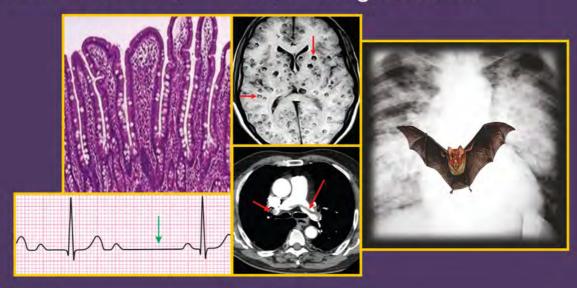


## ONE Touch Medicine



For NEET PG/FMGE/INI-CET/Undergraduates



#### **Special Features**

- · Thoroughly revised and updated edition
- Enriched with latest updates up to Nov 2024
- Previous years' papers coverage (last 5 years)
   up to Nov 2024 (INI-CET Nov 2024 and NEET PG 2024)
- Topic-wise Case Scenario Questions
- Latest 2024 Qs with explanations
- Complete subject is covered in the form of Tables, Figures, Flowcharts, One-liners for last-minute revision in just 280 pages.



2nd Edition

## ONE Touch Medicine



For NEET PG/FMGE/INI-CET/Undergraduates

**Second Edition** 

#### **Deepak Marwah**

Director

Medicine Buster Classes

Dedicated to Education



#### **CBS Publishers & Distributors Pvt Ltd**

### ONE Touch Medicine

For NEET PG/FMGE/INI-CET/Undergraduates

ISBN: 978-93-48426-40-6 Copyright © Publishers Second Edition: 2025

First Edition: 2024

All rights are reserved. No part of this book may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopying, recording, or any information storage and retrieval system without permission, in writing, from the publishers.

Published by Satish Kumar Jain and produced by Varun Jain for

#### **CBS Publishers & Distributors Pvt Ltd**

4819/XI Prahlad Street, 24 Ansari Road, Daryaganj, New Delhi 110 002, India. Ph: +91-11-23289259, 23266861, 23266867 Website: www.cbspd.com

Fax: 011-23243014

e-mail: delhi@cbspd.com; cbspubs@airtelmail.in.

Corporate Office: 204 FIE, Industrial Area, Patparganj, Delhi 110 092

Ph: +91-11-4934 4934 Fax: 4934 4935 e-mail: feedback@cbspd.com; bhupesharora@cbspd.com

#### Branchoo

• Bengaluru: Seema House 2975, 17th Cross, K.R. Road, Banasankari 2nd Stage, Bengaluru-560 070, Karnataka

Ph: +91-80-26771678/79 Fax: +91-80-26771680 e-mail: bangalore@cbspd.com

• Chennai: 7, Subbaraya Street, Shenoy Nagar, Chennai-600 030, Tamil Nadu

Ph: +91-44-26680620, 26681266 Fax: +91-44-42032115 e-mail: chennai@cbspd.com

• Kochi: 68/1534, 35, 36-Power House Road, Opp. KSEB, Cochin-682018, Kochi, Kerala

Ph: +91-484-4059061-65 Fax: +91-484-4059065 e-mail: kochi@cbspd.com

• Kolkata: Hind Ceramics Compound, 1st Floor, 147, Nilganj Road, Belghoria, Kolkata-700056, West Bengal Ph: +033-2563-3055/56 e-mail: kolkata@cbspd.com

• Lucknow: Basement, Khushnuma Complex, 7-Meerabai Marg (Behind Jawahar Bhawan), Lucknow-226001, Uttar Pradesh

Ph: +0522-4000032 e-mail: tiwari.lucknow@cbspd.com

• Mumbai: PWD Shed, Gala No. 25/26, Ramchandra Bhatt Marg, Next to J.J. Hospital Gate No. 2, Opp. Union Bank of India, Noor Baug,

Mumbai-400009, Maharashtra

Ph: +91-22-66661880/89 Fax: +91-22-24902342 e-mail: mumbai@cbspd.com

#### Representatives

• Hyderabad +91-9885175004 • Jharkhand +91-9811541605 • Nagpur +91-9421945513

• Patna +91-9334159340 • Pune +91-9623451994 • Uttarakhand +91-9716462459

Printed at:



#### Dear Students,

It gives me immense pleasure to introduce you to the new edition of "One Touch Medicine", which is a magical bundle of highly organized notes providing you with a laser-focused approach for real-life exam scenario to solve questions with twisted/super-confusing options.

The book has been designed keeping in mind the fact that one needs an interesting and high-yield colorful summary book which he/she would love to open in the morning to learn interesting facts and revise the standard content.

I suggest you to finish the theory part in three to four days by reading 40-50 pages per day. Each word should be imprinted in your brain, as it has been well said that doctors have a photographic memory. In the next step, revise all the PYQ papers (Latest Question Papers) of NEET, INI-CET and FMG for the last 5 years up to November 2024 given in the book. Latest 2024 Qs have been covered with their explanations. Detailed explanation of each of the papers is available free on my YouTube channel: https://www.youtube.com/@DrMarwahLIVE. Keep the book with you while watching the video and underline the keywords in the stem of each question as I discuss each of them. It will help you learn critical exclusion skills for the current exam pattern.

The book contains multiple case scenarios that will be of help to you in your final year of medical college as well. Remember! It is an era where you need precise data to be able to decide why, when, how, and what to do in a case.

Keep Hammering!

**Deepak Marwah** 



### From the Publisher's Desk

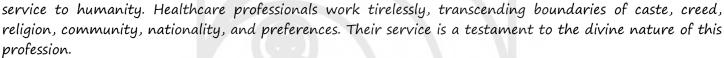
Dear Students,

Let us begin with a power-packed and inspiring quote:

Arise, awake, and stop not until the goal is achieved.

-Swami Vivekananda

Healthcare is undoubtedly one of the most noble and sacred professions. We are truly fortunate to be a part of this field, which stands as a beacon of selfless



We extend our deepest gratitude to all healthcare professionals for their unwavering commitment, particularly during the pandemic. When the world retreated behind closed doors, these brave individuals stood on the frontlines, leaving no stone unturned in saving the lives of people.

At CBS Publishers, we take great pride in supporting the healthcare community by offering resources that empower future professionals. Ten years ago, we laid the foundation in the PGMEE segment with titles such as the Conceptual Review Series, SARP Series, AIIMS MedEasy, NIMHANS, PGI Chandigarh, My PGMEE Notes, ROAMS, PRIMES, FMGE Solutions and many more.

What makes our PGMEE books stand out is the updated, simple, clear, and easy-to-understand language, making study sessions feel less like a challenge and more like an enjoyable learning experience. A team of our esteemed medical educators brings their expertise to create these comprehensive yet compact books, ensuring that all the critical topics are covered.

A special feature of our books is the use of illustrations that simplify complex concepts, making them easier to grasp. We also include previous years' questions, complete with detailed explanations, which are invaluable for exam preparation. Image-Based Questions (IBQs) further enhance the learning experience. The combination of concise theory and multiple-choice questions makes these books the ultimate tool to ease exam-related worries.

FMGE Solutions is one of our best-selling titles, meticulously designed to meet the specific needs of FMG aspirants. This comprehensive guide is an all-in-one resource for FMGE preparation, offering in-depth coverage of essential topics, detailed explanations, and a wide array of questions that reflect the latest exam patterns. Its reputation as a bestseller speaks to its effectiveness and reliability as a trusted resource for future medical professionals.

One Touch Series, is tailored specifically for aspirants of NEET PG, NEXT, FMGE, and INI-CET. Conceptualized with a focus on last-minute revision, the One Touch Series covers a complete range of preclinical, paraclinical, and clinical subjects. These concise, expertly curated books are designed to help students efficiently review key concepts, ensuring they are well-prepared and confident as they approach their exams.

This year, we have introduced a new addition to the CBS Exam Book Series: **Ten into Ten** (Part A and B). According to the market research, at present no book is available for practice and this new addition to our exam book series will fill this gap for sure. Although there are multiple apps from where students can



attempt test series online, not a single updated book is available in the market for offline practice, and this book now in your hand, will fill this vacuum. The motto of this book is Practice: Practice: Practice as this book offers a decent amount of MCQs which will meet the evolving needs of students. **Ten into Ten** is a comprehensive question bank covering 19 medical subjects. It offers over 10,000 meticulously curated questions across 10 key subjects, crafted by 10 renowned medical scholars.

Following this, we will soon release the next part, **Nine into Nine**, further expanding our collection of practice materials for the PGME Examination, with the latest and most effective study approaches.

At CBS, we are committed to revolutionize the medical education and your support and encouragement can make our task easier. So, keep extending your support by sending your feedback to us. We will be highly pleased to serve you and make you victorious in your career. You can share your feedback at feedback@cbspd.com

Wishing you all the best in your endeavors.

Mr Bhupesh Aarora (Sr. Vice President – Publishing & Marketing)

(Sr. Vice President – Publishing & Marketing) bhupeshaarora@cbspd.com| +91 95553 53330



## Contents

THEORY	
Cardiology and Emergency Medicine	
schemic Heart Disease	
Summary for Cardiac Biomarkers	
Infarct Localization at a Glance	
Treatment of STEMI	
Cardiomyopathy	
Integrated Pathology Slide Interpretation	
Therapeutics of Congestive Heart Failure	2
Summary of Important JVP Findings	
ECG Changes	
Causes of ST Depression	
Abnormal ECG Findings	
Heart Rate Determination	
Accessory Pathway-Mediated Disorders	2
ECG Rhythm Disorders	
Bradyarrythmias	2
Hypokalemia	2
Diseases of Pericardium	2
Infective Endocarditis Inclusive of 2023 Update	
What Should You Know About the 2023 Updated Duke ISCVID Criteria?	2
Major Criteria	2
Minor Criteria	2
Rheumatic Fever	
Murmurs	
Aortic Dissection	
Hypertension	
Congenital Heart Defects	
Coarctation of Aorta	
ACLS AHA Guidelines	
Basic Life Support	3
Neurology	
EEG Waves	
Montage Recording Interpretation	
Epilepsy	
Generalized Status Convulsive Epilepticus (GCSE)	
CNS Infections	

Lumbar Puncture (LP)......50

	Neurocysticercosis	
	Brain Tumor	52
	Pediatric Brain Tumors	53
	Transient Ischemic Attack (TIA)	53
	Clinical Features of Localized Cerebral Lesions	54
	Neuroimaging Findings in Acute Ischemic Stroke	56
	Intracerebral Hemorrhage	57
	Stroke Syndromes by Vascular Territory	
	Distinct Clinical Syndromes Associated with Lacunar Infarcts	
	Other Causes of Intracranial Bleeding	
	Headache	
	Myasthenia Gravis	
	Channelopathies	
	Parkinsonism	
3.	Endocrinology	
	Diabetes Mellitus	
	Thyroid Gland	75
	Multiple Endocrine Neoplasia (MEN)	77
4.	ABG Analysis	
	Effect of Extra Heparin on ABG Sample	7.0
	ABG Hacks	
	How to Calculate the Level of Compensation in Respiratory Acidosis and Respiratory Alkalosis?	00
	MMRC Grading of Dyspnea: Modified Medical Research Council Grading of Dyspnea	
	for COPD Patients	
	Anion Gap	
	Metabolic Alkalosis	
	Respiratory Acidosis	
	Respiratory Alkalosis	
	Respiratory Aikaiosis	
5.	Pulmonology and Spirometry	
<u> </u>		
	Pneumothorax	87
	Pleural Effusion	
	Characteristics of Important Exudative Pleural Effusions	89
	Hydropneumothorax	90
	Pneumonia	90
	Pulmonary Embolism (PE)	92
	Fat Embolism Syndrome	93
	Spirometry Interpretation	
	Diffusion Capacity of Lung	
	Pulmonary Capacity	
	Tuberculosis	
	Severity of Asthma	
	Air Embolism	
	Bronchiectasis	
	Cystic Fibrosis	
	Respiratory Failure	
	Respiratory rallure	101

	Acute Respiratory Distress Syndrome (ARDS)	103
	Interstitial Lung Disease (ILD)	104
6.	Nephrology and Electrolytes	
	Important Facts	108
	Chronic Kidney Disease	
	Nephrotic Syndrome	
	Hematuria and Nephritic Syndrome	
	Hyponatremia	
	Hypernatremia	
	Disorders of Phosphate Metabolism	
	Summary of Electrolyte Imbalances	
7.	Hepatology	
	Important Facts	118
	Important Scoring Patterns in Liver Disease	
	Acute Liver Failure	
	Hepatitis B	
	Hepatitis C	
	Hepatitis D	
	Hepatitis A and E	
	Extrahepatic Manifestations of Hepatitis B and Hepatitis C Viruses	
	Incidence of Fulminant Hepatic Failure and Chronic Hepatitis with Hepatotropic Viruses	
	Autoimmune Hepatitis	
	Alcoholic Liver Disease	
	Nonalcoholic Fatty Liver Disease	
	Cirrhosis	
	Grading of Severity of Liver Cirrhosis	
	Features of Decompensated Cirrhosis	
	Portal Hypertension	
	Wilson Disease	
	Budd-Chiari Syndrome	
8.	Rheumatology Dedicated to Education	
	Investigation of Choice for Various Rheumatological Conditions	131
	Patterns in Various Rheumatic Conditions	
	Systemic Lupus Erythematosus	132
	Antiphospholipid Antibody Syndrome (APLAS)	
	Autoantibodies in SLE	
	Scleroderma (Systemic Sclerosis)	135
	Mixed Connective Tissue Disorder	137
	Sjögren's Syndrome	
	Behçet's Syndrome	
	Dermatomyositis	
	Polymyositis	
	Arthritis	
	Sarcoidosis	
	Vasculitis	

 INI-CET NOVEMBER 2022 (Memory-Based)
 222

 INI-CET MAY 2022 (Memory-Based)
 228

 INI-CET NOVEMBER 2021 (Memory-Based)
 232

 INI-CET JULY 2021 (Memory-Based)
 235

 FMGE JULY 2024 (Memory-Based)
 240

 FMGE JANUARY 2024 (Memory-Based)
 246

 FMGE JULY 2023 (Memory-Based)
 253

 FMGE JANUARY 2023 (Memory-Based)
 257

 FMGE JUNE 2022 (Memory-Based)
 261

 FMGE DECEMBER 2021 (Memory-Based)
 264





#### Cardiology and Emergency Medicine

#### ISCHEMIC HEART DISEASE

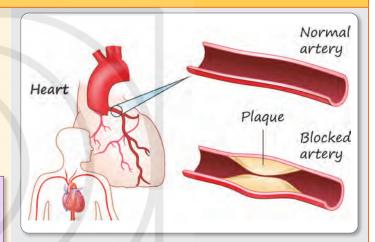
#### Chronic Stable Angina (CSA)

- Occurs due to atherosclerotic narrowing of vessels. Obstruction >70% in any coronary artery will lead to chest pain on exercise like climbing stairs or walking fast.
- · Cardiac biomarkers are always normal.
- Treadmill test/Stress Echocardiography is screening test for work-up

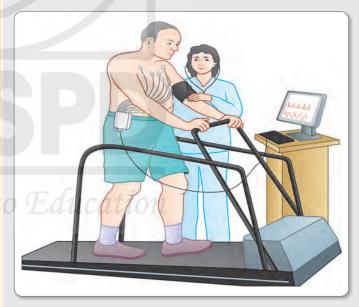
Absolute contraindications for treadmill test

#### Mnemonic: A-PUMP

- Aortic stenosis (severe), Aortic dissection, Arrythmias
- · Pulmonary embolism
- · Unstable angina
- Myocardial infarction
- Pericarditis
- Coronary CT angiography is the investigation of choice for diagnosing CSA. Gold standard is conventional coronary angiography.
- PET-CT differentiates scarred myocardium due to previous MI vs hibernating myocardium of chronic stable angina.
- Drug causing maximum reduction in mortality in chronic stable angina is metoprolol (DOC).
   Other medications for life long use include low dose aspirin 75 mg, statins, Isosorbide mononitrate, sublingual nitrate for emergency use and medications for control of Hypertension and diabetes mellitus.



Chronic stable angina



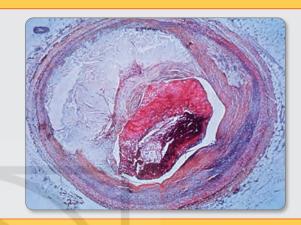
Treadmill test

Revascularization strategy for chronic stable angina is also called Hibernating myocardium

- Triple vessel disease: CABG (Coronary Artery Bypass Graft)
- Double or single vessel disease: PCI (percutaneous coronary intervention) with drug-eluting stents
- LAD (left anterior descending) occlusion with low ejection fraction <40%, the answer as CABG
- Preferred bypass graft used is Internal mammary graft.

#### STEMI (ST-Segment Elevation Myocardial Infarction)

- Fibrin rich clot. Hence, fibrinolysis works.
- Notice that fat in atherosclerotic plague is washed off during preparation of slide.
- ST elevation present and is called Pardee sign/Tombstone pattern.
- Cardiac Biomarkers are elevated. Rise and fall of biomarkers with one value >99th centile of upper reference limit.
- First investigation done is ECG and best for confirmation of diagnosis is cardiac Troponin I.



#### NSTE-ACS (Non-ST-Elevation Acute Coronary Syndrome)

- Platelet rich clot and hence, thrombolysis is contraindicated.
- Causes complete luminal occlusion.
- Hence, biomarkers are elevated. Rise and fall of biomarkers with one value >99th centile of upper reference limit.

#### Unstable Angina

- Platelet rich clot and hence, fibrinolysis is contraindicated.
- Difference from NSTEMI is—It does not cause complete luminal occlusion (Hence, biomarkers are normal).
- · Small clot breaks down sending shower of microemboli downstream into penetrating branches.
- ECG findings of NSTEMI and unstable angina are same as shown in inset.

The plague ruptures and a thrombus forms around the ruptured plague, causing partial occlusion of the vessel. Angina pain occurs at rest or progresses rapidly over a short period of time.

> Subendocardial ischemia, no infarct

Inverted T waves and/or ST depression

### Dedicated to

#### Prinzmetal Angina

- Spasm of epicardial coronary artery. IOC is coronary angiography where spasm can be demonstrated angiographically by injection of acetylcholine in coronaries and relief by nitrates. Hence, it is also called vasospastic angina.
- Only angina with ST elevation. Hence, also called variant angina. Biomarkers are normal
- Right coronary artery is more involved as compared to left
- Past medical history may mention coexistence of vasospastic disorder in the form of Raynaud's phenomenon.

Pharmacological management of STEMI is **same** as NSTE\_ACS with **exception** of alphabet **T** where instead of **T**irofiban in NSTE\_ACS, it would be **T**hrombolysis in STEMI (if patient goes to facility where PCI is not available).

Aspirin 325 mg is the first drug administered along with Ticagrelor 180 mg followed by oxygen, sublingual nitrates and morphine. Note dual antiplatelet therapy is recommended.

#### Mnemonic: BEST MOAN

#### Mnemonic for Pharmacological Management of STEMI and NSTE\_ACS

Beta blocker: Metoprolol to reduce infarct size Enoxaparin: To prevent thrombus progression Statin: To stabilize atherosclerotic plague

Tirofiban: IV GP IIb/IIIa platelet inhibitors before taking up patient for PCI. Thrombolysis is contraindicated.

Morphine: To utilize vagotonic action

Oxygen: To improve SpO, and reduce infarct size

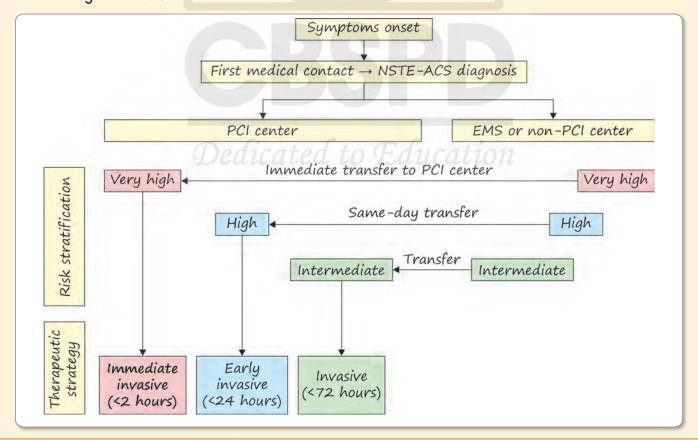
Aspirin: To prevent platelet aggregation

Nitrates: To improve coronary flow and reduce infarct size

#### **MUST KNOW**

- GP IIb/IIIa inhibitors are Eptifibatide, Tirofiban and Cangrelor and any one can be used in NSTE\_ ACS. Tirofiban was selected for sake of mnemonic.
- Cath lab procedure of PCI is done also in NSTE\_ACS within 2 hours of presentation in case of very high-risk patient.

#### Treatment Algorithm: NSTEMI



#### One-Liners

- Door-to-needle time for thrombolysis in STEMI: 30 minutes
- Window period for thrombolysis in STEMI: 12 hours
- · Window period for thrombolysis in Acute ischemic stroke: 4.5 hours
- Upper limit of thrombolysis in acute ischemic stroke: 6 hours
- Thrombolysis is also called fibrinolysis
- Leading side effect of fibrinolysis: Bleeding which can be in the form of intracerebral bleeding
- Antidote for STK toxicity: Epsilon aminocaproic acid (EACA)
- · Antidote for dabigatran toxicity: Idarucizumab
- Antidote for apixaban toxicity: Andexanet alfa
- Rescue PCI is done in: Case of failure of reperfusion by thrombolysis. It is documented by persistence of chest pain and ECG changes of ST elevation persisting beyond 90 minutes of start of infusion.

#### CARDIOMYOPATHY

Characteristics	НСМ	DCM	RCM (Rarest subtype)
Characteristics Leading cause	<ul> <li>HCM</li> <li>Autosomal dominant</li> <li>MYH7 Gene on chromosome 14</li> <li>HOCM is a subtype that has LV outflow tract obstruction called subvalvular aortic stenosis.</li> </ul>	1. Toxin-induced: Alcohol 2. Sequelae of viral myocarditis 3. Titin gene mutation 4. Duchenne muscular dystrophy	RCM (Rarest subtype)  Mnemonic: ACE_SIR  • Amyloidosis (MC)  • Carcinoid syndrome  • Endomyocardial fibroelastosis  • Sarcoidosis  • Scleroderma  • Iron excess/ Hemochromatosis
Gross specimen	Banana-shaped Cavity of left ventricle Predominant diastolic malfunction as LV cavity size is smaller due to asymmetrical septal hypertrophy. Increased thickness of free wall of left ventricle.	Normal  Normal  Normal  A second of the chambers.	• Radiation-induced  Notice the grossly enlarged left and right atria which appear to be bigger than size of ventricles

#### **MUST KNOW**

Causes of ST-segment elevation

#### Mnemonic: ELEVATION-AB

E : Early repolarization variant

L : LBBB

E : Electrolyte imbalance like Hyperkalemia

VA: Ventricular Aneurysm which is complication of MI

T: Trauma during pericardiocentesis or myocardial contusion in car crash

I : Injury due to ischemia having STE with convexity

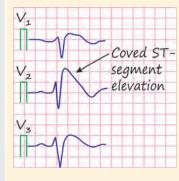
O: Osborn wave/J wave seen in Hypothermia (Core temperature <35°C)

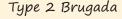
N : Nonocclusive vasospasm (Prinzmetal angina)A : Acute pericarditis having STE with concavity

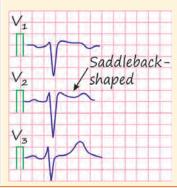
B: Brugada syndrome having Coved ST elevation

## Myocardial infarction • STE with convexity • Present only in overlying leads where infarcted area is occluded. STE with concavity seen in most leads as diffuse involvement of heart is seen. STE with coved pattern/Saddleback pattern seen in lead V<sub>1</sub> and V<sub>2</sub> Type 1 Brugada Type 1 Brugada Type 1 Brugada Type 1 Brugada Type 1 Brugada

Called Pardee sign, Tombstone pattern.





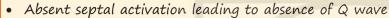


#### ACCESSORY PATHWAY-MEDIATED DISORDERS

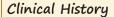
#### WPW Syndrome

#### Pathophysiology

- Early contraction of partially filled ventricles, leading to low cardiac output and resultant symptoms
- · Fast conduction via Bundle of Kent leading to short PR interval



Slower inter-myocyte conduction leading to broadening of QRS complex.



A young school student becomes unconscious during school assembly and is brought to ER. Father tells that he has recurrent episodes of palpitations and syncopal episodes. His elder brother had died suddenly while working in the field in the village.



- 1. Delta waves
- 2. Short PR interval
- 3. Normal PJ interval
- 4. Absent Q waves

#### Treatment:

TOC: Radiofrequency ablation

DOC: Flecainide

DOC for acute episodes: IV Procainamide

#### ECG RHYTHM DISORDERS

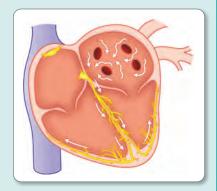
#### MUST KNOW Atrial Fibrillation

The ECG shows absent P waves with irregular RR interval which is diagnostic of **Atrial fibrillation**.

#### Causes:

- 1. Dilated cardiomyopathy due to long-term alcohol use
- 2. Hupertension
- 3. Rheumatic heart disease with mitral stenosis
- 4. Age >65 years

Pathophysiology: Multiple ectopic foci at interface of left atrium with pulmonary veins. The vagal innervation at AV node prevents these impulses to travel to ventricle at same frequency. However, if these patients have AV bypass tract-like bundle of Kent which has no decremental property, then impulses can travel down to ventricles without a hindrance and Atrial fibrillation can degenerate to ventricular fibrillation. The only situation when atrial fibrillation can degenerate into ventricular fibrillation is due to presence of bundle of Kent.



2

#### Neurology

#### EEG WAVES

Normal EEG waves in awake state

Beta waves (eye open): 13-30 Hz Alpha waves (eyes closed): 8-13 Hz

EEG in sleep

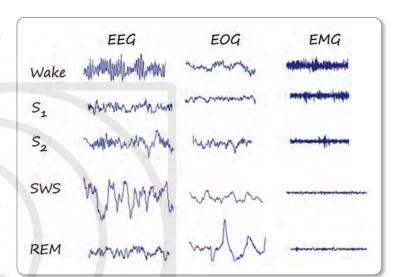
NREM1: Theta waves: 4-7 Hz

NERM2: Sleep spindles and K complexes

NREM3: Delta waves: 0.5-4 Hz

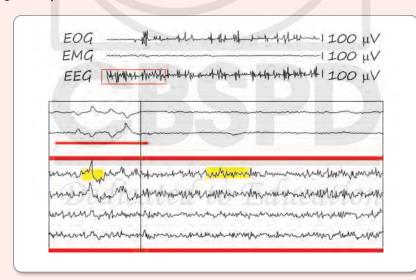
**REM**: Sawtooth waves (Always check corresponding EMG and note that it is always touching the baseline while EOG is having largest spikes in entire

sequence).



#### Case Scenario 17

Which of the following EEG pattern is shown as follows?



a. REM

b. NREM 1

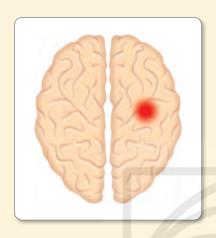
c. NREM 2

d. NREM 3

Ans. The answer is option a.

Notice the Sawtooth pattern along with EMG almost touching the baseline. This is seen in REM sleep.

#### Focal seizures

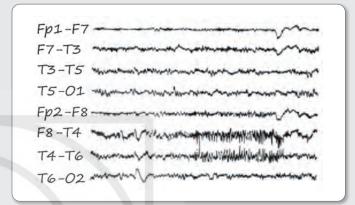


Febrile seizures (Episode management) Febrile seizures (Prevention of episode) West syndrome/Salaam seizures EEG shows hypsarrhythmia and electrodecremental effect.

Preferred antiepileptic drug in pregnancy

Mnemonic: LCOP

Lamotrigine, Carbamazepine, Oxcarbazepine, Phenytoin



Only temporal leads are showing abnormal discharge in focal seizures Intranasal midazolam > rectal diazepam Oral Clobazam > oral rectal diazepam Injection ACTH

Levetiracetam and lamotrigine

#### One-Liners

- Best for medically refractory epilepsy: Surgical resection of epileptogenic focus.
- Keto diet works in genetic epilepsy syndrome due to GLUT 1 deficiency due to SLC2A gene defect.
- · Epilepsy drug leading to weight loss is Topiramate. Topiramate should not be used in patients at risk of glaucoma and kidney stones.
- Epilepsy drug leading to hyperammonemia is valproate.
- · Valproate is the most teratogenic drug with incidence of NTD reaching up to 20%. It is more teratogenic than lithium that causes Ebstein anomaly.
- Most severe neural tube defect is craniorachischisis. Most common NTD is spina bifida occulta. MC NTD in infant of diabetic mother is also spina bifida occulta. Sacral agenesis is most specific NTD seen in infant of diabetic mother.

#### MUST KNOW

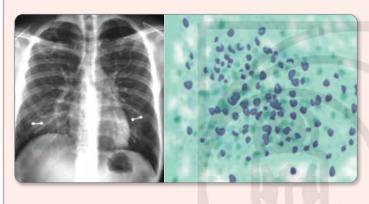
#### When to stop epilepsy medication?

It is reasonable to taper and stop AED after 2 years when all of the following criteria are satisfied.

- Complete medical control of seizures in last 1-5 years
- Single type of seizures
- Normal CNS examination and normal intelligence
- Normal EEG
- No family history

#### Case Scenario 24

An AIDS positive truck driver presents with complaints of fever with shortness of breath for the past 1 week. He is taking amoxicillin but shows no improvement. On auscultation, scattered crepitations are noted bilaterally in bilateral lung fields. The work up done is shown. Which of the following is the most likely cause?



Ans. Diagnosis: Pneumocystis jiroveci Pneumonia (PJP)

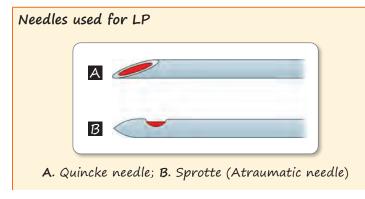
- Pointers are CXR showing bilateral infiltrates, Gomori-Methenamine stain (green background) showing black fungi in cyst form and immunocompromised status.
- · Treatment must be started with cotrimoxazole.
- CXR in PJP shows bilateral diffuse interstitial infiltrates that are perihilar and symmetric. CT chest can show ground glass opacities even before CXR becomes abnormal. Atypical features can be upper lobe infiltrates, mediastinal adenopathy, nodules and cavity formation.
- Cotrimoxazole is used for primary and secondary prophylaxis in AIDS patients when CD4 count >200 cells/mm3 for 3 months.
- Skin manifestation in cryptococcosis resembles molluscum contagiosum.

#### Case Scenario 25

A 30-year-old patient presents with high-grade fever and eccentric behavior for past 2 days. He/she currently does not recognize family members and is making howling noises. Patient is not cooperative for examining meningeal signs. MRI brain shows temporal lobe involvement. What will be the diagnosis?

Ans. Altered mentation and MRI showing temporal lobe involvement points to Herpes simplex 1 encephalitis. Empirical acyclovir should be started in case of pending PCR CSF HSV-1 report. CSF can be bloody. EEG will show periodic lateralized epileptiform discharge.

## LUMBAR PUNCTURE (LP)





#### Endocrinology

#### DIABETES MELLITUS

Diabetes mellitus	Normal	Prediabetes	Diabetes mellitus
Fasting plasma glucose	<100	100-125 Impaired fasting glucose	126 or higher
2 hours value after 75 g glucose intake	<140	140-199 Impaired glucose tolerance	200 or higher

All values are in mg/dL, Postprandial value is not used for diagnosis but for monitoring and follow-up.

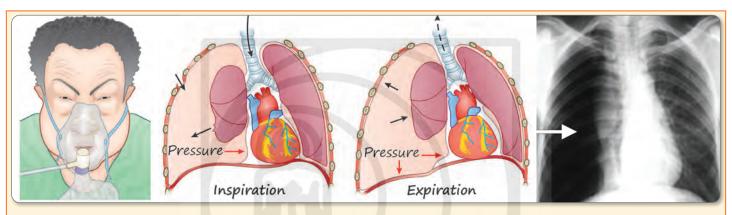
#### One-Liners

#### Must Know One-Liners for Diabetes Mellitus

- Best test for confirming diabetic ketoacidosis (DKA): Plasma beta hydroxybutyrate levels
- Triad of DKA: Hyperglycemia with ketonemia and high anion gap metabolic acidosis
- · Best test for severity of diabetes mellitus: Glycosylated hemoglobin
- · Best test for monitoring long-term control of diabetes mellitus: Glycosylated hemoglobin
- Good control is defined as HbA1c values <7% (take cut off as <8% for elderly and those prone to hypoglycemia)
- Best test for monitoring short-term control of diabetes mellitus: Glycated albumin/
   S. fructosamine
- Best to reduce microvascular complications of diabetes: Strict HbA1c control with values <7%</li>
- Best to reduce macrovascular complications of diabetes: Strict BP control
- · MC overall long-term complication of diabetes mellitus: Diabetic neuropathy
- MC cause of sudden death in diabetes: Silent MI
- Best drug to reduce CV Mortality in diabetes is GLP-1RA, like semaglutide and/or SGLT2i, like empagliflozin.
- Pioglitazone and rosiglitazone are contraindicated in DM with CHF. However, they show benefits in DM with secondary prevention of stroke.
- Diabetic retinopathy and nephropathy progress at the same speed:
  - Type 1 DM: takes 5 years
  - Type 2 DM: takes 15-20 years
- Drugs to slow down the progression of diabetic nephropathy:
  - Low dose ACEi
  - SGLT2i like empagliflozin
  - Finerenone: Selective mineralocorticoid receptor antagonist
- Diabetics have higher incidence of developing vasculopathy leading to:
  - Peripheral artery disease
  - Coronary artery disease
  - Blindness due to vitreous hemorrhage and retinal detachment due to neovascularization.

#### Pulmonology and Spirometry

#### **PNEUMOTHORAX**



- Notice the subcutaneous emphysema leading to puffiness of face and eyes.
- The build-up of air at positive pressure in chest leads to displacement of heart and compression of SVC/IVC. This causes venous return to fall to zero and crashing of SBP causing obstructive shock.
- CXR shows absent vascular markings with hypertranslucent lung and visible collapsed lung tissue.

Keywords in MCQ: Absent air entry and absent/distant breath sounds

When to say tension pneumothorax: Crashing of BP/Hemodynamic instability. Leading cause is ventilator induced barotrauma.

#### First line intervention for tension pneumothorax:

- 1. Wide bore needle in 5th intercostal space in midaxillary line (ATLS guidelines)
- 2. This is followed by tube Thoracostomy/Intercostal drainage tube inserted in triangle of safety anterior to midaxillary line and connected to underwater seal chest drainage system.

#### Traditional water seal drainage system

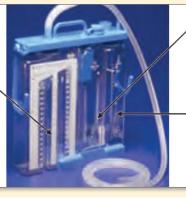
#### Contains three chambers:

- 1. Collection chamber
- 2. Water seal chamber
- 3. Wet suction control chamber

Additional suction source can be added as needed. Intermittent bubbling indicates proper functioning.



Chamber 1
Collects
pleural fluid
drained from
patient



#### Chamber 2

Under water seal—fill with sterile water to the line. Check this chamber for bubbling. If present, it implies ongoing pleural air leak

#### Chamber 3

Suction chamber—fill with sterile water to desired level of suction. Connected to wall suction. This chamber will bubble

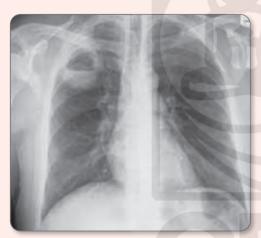
#### Extra Mile

#### Miliary opacities on CXR

- TB/VZ pneumonia
- Pulmonary hemosiderosis
- Silicosis/Pneumoconiosis
- Hypersensitivity pneumonitis
- Pulmonary Alveolar Proteinosis
- Metastasis: Thyroid/Renal/Breast

#### Case Scenario 53

A patient presents with fever of 102° F with cough, foul-smelling sputum and digital clubbing. CXR is given. What is the most likely diagnosis?



- Community-acquired pneumonia
- Pneumatocele
- Empyema
- d. Lung abscess

Ans. The answer is option d.

Fast onset clubbing occurs in lung abscess and empyema. CXR shows thick-walled cavity with air fluid level in left upper lobe indicating lung abscess. Empyema is ruled out as it causes blunting of costophrenic angles.

#### Case Scenario 54

A patient has pleural fluid with an LDH level greater than 0.4 times the serum LDH and protein greater than 0.5 times the serum protein. What is the most likely diagnosis?

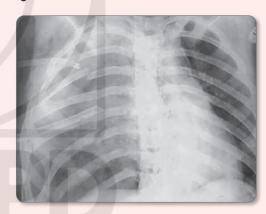
- a. Tuberculosis
- b. Heart failure
- c. Hepatic failure
- d. Renal failure

Ans. The answer is option A.

The question highlights Light's criteria that are used to differentiate between transudate and exudate. Protein value of >0.5 satisfies exudate. Options b, c and d are transudative in nature and only TB is exudate.

#### Case Scenario 55

What is the expected finding in a patient with the following CXR?



- Kussmaul breathing
- b. Paradoxical breathing
- c. Apneustic breathing
- d. Bronchial breathing

Ans. The answer is option b.

CXR shows flail chest that will cause the fractured rib segment to be sucked inside on deep inspiration. This is called paradoxical breathing. Option a is seen in DKA. Option c is seen in damage to pneumotaxic center in brain stem. Option d is seen in lobar pneumonia.

#### Nephrology and Electrolytes

#### IMPORTANT FACTS

#### One-Liners

- Estimated glomerular filtration rate (eGFR) between 30 and 44 mL/min. is stage 3B of grading of CKD.
- AKI stage 1 is rise of Serum creatinine by >0.3 mg/dL or 1.5-1.9 times above index value with urine output <0.5 mL/kg/hr for >6 hours.
- Rise of serum creatinine by >10-20% indicates rejection of allografted kidney.
- Renal tubular acidosis with hyperkalemia is RTA type 4.
- Renal tubular acidosis with hypokalemia and urine pH >5.5 is RTA 1. Rationale is inability to acidify
  the urine due to defect of alpha-intercalated cells. Clinical features given in question will be a child
  with polydipsia, polyuria, muscle weakness, nephrolithiasis, nephrocalcinosis, growth retardation or
  failure to thrive, rickets.
- Renal tubular acidosis with hypokalemia and urine pH <5.5 is RTA 2 (also check bicarbonate value in ABG report and it will be <15 mEq/L due to loss via tubules).</li>
- All types of renal tubular acidosis type 1, 2, 4 cause normal anion gap metabolic acidosis.
- MC opportunistic infection to cause graft failure after kidney transplantation is CMV. Urine m/e shows owl eye basophilic inclusions in shed renal tubular cells.
- MC opportunistic infection to cause graft failure >6 months after kidney transplantation is BK virus/ Polyomavirus. Urine m/e shows decoy cells in shed renal tubular cells.
- MCC of glomerulonephritis is IgA nephropathy/Berger disease.
- · MCC of hematuria is kidney stones.
- Hematuria is defined as >5 RBC/HPF on three consecutive specimens or >100 RBC/HPF in single report.
- Dysmorphic RBC in urine m/e indicates glomerular bleeding.
- Eosinophils in urine m/e: Three must-know scenarios are as follows:
  - 1. Drug exposure like vancomycin along with fever, rash, arthralgia: Allergic interstitial nephritis.
  - 2. Postangiography eosinophils in urine plus livedo reticularis, digital infarcts: Atheroembolic disease.
  - 3. Parasitic infestation of urinary bladder: Schistosomiasis.
- Aggressive hemodialysis can result in the development of altered mentation and seizures in patients and is called dialysis disequilibrium syndrome. It is managed with mannitol that will transiently raise plasma osmolality and counterbalance the excessively low serum osmolality caused by hemodialysis.
- First-line intervention for Aspirin poisoning—Urine alkalization.
- Leading complication of recurrent hemodialysis—accelerated atherosclerosis.
- Post diarrhea, Acute kidney injury (AKI) in child with unilaterally enlarged kidney—Renal vein thrombosis.
- Diarrhea followed by acute kidney injury with P. Smear showing microangiopathic hemolytic anemia/ Schistocytes—Hemolytic uremic syndrome.
- Stroke features with microangiopathic hemolytic anemia and acute kidney injury are seen in TTP.
- Thrombosis is **not** a feature of TTP. The term TTP is misnomer and gives impression of causing thrombosis in large vessels. Rather platelets are consumed in this condition.

#### Hepatology

#### IMPORTANT FACTS

#### One-Liners

- A 3-year-old boy with neurodegenerative findings and poor visual acuity is having low serum copper and serum ceruloplasmin levels. The elder brother died of the same medical issues. Diagnosis is Menkes disease. It occurs due to defect on ATP7A gene on chromosome X. Parenteral copperhistidine is given in the first 4 weeks of life. Wilson disease is discussed at end of this chapter.
- MCC of liver transplantation is NAFLD > Alcohol.
- MC viral cause of End stage liver disease requiring orthoptic liver transplantation is hepatitis C.
- Portal hypertension is defined as hepatic venous pressure gradient >5 mm Hg.
- SGPT is the most specific liver function test.
- SGOT/SGPT ratio >1 is for severity of alcoholic hepatitis.
- Drug of choice for alcoholic hepatitis is prednisone.
- GGT is located in endoplasmic reticulum and bile duct epithelial cells is marker for chronic alcoholism.
- All clotting factors are produced by the liver; except, factor 8.
- Factor VIII is synthesized by hepatic endothelial sinusoidal cells.
- Alkaline phosphatase and 5'nucleotidase are found near the bile canalicular membrane of hepatocytes and gross elevations are seen in obstructive jaundice.
- Basal ganglia involved in Wilson disease is Lenticular nucleus.
- MRI head finding of Wilson disease is Giant face of panda appearance.
- Main defect in Wilson disease is Defective hepatobiliary excretion of copper.
- Protein involved in Wilson disease is P-type ATPase due to defect in ATP7B gene on chromosome 13.
   Pattern of inheritance is autosomal recessive.

#### IMPORTANT SCORING PATTERNS IN LIVER DISEASE

Score	Done for alla to Laucath	Parameters included
MELD score	Predicts short-term mortality in patients with cirrhosis and priority allocation for liver transplantation	Creatinine, Bilirubin, International normalized ratio Mnemonic: CBI
PELD score	Pediatric Liver transplantation	Bilirubin, INR, albumin, age, nutritional status
Nazer index	Liver transplant in Wilson disease	Bilirubin, INR, SGOT
Maddrey's discriminant score	Guide to treatment in alcoholic hepatitis	Bilirubin, INR
Child-Pugh classification	Grading of cirrhosis for survival rate determination and intervention	Bilirubin, albumin, ascites, INR, Asterixis

NAFLD fibrosis score, FIB-4 are the most commonly used noninvasive tests to assess hepatic fibrosis. Parameters are age, BMI, glucose platelet count, Albumin, AST and ACT.

MELD-Na scoring system incorporates serum sodium to increase accuracy.

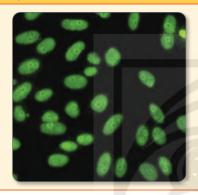
MELD 3.0 includes albumin apart from Bilirubin, INR, creatinine and sodium.

DELTA-MELD: Difference between current MELD and lowest MELD measured within 30 days, prior to current MELD.

#### Investigations

- ESR normal/elevated indicates myositis or malignancy
- ANA positive (Most common antibody positive)
- IOC: Anti-topoisomerase-1 antibody, anti-Scl-70 antibody and anti-RNA polymerase III.

#### Fine speckled pattern in Systemic sclerosis



#### Centromere pattern in CREST Syndrome



#### **Treatment**

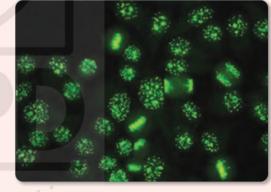
Steroids can worsen scleroderma crisis. For ILD, cyclophosphamide is used. No therapy can alter the course of disease.

#### Case Scenario 65

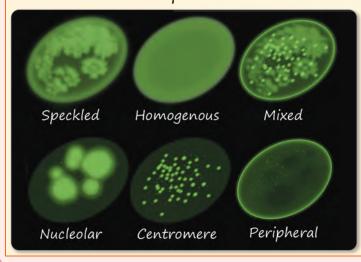
A young lady comes with complaints of difficulty in swallowing food, skin tightening of fingers and finger-tip discoloration on putting hands in cold water. Face shows telangiectasias. Work up shows ANA positive and following IF pattern is present. What will be the most likely diagnosis?

- SLE—peripheral pattern
- b. Systemic sclerosis—speckled pattern
- Systemic sclerosis—centromere pattern
- d. Sjögren syndrome—nucleolar pattern

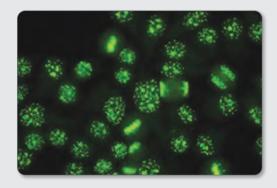
Ans. The answer is option c.



#### Schematic of various IF patterns



The clinical features are diagnostic of CREST syndrome which is diagnosed by anticentromere antibodies. The pattern in image shown is centromere pattern.



9

#### Gastroenterology

#### IMPORTANT FACTS

#### One-Liners

- Leading cause of upper GI bleeding is Peptic ulcer bleeding. In DU source of bleed is gastroduodenal artery, while in GU, source of bleeding is Left gastric artery.
- Leading cause of Lower GI bleeding causing severe hypotension is peptic ulcer bleed. First investigation in this case is upper GI endoscopy. Do **not** answer colonoscopy just because it is written lower GI bleed. It was a large duodenal ulcer that bled profusely.
- Leading cause of lower GI bleeding not requiring hospitalization is Hemorrhoids.
- · Leading cause of melena is peptic ulcer disease.
- Feline esophagus is seen in Eosinophilic esophagitis.
- Truelove and Witts criteria are used for assessment of severity of ulcerative colitis.
- Janus kinase (JAK) inhibitor used in severe ulcerative colitis is Tofacitinib.
- Marsh classification is used for histological grading of celiac sprue.
- Vienna classification and Montreal classification are used for evaluating extent of Crohn disease.
- Demeester score is used to grade severity of Gastroesophageal reflux disease.

#### DISEASES OF ESPOPHAGUS



#### Achalasia Cardia

- · Leading cause is autoimmunity.
- Presents with aperistalsis and increased tone of LES.
- Young female presents with complaints of halitosis, regurgitation of food items eaten last night or previous meal and dysphagia for liquids > solids.
- History of recurrent pneumonia episodes may be present.
- IOC is high resolution esophageal manometry
- Endoscopy shows retained saliva with a puckered gastroesophageal junction on endoscopy
- · Barium swallow shows Bird beak defect

#### Treatment approaches

- Laparoscopic Heller myotomy, preferably with anterior (Dor) being more commonly performed as compared to (Toupet)posterior partial fundoplication
- Pneumatic dilatation
- Intrasphincteric Botulinum toxin injections
- Medical therapy using CCB and nitrates

10

#### Hematology

#### IMPORTANT FACTS

#### One-Liners

- Leading cause of death after blood transfusion is Transfusion-Associated Circulatory Overload (TACO)
- Leading complication of blood transfusion is febrile nonhemolytic transfusion reaction.
- Massive blood transfusion can lead to metabolic alkalosis due to excess citrate entry into recipient.
- Hyperkalemia after blood transfusion occurs usually due to near expiry date blood as sodium potassium pumps shut down.
- Hypokalemia after blood transfusion can appear due to metabolic alkalosis. pH and potassium have inverse relation. Plus, release of catecholamines due to stress can cause potassium to migrate inside the cells and lead to hypokalemia.
- Deleukocytation (WBC depletion) implies <1-5.10° leukocytes per unit. Rationale behind WBC depletion is reduction of all these events:
  - Post-transfusion fever and chills
  - Transfusion of intracellular pathogens including CMV
  - Alloimmunization

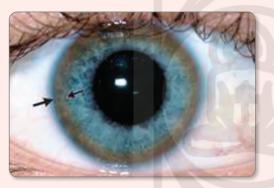
#### HOW TO IDENTIFY VARIOUS TRANSFUSION REACTIONS?

Transfusion adverse reactions: Main warning signs			
Fever (≥38°C)	+1°-2°C within 4 hours +1°-2°C within 15 minutes+/-: • Chills • Dyspnea • Hemolysis • DIC		
>2°C or ≥39°C Hypotension (SBP Falls >30 mm Hg) after BT is started		<ul> <li>Transfusion-transmitted bacterial infection</li> <li>Hemolytic shock</li> <li>Anaphylactic shock</li> <li>Septic shock</li> <li>TRALI</li> </ul>	
Dyspnea		<ul> <li>TRALI (within 6 hours of transfusion): BP normal, BNP normal/elevated</li> <li>TACO (within 6 hours of transfusion): BP elevated, elevated JVP, Gross elevation of BNP</li> </ul>	

#### Miscellaneous Case Scenarios

#### Case Scenario 98

A 15-year-old child with dystonia, dysarthria and poor school performance is found to have the following finding on ophthalmological evaluation. Which of the following is correct about this presentation?



- a. Defect in P type ATPase protein that transports copper into bile
- b. Defect in P type ATPase protein that transports copper to basal ganglia
- c. Defect in P type ATPase that causes excess transports copper across cell membranes
- d. Defect in P type ATPase that causes inhibition of copper across cell membranes

Ans. The answer is option a.

#### Wilson

- ATP 7B protein defect due to defect in chromosome 13
- Copper transporting ATPase is located in the trans-Golgi network of the liver and brain and balances the copper level in the body by excreting excess copper into bile and plasma

- ATP 7A protein defect
- Coppertransporting P-type ATPase which uses the energy arising from ATP hydrolysis to transport copper across cell membranes.

#### Case Scenario 99

A 20-year-old guy doing regular gym workouts and eating good diet presents with severe sensory neuropathy with dysesthesia in glove and stocking pattern and ataxia. He eats both veg and non-veg items with some unknown supplements. Which of the following is likely to be responsible for this presentation?

a. B,

b. B.

c. B<sub>a</sub>

d. B,

Ans. The answer is option b.

The patient is having features of vitamin B toxicity due to his supplements leading to sensory neuropathy with dysesthesias and sensory ataxia. NCS reveals reduced sensory nerve action potentials.

#### Case Scenario 100

A 30-year-old man is having foamy urine for past few days. On work up, massive proteinuria is found and kidney biopsy was done as serum creatinine was rising progressively. Electron extensive microscopy shows foot process obliteration, mesangial sclerosis with increased matrix and collapsed glomerular loops. What should be the diagnosis?

- a. Membranous glomerulopathy
- b. Focal segmental glomerulosclerosis
- c. Minimal change disease
- d. Postinfectious glomerulonephritis

to diagnosis of FSGS.

Ans. The answer is option b.

Keywords are foot process obliteration, mesangial sclerosis with increased matrix and collapsed glomerular loops in an adult patient which point



# LATEST QUESTION PAPERS

- → NEET PG 2024 (SHIFT 1) (MEMORY-BASED)
- → NEET PG 2024 (SHIFT 2) (MEMORY-BASED)
- → NEET PG 2023 (MEMORY-BASED)
- → NEET PG 2022 (MEMORY-BASED)
- → NEET PG 2021 (MEMORY-BASED)
- → INI-CET NOVEMBER 2024 (MEMORY-BASED)
- → INI-CET MAY 2024 (MEMORY-BASED)
- → INI-CET NOVEMBER 2023 (MEMORY-BASED)
- → INI-CET MAY 2023 (MEMORY-BASED)

- → INI-CET NOVEMBER 2022 (MEMORY-BASED)
- → INI-CET MAY 2022 (MEMORY-BASED)
- → INI-CET NOVEMBER 2021 (MEMORY-BASED)
- → INI-CET JULY 2021 (MEMORY-BASED)
- → FMGE JULY 2024 (MEMORY-BASED)
- → FMGE JANUARY 2024 (MEMORY-BASED)
- → FMGE JULY 2023 (MEMORY-BASED)
- → FMGE JANUARY 2023 (MEMORY-BASED)
- → FMGE JUNE 2022 (MEMORY-BASED)
- → FMGE DECEMBER 2021 (MEMORY-BASED)

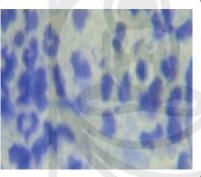
- b. Lamivudine plus tenofovir for 48 weeks
- c. Pegylated interferon with tenofovir for 6 months
- d. Pegylated interferon and Lamivudine for 48 weeks

Ans. a. Tenofovir for 48 weeks

#### NEET PG 2022 (Memory-Based)

90. Gross CSF specimen of patient is shown below along with a smear report. Biochemical evaluation shows moderately low sugar with elevated protein values. Which of the following is the likely etiology?





- a. Pyogenic meningitis
- b. Aseptic meningitis
- c. Viral meningitis
- d. Tubercular meningitis

Ans. d. Tubercular meningitis

91. A known case of C.L.D with Ascites presents with abdominal pain and tenderness. Ascitic tap shows presence of 600 PMN cell/cu.mm. Which of the following is the likely etiology?

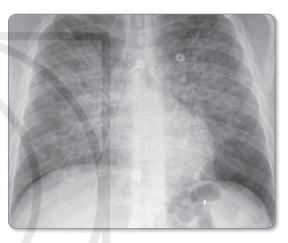


- a. Tubercular ascites
- b. Malignant ascites

- c. Spontaneous Bacterial peritonitis
- d. Chylous ascites

Ans. c. Spontaneous Bacterial peritonitis

92. An 82-year-old patient with hypertension comes with acute onset of breathlessness. Chest X-ray is shown below. What should be given next?



- a. Oxygen inhalation + Antibiotics
- b. IV salbutamol
- c. IV nitroglycerine
- d. Nebulized salbutamol

Ans. c. IV nitroglycerine

- 93. A 25-year-old female patient complains of palpitations and examination shows irregularly irregular pulse, pulse deficit = 20/min.3 and BP = 110/70 mm Hg. Which of the following will be found in this patient?
  - a. Absent a wave
  - b. Canon a wave
  - c. Absent y descent
  - d. Prominent V wave

Ans. a. Absent a wave

- 94. Patient presents after accidental consumption of eight tablets of digoxin each of 0.25 mg. Pulse rate is 40/min. with ECG evidence of 3rd degree heart block. What is the best treatment?
  - a. Digoxin immune fab
  - b. Lidocaine
  - c. DC shock
  - d. Phenytoin

Ans. a. Digoxin immune fab

113. An obese woman came to OPD with dark patches on the neck. What could be the diagnosis?



- a. Metabolic syndrome
- b. Hypothyroidism
- c. Obesity
- d. Addison disease

Ans. a. Metabolic syndrome

#### 114. Which Fluid is shown in the image below?

Sterile, nonpyrogenic, single dose container 500 mL Each 100 mL contains:

Sodium lactate solution USP

0.320 9 Eq. to sodium lactate Sodium chloride IP 0.600 g 0.040 g Potassium chloride IP Calcium chloride IP 0.027 9 Water for injection IP mmol/L: Na+ 131, K+ 5, Ca++ 2. Bicarbonate (as lactate) 29, Cl- 111. mOsmol/L: 278

Dosage: As directed by the Physician

Store at a temperature not exceeding 30°C. To be used with nonpyrogenic IV. administration set with aseptic techniques.

a. RL

b. NS

c. Isolyte-P

d. Isolyte-M

Ans. a. RL

- 115. A patient who suffered from stroke previously has now presented with wild flinging, large amplitude movements affecting proximal limb muscles. Comment on site of lesion.
  - a. Subthalamic nucleus
  - b. Globus pallidus
  - c. Putamen
  - d. Substantia nigra

Ans. a. Subthalamic nucleus

- 116. A 30-year-old patient presents with fever and severe calf pain. On examination conjunctival suffusion is seen with petechial eruption. Per abdomen shows tender hepatomegaly and icterus. KFT is normal. Diagnosis is:
  - a. Leptospirosis
  - b. Viral hepatitis
  - c. Dengue hemorrhagic fever
  - d. Chikungunya

Ans. a. Leptospirosis

#### NEET PG 2021 (Memory-Based)

- 117. A 56-year-old patient has been admitted with complaints of recurrent episodes of retrosternal chest pain with each episode lasting 3-5 minutes, which is relieved on sublingual nitrate. TROP I is normal. ECG shows features of LVH with T wave flattening. He is already on statins, aspirin, metformin, and metoprolol 50 mg. What is the next best step?
  - a. Increase B-blocker dose
  - b. LMWH subcutaneous
  - c. IV NTG drip
  - d. Add clopidogrel

Ans. b. LMWH subcutaneous

- 118. A patient with ECG changes of MI underwent PCI with Stenting. What will you add on to the list along with aspirin?
  - a. PDE-5 inhibitor
  - b. DOAC
  - c. P,Y, inhibitor
  - d. PDE-3 inhibitor

Ans. c. P<sub>2</sub>Y<sub>12</sub> inhibitor

- 119. A patient presents with breathlessness, easy fatigue, and peripheral edema. On general physical examination, large a wave in JVP is seen, and auscultation reveals a mid-diastolic murmur. What is the probable diagnosis?
  - a. TS
  - b. MS
  - c. MR
  - d. TR

Ans. a. TS

#### 151. Which of the following is not correct about management of Post-CPR Return of spontaneous circulation?

- a. Targeted temperature management at 32°-36°C if patient is not cooperative
- b. MAP >65 mm Ha
- c. SpO<sub>2</sub> 92-98%
- d. Emergent cardiac intervention if STEMI present

Ans. a. Targeted temperature management at 32°-36°C if patient is not cooperative

Explanation: Targeted temperature management is done in cases of comatose patient who has sustained CNS damage as therapeutic hypothermia is neuroprotective. It is not done if patient is awake and noncooperative. Options b, c and d are correct as per AHA guidelines. Advanced cardiovascular life support Initial stabilization phase ROSC obtained Manage respiratory parameters: Titrate FiO2 for SpO2 92-98%; Manage airway start at 10 breaths/min; Early placement of endotracheal tube titrate to PaCO, of 35-45 mm Hg Initial Manage respiratory parameters 2. Manage hemodynamic stabilization Start 10 breaths/min parameters: Administer phase SpO, 92-98% crystalloid and/or PaCO2 35-45 mm Hg vasopressor or inotrope for goal systolic blood pressure >90 mm Hg or Manage hemodynamic parameters mean arterial pressure Systolic blood pressure >90 mm Hg >65 mm Hg Mean arterial pressure >65 mm Hg Continued management and additional emergent Obtain 12-lead ECG activities TTM: If patient is not Consider for emergent cardiac intervention if following commands, start · STEMI present TTM as soon as possible; · Unstable cardiogenic shock begin at 32-36°C for · Mechanical circulatory support required 24 hours by using a cooling device with feedback loop Continued management Follows commands? and additional No Yes emergent Comatose Awake activities · TTM Other critical · Obtain brain CT care · EEG monitoring management Other critical care management

#### 152. Which of the following is not a cause of High anion gap metabolic acidosis?

- a. Diarrhea
- b. Acute kidney injury
- c. Ketoacidosis
- d. Inborn errors of metabolism

Explanation:	
Causes of HAGMA	Causes of NAGMA
Mnemonic: KULT	Mnemonic: DR FUSE
Ketoacidosis	Diarrhea
Uremia- AKI, ATN	Renal tubular acidosis
Lactic acidosis	Fistula
Toxins	Ureterosigmoidostomy
Inborn errors of metabolism like organic a, acidemia, pyruvate decarboxylase deficiency, etc.	

#### 153. Which of the following is not included in Thrombotic Thrombocytopenic Purpura?

- a. Thrombosis
- b. Thrombocytopenia
- c. Microangiopathic hemolytic anemia
- d. Renal failure

Ans. a. Thrombosis

Explanation: TTP occurs due to ADAMTS 13 deficiency. ADAMTS 13 lyses large VWF multimers. When it is missing large multimers utilize platelets to form microthrombi that damage RBC and platelets contributing to MAHA and low platelets. Kidney and cerebral microvasculature bear the brunt of disease causing AKI and stroke.

#### 154. Which of the following is correct about resuscitation in children?

- a. Adrenaline 0.01 mg/kg, 1:1000 dilution
- b. CPR@ 30:2 for 2 rescuers
- c. Adenosine is preferred to be given intraosseous
- d. Chest compression should be 1/3rd of AP diameter of chest

Ans. d. Chest compression should be 1/3rd of AP diameter of chest

Explanation: Option a is wrong as dilution used is 1:10,000. Option b is wrong as ratio is 15:2 for 2 rescuers. Option c is wrong as adrenaline is used and route preferred is intravenous. Option d is correct as depth of compression in children should be one-thirds of AP diameter of chest.

#### 155. Which of the following is not a feature of APS-1?

- a. Mucocutaneous candidiasis
- b. Onset in infancy
- c. Type 1 Diabetes mellitus
- d. Hypoparathyroidism

#### Ans. d. Hypoparathyroidism

Explanation: Autoimmune polyendocrine syndrome type 1 has Addison disease, hypoparathyroidism and mucocutaneous candidiasis and presents in early infancy.

#### Mnemonic

#### Mnemonic is CHAI is Tea

- Candidiasis
- Hypoparathyroidism
- · Addison, AIRE gene on chromosome 21, Autosomal recessive
- · Infancy
- Thyroid disorders: Grave and autoimmune thyroiditis

#### 156. Which of the following statements about Behçet's disease is incorrect?

- a. Pathergy test is done under the ISG criteria
- b. Intestinal manifestations resemble those of Crohn's disease
- c. Oral and genital ulcers on dorsal aspect of penis are common features
- d. It is associated with a higher risk of thrombosis

Ans. c. Oral and genital ulcers on dorsal aspect of penis are common features

Explanation: Behçet disease has genital ulcers on scrotum and not on penis. Option a is diagnostic criteria. Pathergy test causes pustule to form after 24-48 hours. Endoscopic findings of Behçet and Crohn's can be similar. Option d can be explained by vasculitis.

157. A patient presents with a history of multiple peptic ulcers located in the jejunum and duodenum. Work up shows gastric pH -2 and elevated fasting serum gastrin levels. What is the most likely diagnosis, and what are the boundaries of the triangle where it commonly occurs?

The application of heat directly to the extremities of patients with chronic severe hypothermia should be avoided because it can induce peripheral vasodilation and precipitate core temperature "after drop", a response characterized by a continual decline in the core temperature.

#### FMGE JULY 2023 (Memory-Based)

- 451. A 33-year-old woman is being evaluated for excessive daytime sleepiness and forgetfulness which have been present for the past 1 year. She has a short neck and her BMI is 43, her BP is 170/98 mm Hg. In an awake state, ABG shows po, 70 mm Hg, pco, 53 mm Hg, HCO, 33 mmol/L. What is the most likely diagnosis?
  - a. Obesity hypoventilation syndrome
  - b. Central sleep apnea
  - c. Narcolepsy
  - d. Obstructive sleep apnea

Ans. a. Obesity hypoventilation syndrome

- 452. All of the following will show hypokalemia; except:
  - a. RTA 1
  - b. RTA 2
  - c. RTA 4
  - d. Ureterosigmoidostomy

#### Ans. c. RTA 4

- 453. A 10-year-old child is brought with a 2-week history of not feeding well and excessive irritability. On examination, neck stiffness is present. His sister has taken treatment for pulmonary TB. In work-up, LP was performed. Which of the following is likely to be found in CSF?
  - a. Lymphocytosis, low sugar and high protein
  - b. Lymphocytosis, high sugar and high protein
  - c. Neutrophils, low sugar and low protein
  - d. Neutrophils, low sugar and high protein

Ans. a. Lymphocytosis, low sugar and high protein

- 454. A 50-year-old patient with lung cancer presents with breathlessness headedness. On lung auscultation bronchial breath sounds are heard on the left inferoscapular region along with focal area of dullness. Diagnosis is:
  - a. Cardiac tamponade
  - b. Pleural effusion
  - c. Constrictive pericarditis
  - d. Pneumothorax

#### Ans. a. Cardiac tamponade

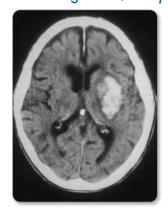
- 455. A 50-year-old patient diagnosed with pneumonia develops increasing breathlessness. Percussion note is stony dull in left infraaxillary and inframammary and infra scapular areas. On lung auscultation, reduced intensity of breath sounds are heard in the same areas mentioned above. Diagnosis is:
  - a. Cardiac tamponade
  - b. Pleural effusion
  - c. Pneumothorax
  - d. Pneumomediastinum

#### Ans. b. Pleural effusion

- 456. ABCD2 scoring includes all of the following parameters; except:
  - a. Age
- b. Hypertension
- c. History of stroke
- d. Diabetes mellitus

#### Ans. c. History of stroke

457. A 60-year-old man presents with sudden onset right-sided hemiparesis for the last 3 hours. BP is 200/100 mm Hg. NCCT head is shown as follows. All of the following are to be done for management; except:





## ONE Touch Medicine



For NEET PG/FMGE/INI-CET/Undergraduates



Theory—A concise form of text covered in just 200 pages. Most important points to remember are given for the last-minute revision. Text of entire book is presented in the form of Tables, Boxes, Flowcharts, and Illustrations for easy recalling.

#### One-Liners

#### Smoking-related ILD:

- Respiratory bronchiolitis-interstitial lung disease (RBILD)
- · Desquamative interstitial pneumonitis (DIP)
- ILD responsive to steroids and seen in young females is nonspecific interstitial pneumonitis.
- ILD that can behave like ARDS is called AIP and Hamman-Rich syndrome.
- Hamman sign is crunching sound heard like someone walking on freshly fallen snow with leather boots and heard in pneumomediastinum.

#### High-Yield Tables and One-Liner Boxes-

Frequently asked topics and clinical correlations are given in bulleted form for ease of learning and more visual impact for long-term memory.

#### Mnemonic

Management of TIA: Mnemonic: ABCD:X

- · Antiplatelet drugs: Aspirin and ticagrelor
- . BP control: ACEI and diuretics
- · Cholesterol control: Atorvastatin
- · Diabetes mellitus control: Pioglitazone
- Apixaban for Nonvalvular atrial fibrillation based on CHA2DS2-VASc score
- VKA for valvular atrial fibrillation and mechanical heart valves.

Mnemonics—Text is supplemented with easy to recall Mnemonic boxes for quick memorization of the concepts.



Clinical Images and Illustrations—Clinical images and other illustrations have been supplemented with the text for better and easy understanding of the concepts.

#### Case Scenario 87

An AIDS-positive truck driver presents with nonneoplastic proliferation of small blood vessels in skin and gingiva. The diagnosis is:

- a. Bacillary angiomatosis
- b. Kaposi sarcoma
- c. Granulocytic sarcoma
- d. Chloroma

Ans. The answer is option a.

The keyword is non-neoplastic proliferation of blood vessels and hence the answer is bacillary angiomatosis. Option b is neoplastic proliferation.

Case Scenario Questions—100+ Case Scenarios added throughout the book in an integration with the respective topics.



#### Last 5 years Exam Questions—Last

5 years' exam question papers up to Nov 2024 (INI-CET Nov 2024 and NEET PG 2024) are provided to develop an idea about the trend of questions and also to know about the recently asked topics.

#### **About the Author**



**Deepak Marwah**, Clinician and Director, Medicine Buster Classes, New Delhi, India, is an alumnus from Maulana Azad Medical College, New Delhi and has been in teaching profession for more than 12 years. His classes are appreciated and accepted by the students in India as well as abroad. He has been invited as a guest lecturer at International Universities on several occasions. His innovative teaching methods and flawless delivery of lecture make him very popular amongst the students. The way he has simplified the complex subject of Medicine is exceptional and has helped the students to score a good rank in the exams.



#### CBS Publishers & Distributors Pvt. Ltd.

4819/XI, Prahlad Street, 24 Ansari Road, Daryaganj, New Delhi 110 002, India **E-mail:** feedback@cbspd.com, **Website:** www.cbspd.com
New Delhi | Bengaluru | Chennai | Kochi | Kolkata | Lucknow | Mumbai
Hyderabad | Jharkhand | Nagpur | Patna | Pune | Uttarakhand

