Enumerate lines of management of ventricular tachycardia.

Treatment

- ① **Emergency treatment** (unstable patients with severe hypotension or shock, heart failure, chest pain):
 - Circulatory support (IV line fluids and circulatory support).
 - Monitoring.
 - Rapid restoration of sinus rhythm, using electric cardioversion or defibrillation (DC shock).
- ② Removal of the cause, and treatment of the primary condition.
- ③ Life-style modification (avoiding certain foods and drinks, modifying physical activity, improving the quality of sleep, relieving stresses).
- ④ Pharmacological / interventional treatment: according to the type of arrhythmia.

Ventricular tachycardia

- ① **Acute management:** restoring sinus rhythm with DC shock regardless of hemodynamic status.
- ② **Long term management** to prevent recurrence:
 - Beta blockers or class III antiarrhythmic drugs.
 - EP study and catheter ablation.
- ③ **Device (ICD) implantation** could be considered to prevent risk of sudden cardiac death.

4 List the intravenous drugs that could be used for acute termination of supraventricular tachycardia in a patient who has dilated cardiomyopathy and reduced ejection fraction.

In stable patients with supraventricular tachycardia, you aim to abort the tachycardia with one of the following AV node blocking strategies:

- * IV adenosine: short acting AV node blocking agent.
- * IV non-dihydropyridine CCB (verapamil).
- * IV beta blockers.
- * IV digoxin.
- * IV amiodarone.

Note The handout does not say which of these drugs to choose when the ejection fraction is reduced, answer incomplete.

Final Exam on the Whole Course

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

The answer key and the explanation of every question of the final exam, from the handout.

Answer Key | Final Exam

Question no.	1	2	3	4	5	6	7	8	9	10	11	12
Answer	c	d	a	c	c	b	c	a	d	c	a	c

Question no.	13	14	15	16	17	18	19	20	21	22	23	24
Answer	a	b	a	d	b	c	b	b	b	a	c	a

Question no.	25	26	27	28	29	30	31	32	33	34	35	36
Answer	c	c	c	d	b	d	c	a	b	d	d	c

Question no.	37	38	39	40	41	42	43	44	45	46	47	48
Answer	d	a	a	c	c	b	d	c	d	c	b	c

Question no.	49	50
Answer	b	a

► Why this answer?

1 Which of the following favours cardiac asthma rather than bronchial asthma?

(c) Morphine improves the condition Lesson 1

Morphine improves cardiac asthma but is contraindicated in bronchial asthma. Cardiac asthma has inspiratory dyspnea, the patient is never free between attacks, and adrenaline is contraindicated.

2 Which of the following favours peripheral rather than central cyanosis?

(d) Cyanosis improves with warming Lesson 1

Warming improves peripheral cyanosis but has no effect on central cyanosis. Clubbing, secondary polycythaemia and low arterial PO₂ belong to central cyanosis.

3 Which of the following is not a manifestation of heart failure in children?

(a) Large volume pulse Lesson 2

In heart failure the pulse is weak because of the low COP, so a large volume pulse is not a feature. Tachycardia, oliguria and an enlarged tender liver are all features of H.F..

- 4 Causes of acute right sided heart failure are all of the following except:
(c) Malignant hypertension Lesson 2
 Malignant hypertension is systemic HPN, a pressure overload on the left ventricle. Pulm. HPN, tricuspid incompetence and right ventricular myocardial disease (the same like Lt. v., e.g. infarction) are causes of right ventricular failure.
- 5 Systemic congestive manifestations (right-sided heart failure) do not include:
(c) Bilateral basal crepitations Lesson 2
 Bilateral basal crepitations are a backwards sign of left-sided H.F., due to pulmonary congestion. Oedema of the lower limbs, ascites and pain in the Rt. hypochondrium are systemic congestion features of right-sided H.F..
- 6 Which side effect is reported with thiazides but not with loop diuretics?
(b) Hypercalcaemia Lesson 2
 Hypercalcaemia is listed only with thiazides. Hypokalaemia, hyperglycaemia and hyperuricemia are side effects of both loop diuretics and thiazides.
- 7 Dopamine infusion at a dose of 2-4 µg/kg/min acts mainly on:
(c) Dopaminergic receptors to increase renal blood flow Lesson 2
 At 2-4 µg/kg/min dopamine acts on dopaminergic receptors to increase renal blood flow. B1 receptors are reached at 5-10 µg/kg/min and alpha receptors at 10 µg/kg/min, while Na-K ATPase inhibition is the action of digitalis.
- 8 Which of the following is an absolute contraindication to digitalis?
(a) Ventricular tachycardia Lesson 2
 Ventricular tachycardia is a contraindication to digitalis for fear of changing it to V.F.. AF, atrial flutter and P.S.V.T. are indications of digitalis.
- 9 What is the recommended duration for secondary prophylaxis after rheumatic fever?
(d) At least until 40 years of age / for life in patients... Lesson 3
 Secondary prevention lasts till age 40 years or lifelong in patients with severe valvular disease or after valvular replacement. Without carditis it is till age 21 (at least 5 years) and with mild regurgitant lesion till age 25 (at least 10 years), so a, b and c mix up the groups.
- 10 What is the most commonly affected valve in rheumatic heart disease?
(c) Mitral valve Lesson 3
 Mitral valve is commonly involved, followed by aortic valve then tricuspid valve.
- 11 Which of the following is a minor criterion of acute rheumatic fever?
(a) Fever Lesson 3
 Fever is a minor criterion. Erythema marginatum and carditis are major criteria, and a raised ASO titre is evidence of recent streptococcal infection.
- 12 Which of the following is false regarding rheumatic chorea in children?
(c) Associated with hypertonia and hyperreflexia Lesson 3
 Sydenham's chorea gives hypotonia and hyporeflexia, not hypertonia and hyperreflexia. It is more common in girls over 8 years, gives emotional instability and interrupted speech, and could occur alone weeks or months after the streptococcal infection.
- 13 In the treatment of acute rheumatic fever, which of the following is correctly matched?
(a) Carditis: corticosteroids Lesson 3
 Carditis is treated with corticosteroids. Arthritis is treated with salicylates, chorea with haloperidol, and penicillin allergic patients take daily erythromycin.
- 14 In a recurrent attack of rheumatic fever, the diagnosis can be made by:
(b) 3 minor criteria + evidence of recent streptococcal... Lesson 3
 In recurrent attacks, 3 minor criteria are enough, but evidence of recent streptococcal infection is still required. One major criterion needs 2 minor criteria.

- 15 * The most common congenital cyanotic heart disease in children is:
(a) Fallot tetralogy *Lesson 4*
 The handout calls TOF one of the most common CHDs, and it is one of the two cyanotic CHDs it studies, with reduced pulmonary blood flow. The answer is inferred from these lines; the handout does not rank the cyanotic lesions.
- 16 Which of the following is not used in treatment of cyanotic spells of Fallot's tetralogy?
(d) Hydrocortisone *Lesson 4*
 For cyanotic spells the handout gives the knee-chest position, oxygen, IV morphine and IV propranolol; hydrocortisone is not among them. Sodium bicarbonate is used to correct the acidosis of a severe spell.
- 17 In MOST patients with an ASD, the characteristic physical finding is:
(b) A wide and fixed splitting of 2nd heart sound *Lesson 4*
 The RV impulse is also increased in ASD, but wide and fixed splitting of S2 is the most characteristic sign of ASD. There is no audible murmur because of flow across the ASD, so a pansystolic murmur is not expected.
- 18 Regarding Ebstein's anomaly, which of the following is true?
(c) The most common associated defect is secundum ASD or PFO *Lesson 4*
 The most common associated defect is a secundum ASD or PFO, in over 50% of patients. The displaced leaflet is the septal leaflet of the tricuspid valve, the atrialized RV works as RA, and the ECG shows huge P waves.
- 19 Regarding closure of atrial septal defect (ASD), which of the following is true?
(b) RV volume overload with Qp:Qs > 1.5 is the main indication... *Lesson 4*
 RV volume overload on echo with Qp:Qs above 1.5 is the main indication for ASD closure, even without symptoms. Ostium primum and sinus venosus defects are not amenable to transcatheter closure.
- 20 Regarding coarctation of the aorta in adolescents and adults, which of the following is true?
(b) The constriction typically lies just distal to the left... *Lesson 4*
 Coarctation typically lies just distal to the left subclavian at the site of the ductal attachment. A bicuspid aortic valve is the most common coexisting anomaly, true claudication is rare, and surgery is more hazardous in adults than in children.
- 21 The following investigations are used in diagnosis of valvular heart lesions except:
(b) CT chest *Lesson 5*
 Trans-thoracic and trans-esophageal echo and coronary angiography before open heart surgery are the investigations used. CT chest is not one of them; only a CT aortogram is used, in patients with aortic aneurysm.
- 22 In chronic aortic regurgitation, Quincke's sign is:
(a) Capillary pulsation at the root of the nail when pressure... *Lesson 5*
 Quincke's sign is capillary pulsation seen at the root of the nail when pressure is applied to the tip. Head nodding is de Musset sign, higher lower limb pressure is Hill's sign and pistol-shot sounds are Traube's sign.
- 23 A man with infective endocarditis develops acute severe aortic regurgitation with acute pulmonary oedema. He...
(c) Beta-blockers *Lesson 5*
 In acute AR, beta-blockers are avoided as they reduce the cardiac output and the slower rate allows more time for diastolic filling of the LV. Oxygen, diuretics (if no hypotension) and inotropes such as dobutamine are used.
- 24 Regarding mitral stenosis, which of the following is true?
(a) Oral anticoagulation is indicated if the left atrium is... *Lesson 5*
 A dilated left atrium > 50 mm is an indication for oral anticoagulation in mitral stenosis. The MS murmur increases on lying on the left lateral position, AF is a common finding, and surgery is for anatomy not suitable for balloon valvotomy.
- 25 After assessment of cardiovascular risk in an asymptomatic patient with coronary heart disease, if the...
(c) Referral for invasive testing. *Lesson 6*
 Invasive coronary angiography is recommended in patients with positive stress test, while CT coronary angiography is a good negative test in patients less likely to have coronary artery disease. So a high-risk patient goes on to invasive testing; retesting later and non-invasive testing suit lower risk, and folic acid has no role.

- 26 Which of the following is NOT one of the criteria of typical chest pain?
(c) Pain provocation by deep inspiration Lesson 6
 Typical angina is constricting chest discomfort, brought on by exertion or emotion, and relieved within minutes by rest or nitroglycerin. Pain made worse by deep breathing is pleuritic pain and points to a non-ischemic cause. A duration of about 15 minutes is still consistent with angina, which is short-lived (minutes, not seconds or hours), so it does not exclude typical pain.
- 27 Nitrates are anti-anginal drugs. Which of the following is a common side effect?
(c) Headache. Lesson 6
 Headache is a common side effect of nitrates that could be managed with paracetamol. Nitrates are vasodilators, so they lower rather than raise blood pressure.
- 28 Regarding third-line anti-anginal drugs, which of the following is true?
(d) Ivabradine may cause visual disturbance (phosphenes) Lesson 6
 Ivabradine may cause visual disturbance (phosphenes) due to retinal side effects. Nicorandil must not be combined with nitrates, ranolazine prolongs QTc, and trimetazidine changes the heart fuel from fatty acids to glucose.
- 29 A patient with NSTEMI has elevated troponin and dynamic ST segment changes. He is haemodynamically stable,...
(b) Within less than 24 hours Lesson 6
 Elevated troponin and dynamic ST or T changes make the patient high risk, so PCI is done within less than 24 hours. The 2 hour window is for hemodynamic or electrical instability or refractory pain, and fibrinolytic therapy is only for STEMI.
- 30 In cardiovascular disease, reduction in complications or disability with rehabilitation is called:
(d) Tertiary prevention Lesson 6
 Tertiary prevention is reduction in complications or disability, rehabilitation, or restoration of function. Primordial prevents the risk, primary reduces incidence, and secondary reduces prevalence or consequence of disease.
- 31 Which of the following is the least cardiac lesion that can develop infective endocarditis?
(c) ASD Lesson 7
 Infective endocarditis is rare in ASD due to the low pressure gradient between the two atria. VSD and PDA are among the congenital lesions where it is common, and pulmonary stenosis is not named as a rare site.
- 32 The causative organism of infective endocarditis when the route of entry is dental procedures is:
(a) Streptococcus viridans Lesson 7
 After dental procedures and oral or upper respiratory surgery the organism is usually Strept. viridans. Staph. aureus follows cardiac surgery and catheterization, and Strept. faecalis follows gastrointestinal and genitourinary procedures.
- 33 Which of the following laboratory findings may be found in infective endocarditis?
(b) False positive rheumatoid factor Lesson 7
 Immunological tests in infective endocarditis show increased gamma globulins and a false positive rheumatoid factor. The ESR is increased but not as high as in rheumatic fever, and blood culture is positive in 80% of cases.
- 34 All are causes of hemorrhagic pericardial effusion except:
(d) Heart failure Lesson 8
 Haemorrhagic pericarditis occurs in malignant infiltration, uraemia, myocardial infarction and T.B.. Right-sided heart failure causes hydropericardium, an accumulation of transudate.
- 35 All are signs of pericardial tamponade except:
(d) Pulmonary crepitations Lesson 8
 Signs of tamponade include signs of low COP, pulsus paradoxus, congested neck veins and weak distant heart sounds. Pulmonary crepitations are not among them; the chest x-ray shows pulmonary oligoemia.

- 36** Which of the following favours constrictive pericarditis rather than pericardial effusion?
(c) Early diastolic dip followed by plateau (square root sign)... *Lesson 8*
 An early diastolic dip followed by plateau (square root sign) on ventricular pressure tracing is a finding of constrictive pericarditis. A flask-shaped shadow that changes from day to day and Ewart's sign belong to pericardial effusion.
- 37** A pericardial aspirate shows proteins > 3 gm%, LDH > 200 IU/L and white cells > 1000/cmm. Which of the...
(d) Tuberculous pericarditis *Lesson 8*
 Proteins > 3 gm%, LDH > 200 IU/L and cells > 1000/cmm define an exudate, and T.B. is the most common cause of exudative effusion. Right sided heart failure, liver cell failure and nephrotic syndrome give a transudate.
- 38** In hypertrophic obstructive cardiomyopathy (HOCM), the midsystolic murmur along the left sternal border:
(a) Increases on standing *Lesson 9*
 The HOCM midsystolic murmur increases on standing and decreases with squatting or leg raising, because more venous return fills the ventricle and decreases the obstruction.
- 39** From the following scenarios, who is NOT suspected to have secondary hypertension?
(a) A 33-year-old obese woman presenting with BP 160/90 and a... *Lesson 10*
 Heredity and obesity are predisposing factors of essential HPN, so the young obese woman with a family history fits essential HPN. Sudden development, HPN resistant to treatment and onset after 50 years are clues to renal artery stenosis, a secondary cause.
- 40** Which of the following statements regarding the treatment of hypertensive emergencies and urgencies is FALSE?
(c) All patients with marked blood pressure elevation should... *Lesson 10*
 Only patients with acute target organ damage (emergencies) need hospitalization, preferably in ICU. Urgencies have no acute end organ damage and blood pressure is lowered over 1-2 days.
- 41** Regarding severe elevation of blood pressure, which is incorrect?
(c) Needs only reassurance of the patient and relatives. *Lesson 10*
 Severe elevation of blood pressure always needs treatment, never reassurance alone. Without acute end organ damage it is an urgency, lowered over 1-2 days; with acute Lt.v. failure and pulmonary oedema it is an emergency needing hospital and rapid reduction.
- 42** All of the following antihypertensive drugs are safe in pregnancy except:
(b) ACEI *Lesson 10*
 ACE inhibitors are on the 'Avoid' list of HPN with pregnancy. Alpha methyl dopa and CCBs (not at labour) are on the 'Use' list, and labetalol is a safe combined alpha and beta blocker.
- 43** Grade IV hypertensive retinopathy is defined by:
(d) Papilloedema plus grade III changes *Lesson 10*
 Grade IV hypertensive retinopathy is papilloedema plus grade III changes. Narrowing is grade I, silver wiring with kinking of veins is grade II, and flame shaped hge with cotton exudate is grade III.
- 44** Which antihypertensive drug is correctly matched with its side effect?
(c) Clonidine: rebound hypertension *Lesson 10*
 Clonidine causes postural hypotension and rebound hypertension. Hydralazine causes tachycardia, minoxidil causes hirsutism, and reserpine causes depression.
- 45** What is the most probable group of pulmonary hypertension in a patient with history of recurrent pulmonary...
(d) Group 4 *Lesson 11*
 Recurrent pulm. emboli cause obstructive (obliterative) pulmonary hypertension by obstruction of the pulm. arterioles. Chronic thromboembolic pulmonary hypertension is WHO group 4.
- 46** In pulmonary hypertension, the Graham Steell murmur is:
(c) Early diastolic murmur due to functional pulmonary... *Lesson 11*
 The Graham Steell murmur is an early diastolic murmur due to functional PR in pulmonary hypertension. The ejection systolic murmur of relative PS and functional TR are separate findings, and the machinery murmur belongs to PDA.

47 Which of the following is considered an indirect marker of DVT?

(b) D-dimer *Lesson 12*

D-dimer is raised in thromboembolism but is not specific; it is a breakdown product of fibrin, so it points to a clot indirectly.

48 In suspected cases of deep vein thrombosis, which of the following is the most appropriate investigation?

(c) Duplex ultrasound scan *Lesson 12*

Colored Doppler and venography are both listed for diagnosis of DVT; duplex ultrasound is preferred because it is accurate and non-invasive. D-dimer is not specific, and CT pulmonary angiography looks for a lung embolus.

49 A patient with massive pulmonary embolism presents with shock. Which of the following is the treatment of...

(b) Thrombolytic therapy *Lesson 12*

A hemodynamically unstable patient, with shock or heart failure, is treated with thrombolytic therapy. Embolectomy is done when thrombolysis is contraindicated, IVC interruption is for stable patients who cannot take anticoagulants, and morphine is avoided.

50 A patient with atrial fibrillation and rheumatic mitral stenosis needs long-term anticoagulation. Which drug...

(a) Warfarin with INR 2-3 *Lesson 13*

AF with mitral stenosis or a prosthetic valve needs VKA (warfarin) after bridging with parenteral anticoagulation till INR 2-3. Rivaroxaban and apixaban are used only when there is no mitral stenosis or prosthetic valve.