UPSC CMS PYQ MEDICINE (2023)

Medsynapse by Dr. Nikita

Which one of the following diseases affects predominantly large arteries?

(a) Granulomatosis with polyangiitis

(b) Polyarteritis nodosa

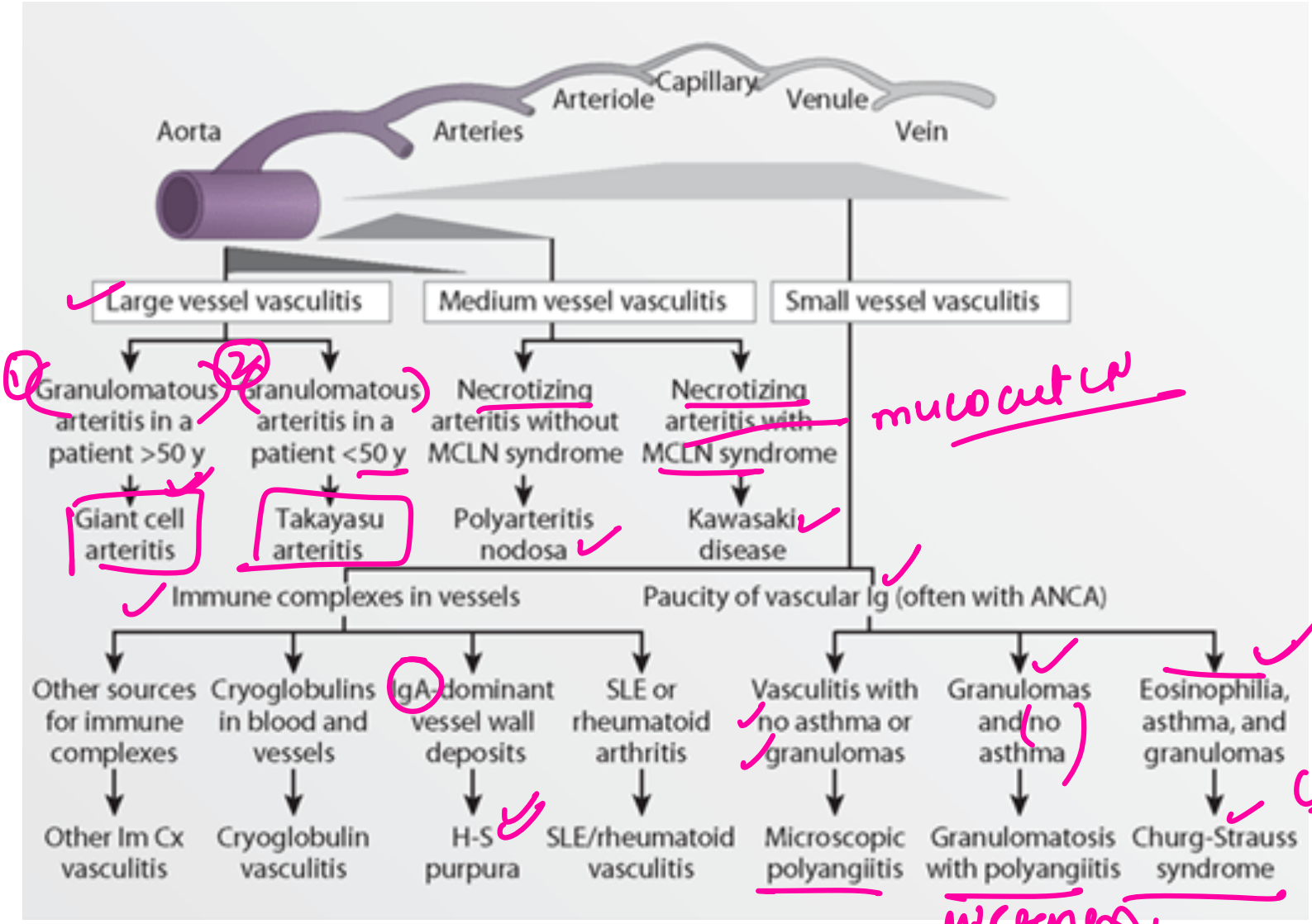
(c) Giant cell arteritis

(d) Eosinophilic granulomatosis with polyangiitis

large – GT – Takayasu → pulseless

CLASSIFICATION (Vessel Size)

- **Large vessel vasculitis**
  - Giant cell arteritis
  - Takayasu's arteritis
- **Medium-sized vessel vasculitis**
  - Polyarteritis nodosa
  - Kawasaki's disease
  - Primary granulomatous CNS vasculitis
- **Small vessel vasculitis**
  - ANCA associated vasculitis
  - Immune complex small vessel vasculitis
  - Paraneoplastic small vessel vasculitis
  - Inflammatory bowel disease vasculitis


Aorta
Arteries
Arteriole
Capillary
Venule
Vein
Large vessel vasculitis
Medium vessel vasculitis
Small vessel vasculitis
Granulomatous arteritis in a patient >50 y
Granulomatous arteritis in a patient <50 y
Necrotizing arteritis without MCLN syndrome
Necrotizing arteritis with MCLN syndrome
Giant cell arteritis
Takayasu arteritis
Polyarteritis nodosa
Kawasaki disease
Immune complexes in vessels
Paucity of vascular Ig (often with ANCA)
Other sources for immune complexes
Cryoglobulins in blood and vessels
IgA-dominant vessel wall deposits
SLE or rheumatoid arthritis
Vasculitis with no asthma or granulomas
Granulomas and no asthma
Eosinophilia, asthma, and granulomas
Other Im Cx vasculitis
Cryoglobulin vasculitis
H-S purpura
SLE/rheumatoid vasculitis
Microscopic polyangiitis
Granulomatosis with polyangiitis
Churg-Strauss syndrome

GnRH deficiency with hyposmia is typically seen in

(a) Kallmann syndrome → Hypogonadotrophic hypogonadism

(b) Bardet-Biedl syndrome → Obese, Retinitis pigmentosa.

(c) Prader-Willi syndrome →

(d) Wallenberg syndrome

Iodine has complex effects on thyroid function. Very high concentrations of iodine inhibit thyroid hormone synthesis and release. This effect is known as

(a) Wolff-Chaikoff effect

(b) Jod-Basedow effect

(c) reverse Wolff-Chaikoff effect

(d) reverse Jod-Basedow effect

Low serum thyroglobulin levels are seen in

Thyroid gland

(a) thyrotoxicosis factitia → exogenous NOT from thyroid gland

(b) subacute thyroiditis

(c) Graves' disease

(d) toxic multinodular goitre

Serum Tg levels are increased in all types of thyrotoxicosis except *thyrotoxicosis factitia* caused by self-administration of thyroid hormone. Tg levels are particularly increased in thyroiditis, reflecting thyroid tissue destruction and release of Tg. The main role for Tg measurement, however, is in the follow-up of thyroid cancer patients. After total thyroidectomy and radioablation, Tg levels should be undetectable; in the absence of anti-Tg antibodies, measurable levels indicate incomplete ablation or recurrent cancer.

Consider the following pharmacological agents :

1. Propranolol
2. Sodium ipodate
3. Propylthiouracil → antithyroid
4. Liothyronine → Lio → T3

Which of the above can be used for the treatment of thyrotoxic crisis?

(a) 3 only
(b) 1 and 2 only
(c) 1, 2 and 3
(d) 2, 3 and 4

↓ thyroid hormone.

active → T3

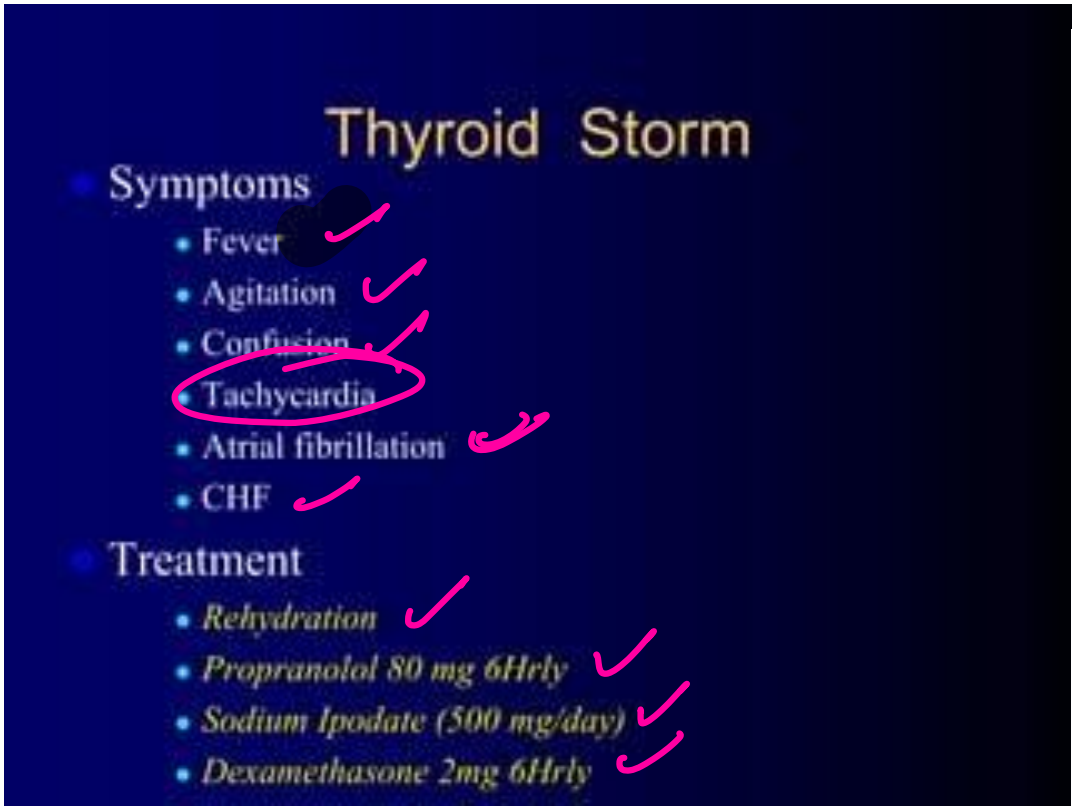
T4 ⟶ T3 active

↓ ⊖

. propranolol
. ipodate
.

Thyroid Storm

- Antithyroidal drugs
  - Propylthiouracil
    - Loading dose of 600 mg PO/NG/PR once
    - Maintenance dose of 200-300 mg every 6 hours PO/NG/PR
  - Methimazole
    - Loading dose of 20 mg PO every 6 hours
- Saturated solution of potassium iodide
  - 5 drops PO every 6 hours; must be started after antithyroid drug therapy is initiated to avoid potential worsening of hyperthyroidism
- Glucocorticoids
  - Hydrocortisone 100 mg IV every 8 hours
- Beta-blocker therapy
  - Propranolol 40-80 mg PO every 4 hours or 2 mg IV every 4 hours
- Supportive measures
  - IV fluids



Thyroid storm/ Thyrotoxic crisis

Management:

- Adequate hydration with I. V. fluid.
- oral Propranolol 80mg QDS / I.V. 1-4mg QDS.
- Injection Hydrocortisone 100mg TDS
- Oral Sodium Ipodate 500mg daily for 48 to72 hours (or KI/ Lugol's solution)

Consider the following pharmacological agents :

1. Liothyronine – Lio T3 ✓
2. Levothyroxine T4 ✓
3. Carbimazole ✗
4. Sodium ipodate ✗

Which of the above may be included in the treatment of myxoedema coma?

↳ Hypothyroidism

(a) 1 only
(b) 1 and 2
(c) 2 and 3
(d) 3 and 4

Table 4. Initiation of Treatment in ED for Myxedema Coma

1. 200-300 mcg (4 mcg/kg) IV bolus thyroxine, followed by 50-100 mcg/day[31]
2. T_3 20 mcg IV bolus (loading dose 10-25 mcg), then 10 mcg every 8-12 hours for 24-48 hours until the patient is conscious and taking maintenance T_4[12]
3. Hydrocortisone 100 mg every 8 hours
4. Broad spectrum antibiotics pending culture results recommended if there is evidence of infection[29,47]
5. Supportive care for underlying illness
6. Consider elective intubation for myxedema coma
7. Consider hypertonic saline for severe hyponatremia
8. Consider appropriate rewarming techniques

∵ Hypothermia (+)

Which one of the following insulin preparations has the longest effective duration of action?

(a) Detemir
(b) Glargine
(c) NPH
(d) Degludec

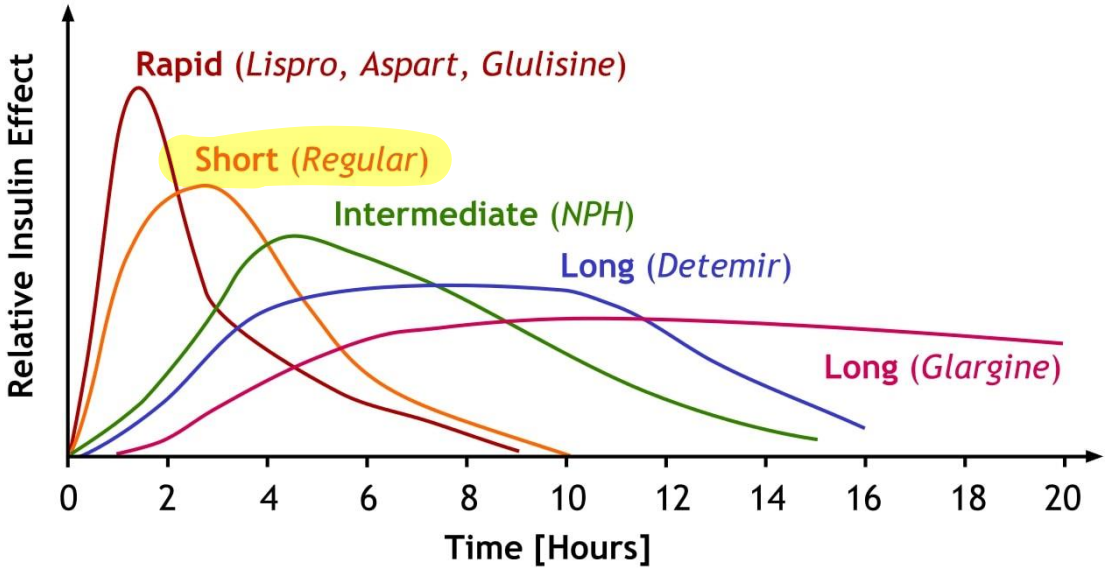
long acting

rap'd → No LAG // glulysine

glargine
large

for s.c. administration

Insulin type	Onset of action (h)	Peak of action (h)	Duration of action (h)
Ultra-rapid acting analog (faster aspart)[a,c]	0.1-0.2	1-3	3-5
Rapid-acting analogs (aspart, glulisine, and lispro)	0.15-0.35	1-3	3-5
Regular/soluble (short acting)	0.5-1	2-4	5-8
NPH*	2-4	4-12	12-24[a]
Basal long-acting analogs			
Glargine[b]	2-4	8-12	22-24[a]
Detemir	1-2	4-7	20-24[a]
Glargine U300*+*	2-6	Minimal peak	30-36
Degludec[c]	0.5-1.5	Minimal peak	>42



Functioning pituitary adenoma most commonly arises from which one of the following cells?

(a) Somatotroph

(b) Lactotroph – prolactin → acidophil.
↳ Tyrosine kinase

(c) Corticotroph

(d) Thyrotroph

prolactinoma
m/c

FLAT
base

A 45-year-old male presented with polyuria, polydipsia and polyphagia for the last 3 months. His fasting plasma glucose is 106 mg/dL and random blood glucose is 220 mg/dL. Which one of the following statements is correct regarding his diagnosis?

(a) He has impaired glucose tolerance (IGT).

(b) He has definitive diagnosis of diabetes mellitus.

(c) He does not have diabetes mellitus.

(d) Diagnosis of diabetes mellitus is indeterminate as HbA1c value is not provided.

x > 126

>200 + symptoms

HbA1c > 6.5

Parameter	Diagnosis	
	Prediabetes	Diabetes Mellitus
FPG [a]	IFG: 100–125 mg/dL (5.6–6.9 mmol/L) AND/OR	≥126 mg/dL (7.0 mmol/L). OR
2-h PG [b]	IGT: 140–199 mg/dL (7.8–11.0 mmol/L) AND/OR	≥200 mg/dL (11.1 mmol/L) during OGTT. OR
HbA1c [c]	5.7–6.4% (39–47 mmol/mol) or ≥10% increase in HbA1C	≥6.5% (48 mmol/mol). OR
RPG	NA	≥200 mg/dL (11.1 mmol/L) plus symptoms (polyuria, polyuria, polydipsia, weight loss, fatigue)

Which one of the following statements is true regarding the use of SGLT2 inhibitor in management of diabetes mellitus?

(a) They are used in type-1, type-2 and pancreatogenic forms of diabetes mellitus.

(b) They cause increase in blood pressure by 3–6 mm Hg and hence avoided in hypertensive patients.

(c) Their glucose-lowering effect is dependent on insulin secretion and insulin sensitivity.

(d) Euglycemic diabetic ketoacidosis may occur during treatment if there is a concurrent illness.

→ Kidneys
MOA → ⊖ glucose reab PCT
↑ urine glucose.

→ ↓BP ∴ ⊖ Na reab.

SGLT2⊖ → ↑ glucagon
↓
⊕ ketone production.

Sodium-Glucose Co-Transporter 2 (SGLT2) Inhibitors

These agents (Table 397-5) lower the blood glucose by selectively inhibiting this co-transporter, which is expressed almost exclusively in the proximal, convoluted tubule in the kidney. This inhibits glucose reabsorption, lowers the renal threshold for glucose, and leads to increased urinary glucose excretion. Thus, the glucose-lowering effect is insulin independent and not related to changes in insulin sensitivity or secretion. The loss of urinary glucose may promote modest weight reduction. Since these agents also impair proximal reabsorption of sodium, their use is associated with a diuretic effect and 3–6 mm Hg reduction in systolic blood pressure. Due to the increased urinary glucose, urinary and genital mycotic infections are more common in both men and women, and the diuretic effect can lead to reduced intravascular volume and acutely impaired kidney function. Inhibition of SGLT2 on the alpha cell may lead to increased glucagon and consequently liver production of glucose and ketones. Euglycemic DKA may occur during illness or when ongoing glucosuria masks stress-induced requirements for insulin. These agents should not be prescribed for patients with type 1 DM or pancreatogenic forms of DM associated with insulin deficiency. Empagliflozin and canagliflozin reduces CVD events and all cause cardiovascular mortality in patients with type 2 DM and established CVD, the risk for nephropathy, and the rate of hospitalization for CHF. A possible increased risk of bladder cancer has been seen with dapagliflozin; canagliflozin is associated with an increased risk of leg and foot amputation and bone fractures.

Which of the following antidiabetic drugs may cause weight gain?

1. Glimepiride → SU → ↑insulin → ↑insulin anabolic
2. Pioglitazone → fluid retention → KCB
3. NPH insulin
4. Sitagliptin ×

5. tide → wt loss

Select the correct answer using the code given below.

(a) 1, 2 and 3
(b) 1, 2 and 4
(c) 1, 3 and 4
(d) 2, 3 and 4

TABLE 397-5 Agents Used for Treatment of Type 1 or Type 2 Diabetes

	MECHANISM OF ACTION	EXAMPLES[a]	HBA_{1c} REDUCTION (%)[b]	AGENT-SPECIFIC ADVANTAGES	AGENT-SPECIFIC DISADVANTAGES	CONTRAINDICATIONS
Oral						
Biguanides[c*]	↓ Hepatic glucose production	Metformin	1–2	Weight neutral, do not cause hypoglycemia, inexpensive, extensive experience, ↓ CV events	Diarrhea, nausea, lactic acidosis, vitamin B12 deficiency	Renal insufficiency (see text for GFR <45 mL/min), CHF, radiographic contrast studies, hospitalized patients, acidosis
α-Glucosidase inhibitors[c**]	↓ GI glucose absorption	Acarbose, miglitol, voglibose	0.5–0.8	Reduce postprandial glycemia	GI flatulence, liver function tests	Renal/liver disease
Dipeptidyl peptidase IV inhibitors[c***]	Prolong endogenous GLP-1 action; ↑ Insulin, ↓ glucagon	Alogliptin, linagliptin, saxagliptin, sitagliptin, vildagliptin	0.5–0.8	Well tolerated, do not cause hypoglycemia	Angioedema/ urticarial and immune-mediated dermatologic effects	Reduced dose with renal disease
Insulin secretagogues: Sulfonylureas[c*]	↑ Insulin secretion	Glibornuride, gliclazide, glimepiride, glipizide, gliquidone, glyburide, glyclopyramide	1–2	Short onset of action, lower postprandial glucose, inexpensive	Hypoglycemia, weight gain	Renal/liver disease
Insulin secretagogues: Nonsulfonylureas[c****]	↑ Insulin secretion	Mitiglinide nateglinide, repaglinide	0.5–1.0	Short onset of action, lower postprandial glucose	Hypoglycemia	Renal/liver disease
Sodium-glucose cotransporter 2 inhibitors[***]	↑ renal glucose excretion	Canagliflozin, dapagliflozin, empagliflozin, ertugliflozin	0.5–1.0	do not cause hypoglycemia, ↓ weight and BP; see text for CVD effect	Urinary and genital infections, polyuria, dehydration, exacerbate tendency to hyperkalemia and DKA; see text	Moderate renal insufficiency, insulin-deficient DM
Thiazolidinediones[c****]	↓ Insulin resistance, ↑ glucose utilization	Pioglitazone, rosiglitazone	0.5–1.4	Lower insulin requirements	Peripheral edema, CHF, weight gain, fractures, macular edema	CHF, liver disease

Parenteral						
Amylin agonists[c,d***]	Slow gastric emptying, ↓ glucagon	Pramlintide	0.25–0.5	Reduce postprandial glycemia, weight loss	Injection, nausea, ↑ risk of hypoglycemia with insulin	Agents that also slow GI motility
GLP-1 receptor agonists[c***]	↑ Insulin, ↓ glucagon, slow gastric emptying, satiety	Albiglutide, dulaglutide, exenatide, liraglutide, lixisenatide, semaglutide	0.5–1.0	Weight loss, do not cause hypoglycemia; see text for CVD effect	Injection, nausea, ↑ risk of hypoglycemia with insulin secretagogues	Renal disease, agents that also slow GI motility; medullary carcinoma of thyroid, pancreatic disease
Insulin[c,d****]	↑ Glucose utilization, ↓ hepatic glucose production, and other anabolic actions	See text and Table 397-4	Not limited	Known safety profile	Injection, weight gain, hypoglycemia	
Medical nutrition therapy and physical activity[c*]	↓ Insulin resistance, ↑ insulin secretion	Low-calorie, low-fat diet, exercise	1–3	Other health benefits	Compliance difficult, long-term success low	

A tall thin adolescent boy is found to have gynaecomastia and small testes. His blood testosterone level is undetectable, and serum FSH and LH levels are elevated. Which one of the following is the likely karyotype pattern?

(a) 46 XY

(b) 45 XO → Turner short stature

(c) 47 XXY

(d) 21 Trisomy

Klinefelter

↓

Testis failed

↑FSH LH

Focal seizures may be associated with which of the following?

1. Jacksonian march
2. Todd's paralysis
3. Epilepsia partialis continua

Select the correct answer using the code given below.

(a) 1 and 2 only

(b) 2 and 3 only

(c) 1 and 3 only

(d) 1, 2 and 3

Three additional features of focal motor seizures are worth noting. First, in some patients, the abnormal motor movements may begin in a very restricted region such as the fingers and gradually progress (over seconds to minutes) to include a larger portion of the extremity. This phenomenon, described by Hughlings Jackson and known as a "Jacksonian march," represents the spread of seizure activity over a progressively larger region of motor cortex. Second, patients may experience a localized paresis (Todd's paralysis) for minutes to many hours in the involved region following the seizure. Third, in rare instances, the seizure may continue for hours or days. This condition, termed *epilepsia partialis continua*, is often refractory to medical therapy.

Focal seizures may also manifest as changes in somatic sensation (e.g., paresthesias), vision (flashing lights or formed hallucinations), equilibrium (sensation of falling or vertigo), or autonomic function

Which of the following are contra-indications to thrombolysis in acute ischemic stroke?

1. Recent head injury
2. Recent MI
3. Hypertension > 150/100 mm Hg
4. GI bleeding in last 3 weeks

Select the correct answer using the code given below.

(a) 1, 2 and 3
(b) 1, 2 and 4
(c) 1, 3 and 4
(d) 2, 3 and 4

risk → Hx

xn > 180/110

in 4.5 hr

- First Ix → NCCT
- CT (N) → MRI → DWI most sensitive
- CT angio → vessel block

Contraindications to Thrombolytics
Absolute Contraindications
Prior intracranial hemorrhage
Known cerebral arteriovenous malformation
Known cerebral neoplasm (primary or metastatic)
Ischemic stroke within 3 months
Suspected aortic dissection
Active bleeding or bleeding diathesis (Excludes menses)
Significant trauma within past 3 months
Relative Contraindications
Severe uncontrolled HTN on presentation (SBP >180mmhg or DBP >110mmHg)
Prolonged (>10min) CPR
History of prior ischemic stroke >3 months
Major surgery <3 weeks
Recent internal hemorrhage (Within 2 to 4 weeks)
Noncompressible vascular punctures
Pregnancy
Active peptic ulcer
Current use of anticoagulants

Computerized tomography findings of lobar, subdural, intraventricular, or subarachnoid hemorrhage
History of intracerebral hemorrhage
Cerebral arterio-venous malformation or giant thrombosed cerebral aneurysm*
Brain tumor (meningioma not included)
Computerized tomography evidence of acute >1/3 middle cerebral artery territory
Infarct or large ischemic core on perfusion imaging†
Uncontrolled hypertension >185/110 mm Hg (despite medical intervention)
Unknown stroke duration or duration >4.5 hours
Thrombocytopenia <100,000
Bleeding diathesis or internal bleeding within 21 days
International normalized ratio (INR) >1.7
History of advanced Alzheimer's disease or amyloid angiopathy†
Seizure at stroke onset (unless an acute arterial occlusion is documented)
Recent surgery or trauma within 14 days
Intracranial or spinal surgery, head trauma, or stroke within 3 months
Age >80 years old‡
History of prior stroke and diabetes‡
Any anticoagulant use regardless of INR‡

*Unruptured, incidental, nonthrombosed aneurysms are not a contraindication.
†Relative contraindication.
‡Contraindications for thrombolysis between 3 hours to 4.5 hours from stroke onset.

Which one of the following drugs helps to maintain abstinence by reducing craving for alcohol?

(a) Apomorphine

(b) Acamprosate

(c) Atropine

(d) Azathioprine

Medications for the Alcohol Rehabilitation Treatment Phase

Several medications have modest benefits when used in the first 6–12 months of recovery. The opioid antagonist, naltrexone, may shorten subsequent relapses, whether used in the oral form (50–150 mg/d) or as a once-per-month 380-mg injection. By blocking opioid receptors, naltrexone decreases activity in the dopamine-rich ventral tegmental reward system and decreases the feeling of pleasure if alcohol is imbibed. A second medication, acamprosate (Campral) (~2 g/d divided into three oral doses), has similar modest effects. Acamprosate inhibits NMDA receptors, decreasing mild symptoms of protracted withdrawal. Several trials of combined naltrexone and acamprosate have reported that the combination is well tolerated and the efficacy might be superior to either drug alone, although not all studies agree.

It is more difficult to establish the asset-to-liability ratio of a third drug, disulfiram, an ALDH inhibitor, used clinically at doses of 250 mg/d. This drug produces vomiting and autonomic nervous system instability in the presence of alcohol as a result of rapidly rising blood levels of acetaldehyde. This reaction can be dangerous, especially for patients with heart disease, stroke, diabetes mellitus, or hypertension. The drug itself carries potential risks of temporary depressive or psychotic symptoms, peripheral neuropathy, and liver damage. Disulfiram is best given under supervision by someone (such as a spouse), especially during high-risk drinking situations (such as the Christmas holidays). Additional drugs under investigation include another opioid antagonist nalmefene, the nicotinic receptor agonist varenicline, the serotonin antagonist ondansetron, the α-adrenergic agonist prazosin, the $GABA_B$ receptor agonist baclofen, the anticonvulsant topiramate, and cannabinol receptor antagonists. At present, there are insufficient data to determine the asset-to-liability ratio for these medications in treating alcohol use disorders and, therefore, few data yet offer solid support for their routine use in clinical settings.

Typical absence seizures are characterized by

(a) abrupt 3 Hz spike-and-slow wave discharges on EEG

(b) postictal confusion in children

(c) multifocal structural abnormalities of brain

(d) less responsiveness to anti-convulsants as compared to atypical absence seizures

ethosux Absence seiz valproate

Factor	Typical	Atypical
Age of onset	Childhood	Any age
Onset/offset of seizure	Abrupt	Often gradual
Consciousness	Totally lost	Often partially impaired
Other clinical features during seizure	Slight (eye flickering)	Can be prominent, including aura, automatism
Duration of seizures	Short (usually <10 sec)	Long (usually several minutes)
Frequency of seizures	Numerous, frequently in clusters	Usually less frequent
Postictal	None	Confusion, headache, emotional disturbance are common
Coexisting seizure types	Sometimes tonic-clonic and myoclonic	Mixed seizure disorder is common; all seizure types
Cause	Idiopathic generalized epilepsy	Any focal pathology or probably symptomatic epilepsy
Underlying focal anatomic lesion	None	Limbic structures, neocortex
Other neurologic signs and symptoms	None	Usually learning difficulties
Ictal EEG appearance	3-Hz spike and wave	2 to 2.5-Hz spike and wave
Interictal EEG appearance	Usually normal	Abnormal

Osborn waves in ECG show prolonged repolarization with a distinctive convex elevation of the 'J' point. These waves are associated with

(a) systemic hypothermia

(b) acute pericarditis

(c) acute myocarditis

(d) Brugada syndrome

Thickened ventricular wall with normal diastolic function is a feature of

(a) hypertrophic cardiomyopathy

(b) restrictive cardiomyopathy → RCM

(c) endomyocardial fibrosis →

(d) athlete's heart

Causes of diastolic dysfunction

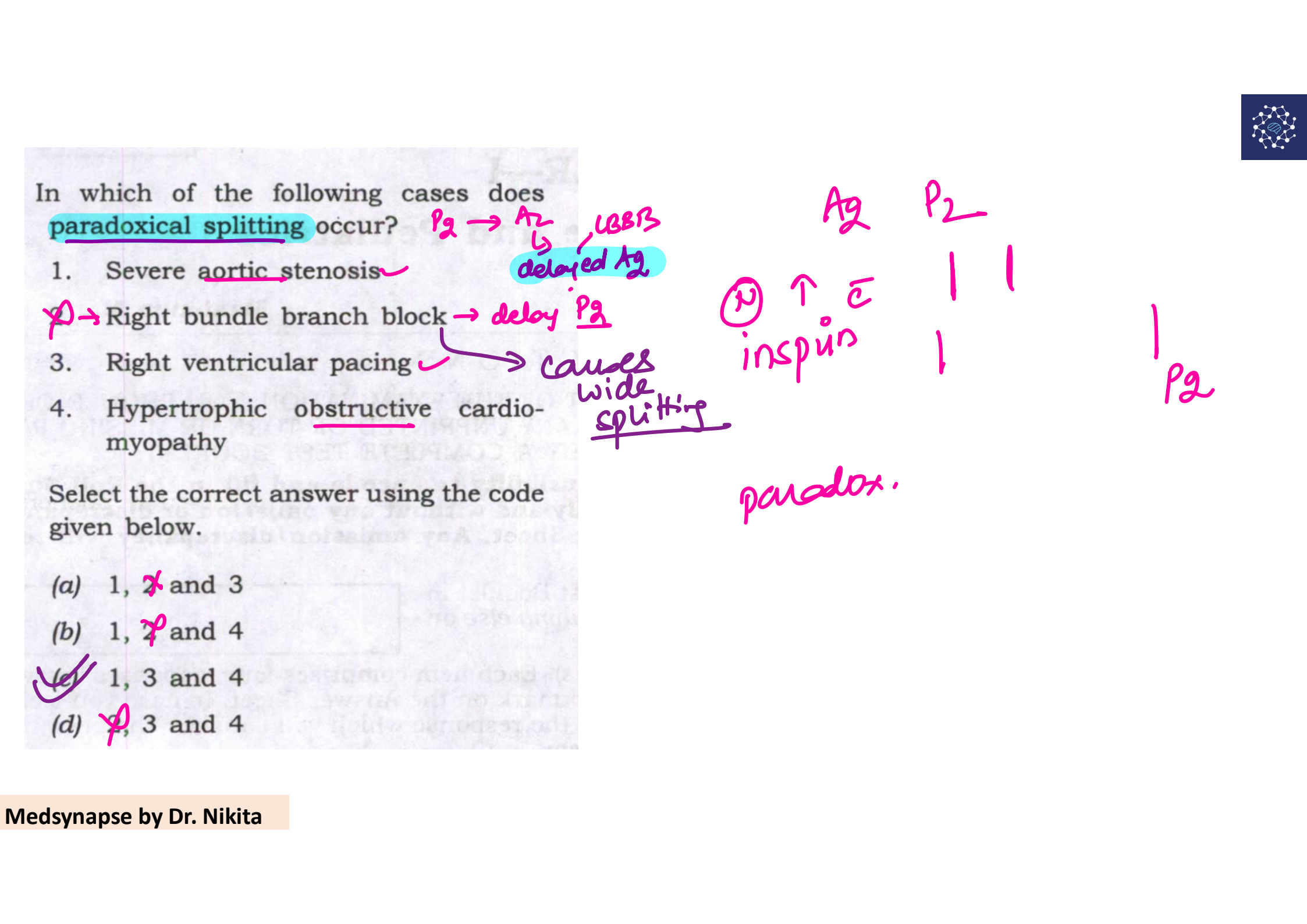
Common causes
Ischaemic cardiomyopathy
Systemic hypertension
Hypertrophic cardiomyopathy
Aortic valve stenosis
Other causes
Infiltrative cardiomyopathies
Amyloidosis
Sarcoidosis
Iron overload cardiomyopathy (primary or secondary)
Other storage diseases
Glycogen storage disease
Anderson-Fabry disease
Mucopolysaccharidosis
Fibroplastic cardiomyopathies
Endomyocardial fibrosis
Endocardial fibroelastosis
Loffler's fibroplastic endocarditis
Pericardial disorders
Constrictive pericarditis
Pericardial effusion and tamponade

In which of the following cases does paradoxical splitting occur?

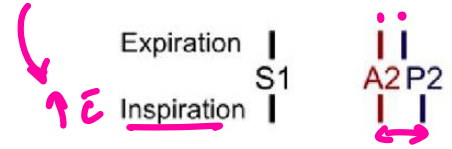
1. Severe aortic stenosis
2. Right bundle branch block
3. Right ventricular pacing
4. Hypertrophic obstructive cardiomyopathy

Select the correct answer using the code given below.

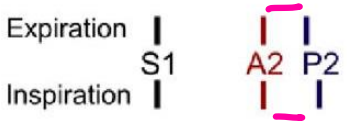
(a) 1, 2 and 3
(b) 1, 2 and 4
(c) 1, 3 and 4
(d) 2, 3 and 4



Normal Splitting



Wide Splitting



Pathological Examples

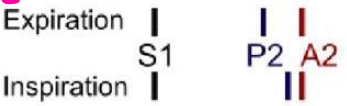
- Right bundle branch block (RBBB)
- Pulmonary hypertension with right heart failure
- Outflow obstruction of right ventricle (e.g., pulmonary stenosis)
- Left-ventricular pre-excitation (e.g., Wolff-Parkinson-White syndrome)

Fixed Splitting

Pathological Examples

- Atrial septal defects

Paradoxical Splitting



Pathological Examples

- Left bundle branch block (LBBB)
- Aortic stenosis

A patient with peripheral edema has the following findings on clinical examination :

A soft systolic murmur at the lower left sternal border with raised JVP showing prominent C-V wave. The murmur increases in intensity on deep inspiration. Rt sided.

The most likely valvular abnormality is

(a) ventricular septal defect

(b) mitral regurgitation

(c) tricuspid regurgitation

(d) mitral valve prolapse

A 28-year male is suspected of having hypertrophic obstructive cardiomyopathy (HOCM). Which of the following statements are likely to be true on his examination?

1. Maneuvers that decrease LV preload will cause the murmur to intensify.

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2. Maneuvers that decrease LV afterload will cause decrease in intensity of murmur.
3. Murmur of HOCM becomes softer with passive leg raising.
4. Murmur of HOCM becomes louder with squatting.

Select the correct answer using the code given below.

(a) 1 and 3
(b) 2 and 3
(c) 2 and 4
(d) 1 and 4

↑ C → ↓ AST

amyl nitrite ↓ afterload

. standing
. ↓ VR / preload

(F) → ↓ afterload → ↑ contractility → ↓ LV size → ↓ VR

↓ LV size → murmur. → ↑ murmur.

HOCM murmur ↑ C
① preload ↓
② afterload ↓

A 56-year male, chronic heavy smoker, presented with breathlessness. On examination, pulse : 96/minute, BP : 11[illegible] and a pansystolic murmur is showing Carvallo's sign with murmur getting louder on deep inspiration. Which one of the following statements is true regarding examination of JVP in him?

(a) V wave is attenuated.

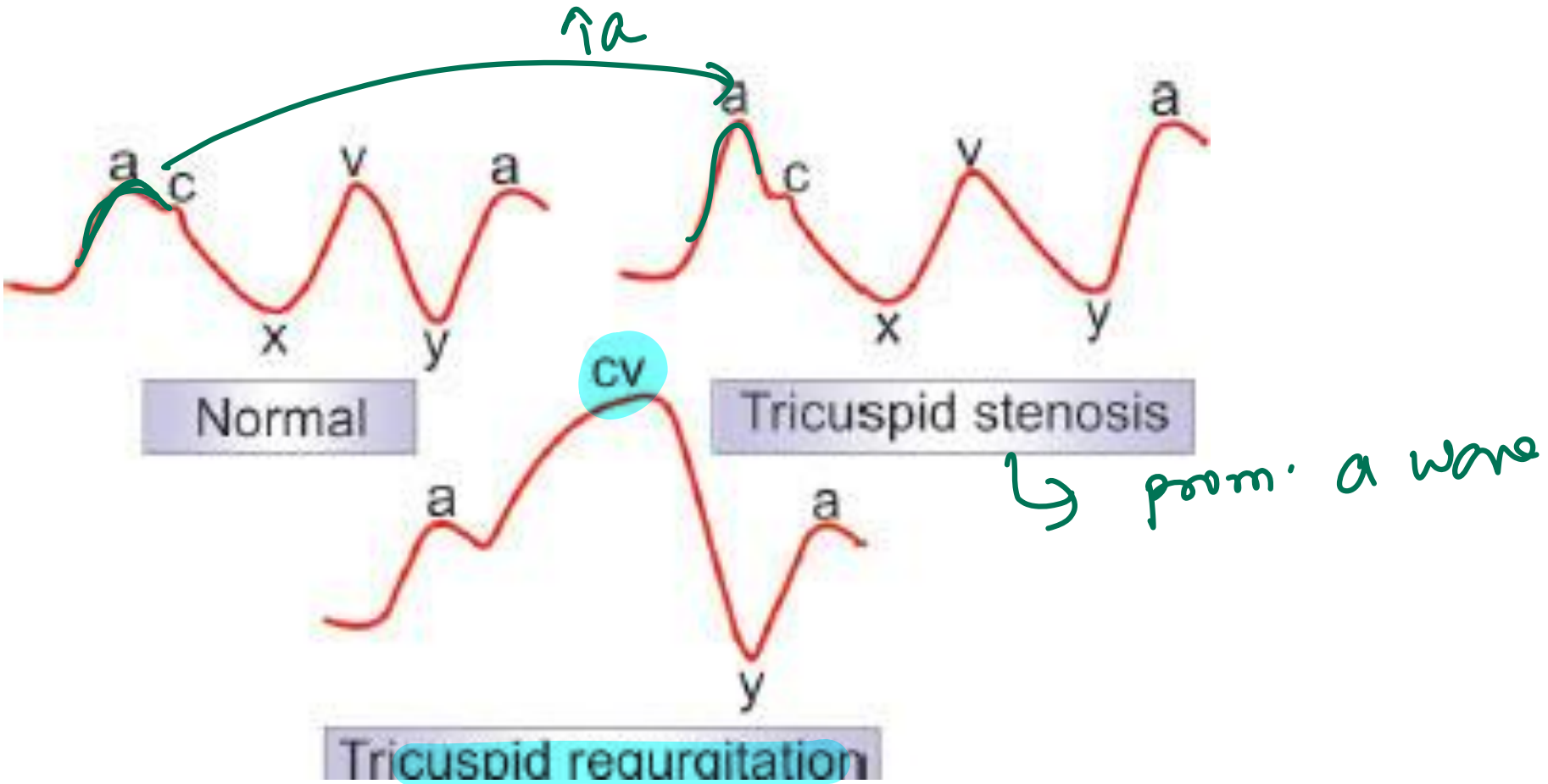
(b) C wave is attenuated and V wave is accentuated.

(c) V wave and C wave merge.

(d) V wave and C wave merge and Y descent is blunted.

TR | MR | VSD

CV


↑a
a
c
v
a
x
y
Normal
a
c
v
a
x
y
Tricuspid stenosis
cv
a
a
y
Tricuspid regurgitation

↳ prom. a wave

Kussmaul's sign is a clinical feature of

(a) constrictive pericarditis

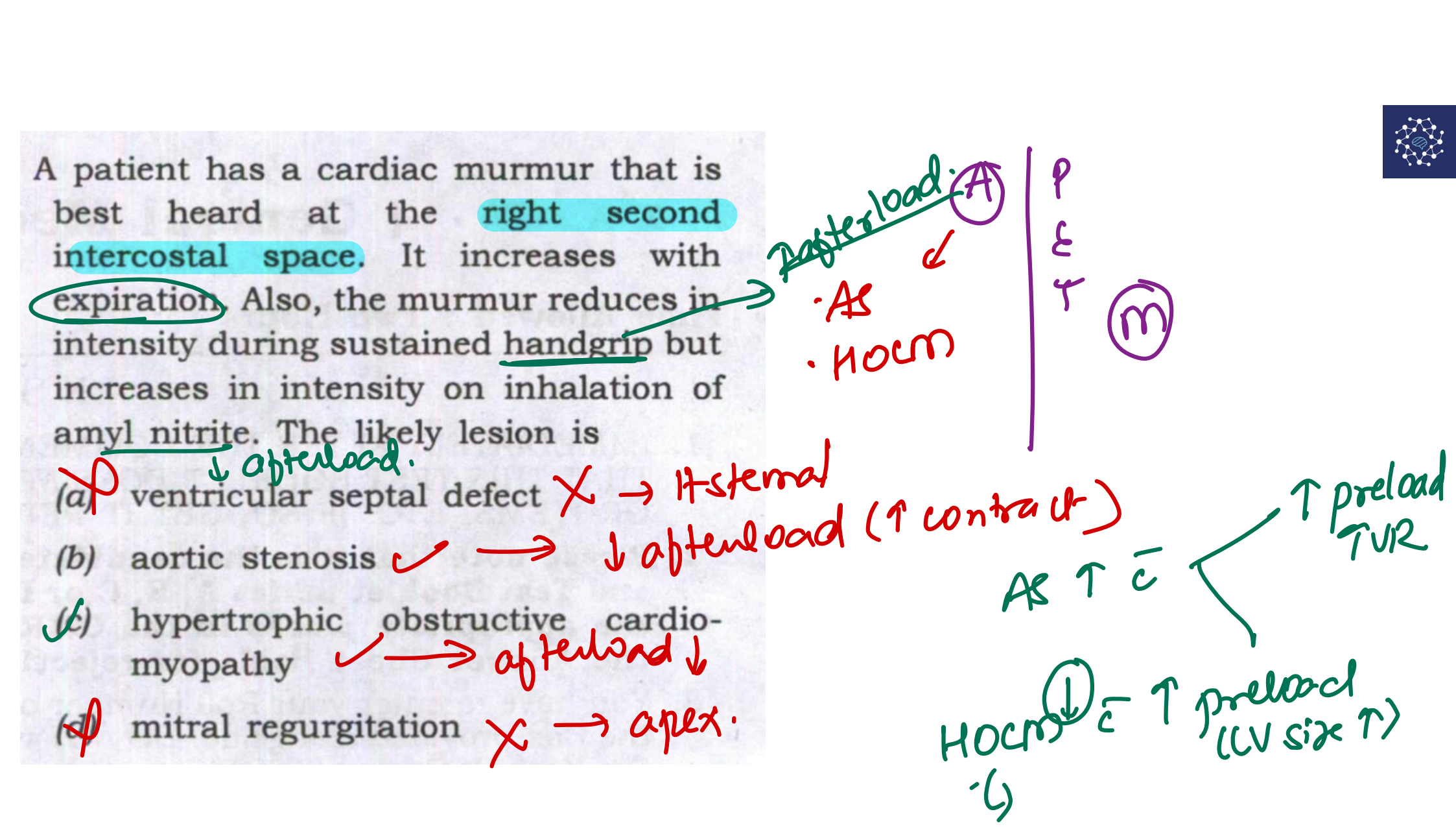
(b) hypertrophic obstructive cardiomyopathy

(c) anteroseptal myocardial infarction

(d) dilated cardiomyopathy

A patient has a cardiac murmur that is best heard at the right second intercostal space. It increases with expiration. Also, the murmur reduces in intensity during sustained handgrip but increases in intensity on inhalation of amyl nitrite. The likely lesion is

(a) ventricular septal defect
(b) aortic stenosis
(c) hypertrophic obstructive cardiomyopathy
(d) mitral regurgitation



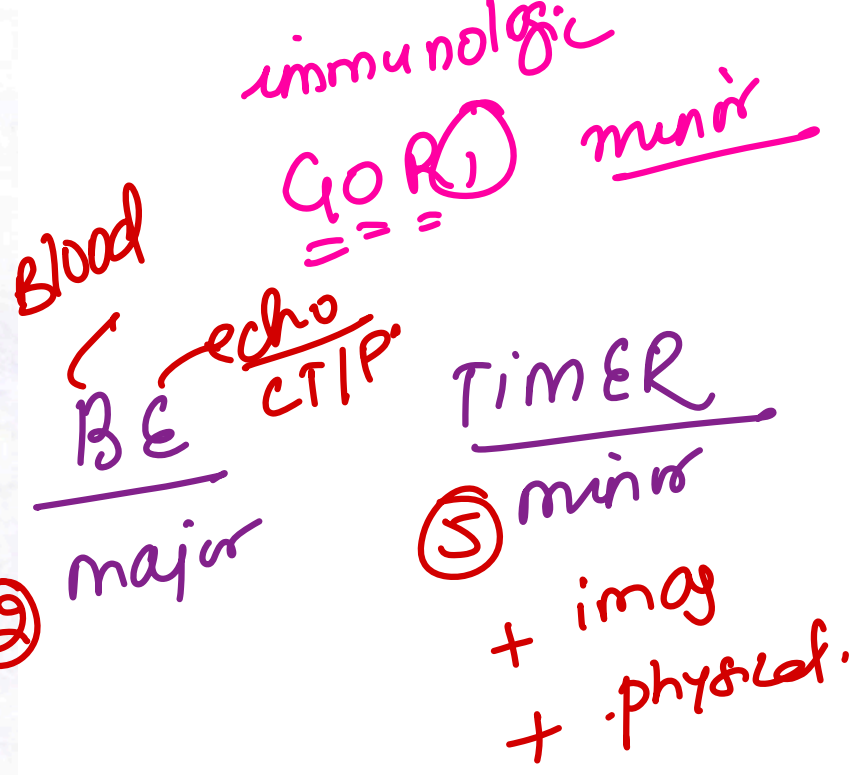
In the modified Duke criteria for infective endocarditis, which one of the following is **not** a major criterion?

(a) New partial dehiscence of prosthetic valve

(b) Positive blood culture

(c) Roth's spots (immunologic)

(d) New valvular regurgitation



Modified Duke Criteria

Pathological Criteria

Positive histology or culture from pathological material obtained at autopsy or cardiac surgery

Major Criteria

Two positive blood cultures with typical organism

Persistent bacteraemia

Positive serology for Coxiella

Positive echocardiogram

1) Vegetation OR
2) Abscess OR
3) New regurgitation OR
4) Dehiscence of prosthetic valves

TIMER

Minor Criteria

Predisposing heart disease or IVDA (Risk factor)

Fever > 38% Temp

Immunological phenomena

Vascular Phenomena (Embolic)

Microbiological evidence not fitting major criteria

2023 Duke-ISCVID criteria IE (1)

Clinical criteria	Definite	• 2 major	• 1 major + 3 minor	• 5 minor
	Possible	• 1 major + 1 minor		• 3 minor
Pathological criteria (1 or 2)	1	Microorganisms identified with clinical sign of active endocarditis from cardiac tissue, prosthesis, ascending aortic graft, CIED or embolus (by culture, PCR or other molecular technique, etc.)		
	2	Active endocarditis identified from cardiac tissue, prosthesis, ascending aortic graft, CIED or embolus		
Rejected criteria (1 or 2 or 3 or 4)	1	Firm alternative diagnosis		
	2	Lack of recurrence despite ATB < 4 days		
	3	No evidence of IE at surgery with ATB < 4 days		
	4	Not meet possible clinical criteria for IE		

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2023 Duke-ISCVID criteria IE (2)

Major criteria			
1. Microbiologic	1. Positive blood culture	• Common cause IE	Require ≥ 2 separate blood culture sets
		Typical pathogens: • *S. aureus, S. lugdunensis, E. faecalis* • All streptococci except *S. pneumoniae* & *S. pyogenes* • *Granulicatella* & *Abiotrophia* spp., *Gemella* spp. • HACEK group **Typical pathogens when +ve intracardiac prosthesis:** • Coagulase-negative Staphylococci • *C. striatum* & *C. jeikeium, S. marcescens, P. aeruginosa, C. acnes* • NTM (esp. *M. chimarae*), *Candida* spp.	
		• Rarely cause IE	Require ≥ 3 separate blood culture sets
	2. Positive laboratory tests	• PCR or NAT for *C. burnetii, Bartonella* spp. or *T. whipplei* from blood • *C. burnetii* antiphase IgG titer > 1:800 or isolated from 1 blood culture • IFA for IgM & IgG to *B. hensalae* or *B. quintana* (IgG ≥ 1:800)	
2. Imaging	• Echo or cardiac CT: vegetation, perforation, aneurysm, pseudoaneursym, abscess or fistula • Significant new valvular regurgitation OR new partial dehiscence of prosthesis compared to previous echo • [18F] FDG PET/CT: abnormal metabolic activity of native or prosthesis (valve, graft, CIED leads or others) perform ≥ 3 months after prosthesis implantation		
3. Surgical	Evidence of IE by direct inspection during heart surgery (irrespective of imaging, histology or microbiology)		

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2023 Duke-ISCVID criteria IE (3)

Minor criteria				
1. Predisposition	Previous history of IE	Prosthetic valve	Previous valve repair	Congenital HD
	> Mild regurgitation or stenosis of any etiology	Endovascular CIED	HOCM	IVDU
2. Fever	T > 38 C			
3. Vascular phenomenon	Septic pulmonary infarct	Cerebral or splenic abscess		Mycotic aneurysm
	Intracranial hemorrhage	Conjunctival H	Janeway lesions	Purulent purpura
4. Immunologic phenomenon	Immune complex GN	Rheumatoid F	Osler's nodes	Roth's spots
5. Microbiologic evidence	Positive blood cultures for organism consistent with IE but not meeting major criteria			
	Positive culture, PCR or other NAT for organism consistent with IE from other sterile site			
6. Imaging	[18F] FDG PET/CT: abnormal metabolic activity of native or prosthesis (valve, graft, CIED leads or others) perform < 3 months after prosthesis implantation			
7. Physical examination	New valvular regurgitation on auscultation if echo not available			

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The 2023 Duke-ISCVID Criteria for Infective endocarditis

Definite endocarditis	Pathologic Criteria	At least 1 of 2
	Clinical Criteria	2 Major
		1 Major and 3 Minor
		5 Minor
Possible endocarditis	Clinical Criteria	1 Major and 1 Minor
		3 Minor

Typical microorganism from blood cultures
(proposed change in **bold** type)

Staphylococcus aureus, ***Staphylococcus lugdunensis***
***Enterococcus faecalis*, all streptococcal species (except for *S. pneumoniae and S. pyogenes*),**
***Granulicatella* spp., *Abiotrophia* spp., and *Gemella* spp.**
HACEK group

For setting of intracardiac prosthetic material:

- **coagulase negative staphylococci**
- ***Corynebacterium striatum* and *C. jeikeium***
- ***Serratia marcescens***
- ***Pseudomonas aeruginosa***
- ***Cutibacterium acnes***
- **NTM (especially *M. chimaerae*)**
- ***Candida* spp.**

Criteria	Change
Pathologic criteria	
• microorganism or active endocarditis identified	Added PCR, amplicon or metagenomic sequencing, or in situ hybridization for microorganism identification from specimen.
Major clinical criteria	
• Microbiology	Removed requirements for timing and separate venipunctures. Added typical pathogens from blood culture. Added PCR or sequencing identified *C. burnetii, Bartonella* sp., or *T. whipplei* and IFA for *B. henselae* or *B. quintana* IgG.
• Imaging	Added cardiac CT and [18f]FDG PET/CT.
• Surgical	Added surgical criteria; intraoperative inspection in absence of cardiac imaging or histopathology.
Minor clinical criteria	
• Predisposition	Added transcatheter valve implant/ repair, endovascular CIED and prior IE.
• Fever	Unchanged(>38 C).
• Vascular phenomena	Added splenic and cerebral abscess.
• Immunologic phenomena	Added definition for immune complex mediated Glomerulonephritis.
• Microbiological	Added PCR or amplicon/metagenomic sequencing evidence of typical pathogen.
• Imaging	Added PET/CT evidence < 3 months of cardiac surgery.
• Physical examination	New auscultation of regurgitant murmur when Echocardiography is unavailable.

Which of the following statements are correct for the treatment of chronic hepatitis B with pegylated interferon (PEG-IFN)?

1. PEG-IFN is poorly tolerated drug as compared to nucleoside analogues.
2. Resistance to treatment with PEG-IFN is common than nucleoside analogues.
3. PEG-IFN is not useful in patients of cirrhosis.
4. PEG-IFN is administered every week for 48 weeks.

Select the correct answer using the code given below.

(a) 1, 3 and 4
(b) 1, 2 and 3
(c) 2 and 4
(d) 1 and 3 only

injections

no resist

Natural (3C)

IFN — α — viral
IFN β → M.S.
IFN γ → C.GD

PEG IFN → ① 48 wks
② no resistance

	(PEG-)IFN	NAs
Advantages	• Finite duration • Absence of resistance • Higher rates of anti-HBe and anti-HBs seroconversion with 12 mo of therapy	• Potent antiviral effect • Good tolerance • Oral administration
Disadvantages	• Moderate antiviral effect • Inferior tolerability • Risk of adverse events • Subcutaneous injections	• Indefinite duration • Risk of resistance • Unknown long-term safety

TABLE 156-1 Comparison of Pegylated Interferon (PEG IFN), Lamivudine, Adefovir, Entecavir, Telbivudine, and Tenofovir Therapy for Chronic Hepatitis B[a]

FEATURE	PEG IFN[b]	LAMIVUDINE	ADEFOVIR	ENTECAVIR	TELBIVUDINE	TENOFOVIR
Route of administration	Subcutaneous injection	Oral	Oral	Oral	Oral	Oral
Duration of therapy[c]	48–52 weeks	≥52 weeks	≥48 weeks	≥48 weeks	≥52 weeks	≥48 weeks
Tolerability	Poorly tolerated	Well tolerated	Well tolerated; creatinine monitoring recommended	Well tolerated	Well tolerated	Well tolerated; creatinine monitoring recommended
HBeAg seroconversion						
1 yr Rx	18–20%	16–21%	12%	21%	22%	21%
>1 yr Rx	NA	up to 50% @ 5 yrs	43% @ 3 yrs[d]	31% @ 2 yrs 44% @ 6 yrs	30% @ 2 yrs	40% @ 5 yrs
Log_{10} HBV DNA reduction (mean copies/mL)						
HBeAg-reactive	4.5	5.5	median 3.5–5	6.9	6.4	6.2
HBeAg-negative	4.1	4.4–4.7	median 3.5–3.9	5.0	5.2	4.6
HBV DNA PCR negative (<300–400 copies/mL; <1000 copies/mL for adefovir) at end of yr 1						
HBeAg-reactive	10–25%	36–44%	13–21%	67% (91% @ 4 yrs)	60%	76%
HBeAg-negative	63%	60–73%	48–77%	90%	88%	93%
ALT normalization at end of yr 1						
HBeAg-reactive	39%	41–75%	48–61%	68%	77%	68%
HBeAg-negative	34–38%	62–79%	48–77%	78%	74%	76%
HBsAg loss yr 1	3–4%	≤1%	0%	2%	<1%	3%
>yr 1	12% 5 yr after 1 yr of Rx	No data	5% at yr 5	6% at yr 6	No data	8% at yr 5
Histologic improvement (≥2 point reduction in HAI) at yr 1						
HBeAg-reactive	38% 6 months after	49–62%	53–68%	72%	65%	74%
HBeAg-negative	48% 6 months after	61–66%	64%	70%	67%	72%
Viral resistance	None	15–30% @ 1 yr	None @ 1 yr	≤1% @ 1 yr[e]	Up to 5% @ yr 1	0% @ yr 1