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 Rigler’s sign, which indicates a perforated viscus. also known as the double wall sign, is seen on an X-ray of the abdomen when the air is present on both sides of the intestine, (luminal and peritoneal side of the bowel wall). Dome sign Air on the top of the liquid (fluid level) pneumatosis coli which are suggestive of ischaemic bowel but not diagnostic of this or perforation.

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Gastroenterology

Endoscopy in patients on antiplatelet or anticoagulant therapy
British Society of Gastroenterology (BSG) and European Society of Gastrointestinal Endoscopy (ESGE) guidelines (2016)

• The risk of endoscopy in patients on anti-thrombotics depends on the risks of procedural haemorrhage versus thrombosis due to discontinuation of therapy. • Where the endoscopic procedure carries a high risk of bleeding and the indication for anticoagulation is low risk for discontinuation then anticoagulation should be discontinued until the INR is <1.5 and restarted post-procedure. Bridging with heparin is not required. Bridging is only recommended if the indication for anticoagulation is high risk - for example, mechanical mitral valve, atrial fibrillation (AF) and prosthetic valve, recent venous thromboembolism (VTE) (less than three months), thrombophilia. Low molecular weight heparin (LMWH) is relatively contraindicated in patients with an estimated glomerular filtration rate (eGFR) less than 30 mls/min, these patients may require admission for unfractioned heparin (UFH) infusion. • Where an endoscopic procedure is associated with a low risk of haemorrhage then the BSG recommends continuation of anticoagulation at the current dosage providing an INR within the last seven days is within the therapeutic range.

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Gastroenterology

Updated 2022

Third edition Notes & Notes

For MRCP By Dr. Yousif Abdallah Hamad Volume 2

Foreword

With the grace of the Almighty Allah, I have introduced the third edition of the popular book, the Notes & Notes for MRCP Part & 2. The MRCP exam requires a wide range of information, particular thinking, and question directed experience. This book is directed mainly at those who need comprehensive revision of the topics which commonly appear in the written MRCP exams. It will be helpful to go through these topics before you start solving the best of the five questions; it is also recommended to go quickly over this book in the last few weeks before the day of your exam. This new edition contains the new published guidelines.

I hope you will find the maximum benefits from this book to get through MRCP written exams.

To practice the best of five questions we advise you to join the best website for MRCP passonexam.com For any enquiry or comment, please do not hesitate to contact me.

“The mind is not a vessel to be filled, but a fire to be kindled.” — Plutarch.

March - 2022 Dr. Yousif Abdallah Hamad
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The 10 Golden Tips for MRCP written exams you will ever need

1. ```
   For MRCP, do not read hard; read smart.
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2. Three to six months is usually enough for preparation.
3. Practice the best of the five questions as much as possible.
4. The few days before the exam date, stop revising questions and concentrate on your MRCP notes and top tips.
5. Remember, you are getting ideas and concepts from the questions.
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6. Time factor in the exam room is the leading killer after poor preparation.
7. Manage your time wisely.
8. Read the end of the question first; if you can answer it without reading the whole scenario, it will save your time for the other tuff question (long scenario, .what is the action of imatinib?)
9. Take care for any single word in the question, e.g. (the initial test, the diagnostic test, the best test, the next step)
10. Practice, practice and practice.

Tarsal tunnel syndrome Physiological changes during pregnancy Physiological changes during exercise ..... Physiological changes associated with age Valsalva manoeuvre ..... Cardiac action potential ..... Pulses ..... Heart sounds .....

Murmurs ..... Syncope ..... Vasovagal syncope (VVS) ..... Vertigo & Dizziness ..... Sudden cardiac death ..... Exercise tolerance tests ..... Cardiac enzymes and protein markers ..... ECG: axis deviation ..... ECG: coronary territories ..... ECG: digoxin / ECG: hypothermia ..... ECG: left bundle branch block ..... Right bundle branch block (RBBB) .....
Trifascicular block ..... ECG: normal variants / ECG: PR interval ECG: ST depression / T wave .....

Biphasic T wave / Q waves / ECG: ST elevation (STE) QT Interval .....
ECG: Junctional escape rhythm ..... Cardiac amyloidosis ..... ECG: Wrong leads ..... Early repolarization variant / ECG: U wave Cardiac catheterisation and oxygen saturation levels Pulmonary capillary wedge pressure ..... Cardiac imaging: non-invasive techniques excluding echocardiography Mitral stenosis (MS) ..... Mitral regurgitation (MR) ..... Mitral valve prolapse (MVP) ..... Aortic dissection ..... Aortic aneurysms ..... Aortic regurgitation (AR) ..... Aortic stenosis (AS) ..... Heyde’s syndrome / Williams syndrome Coarctation of the aorta ..... Bicuspid aortic valve .....
Tricuspid regurgitation / Prosthetic valves Supraventricular tachycardia (SVT) .....

Sinus arrhythmia / Premature ventricular ectopic (PVEs) Arrhythmogenic right ventricular cardiomyopathy(ARVC) Atrial fibrillation (AF) ..... Atrial flutter ..... Multifocal atrial tachycardia (MAT) / Atrial myxoma Heart block / First-degree heart block ..... Second-degree heart block ..... Third degree (complete) heart block ..... Pacemakers .....

Pacemaker syndrome ..... Brugada syndrome .....

Catecholaminergic polymorphic ventricular tachycardia (CPVT) Ventricular tachycardia ..... QT interval ..... Long QT syndrome ..... Torsades de pointes (TdP) ..... Adult advanced life support ..... Peri-arrest arrhythmias ..... Wolff-Parkinson White (WPW) ..... Implantable cardiac defibrillators (ICD) ..... Acute pericarditis ..... Pericardial effusion / Constrictive pericarditis Cardiac tamponade ..... Hypertension ..... Hypertensive emergency ..... Hypertensive urgency ..... Malignant hypertension (Accelerated hypertension) Secondary hypertension ..... Hypokalaemia and hypertension ..... Hypertension in pregnancy ..... Pre-eclampsia/Eclampsia ..... Pulmonary arterial hypertension (PAH) ..... Angina pectoris ..... Coronary artery bypass graft (CABG) ..... Cardiac syndrome X / Acute coronary syndrome Myocardial infarction ..... Heart failure

adults ..... Asymtomatic bacteriuria (ABU) ..... UTI in childhood / Recurrent urinary tract infection (rUTI) Catheter-Associated UTI ..... Urinary tract obstruction in children (posterior urethral valves) Coagulation study ..... Assessment of anaemia ..... Iron metabolism ..... Transferrin / Iron studies ..... Iron deficiency anaemia (IDA) ..... Anemia of Chronic Disease / Hepcidin ..... Thalassaemias

Beta-thalassaemia ..... Aplastic anaemia .....

Pure Red Cell Aplasia (PRCA) / Fanconi's Anaemia Macrocytic anaemia /Vitamin B12 (cobalamin) deficiency Pernicious anaemia ..... Sickle cell disease (SCD) ..... Sideroblastic anaemia ..... Haemolytic anaemias: by site ..... Autoimmune haemolytic anaemia (AIHA) Hereditary spherocytosis ..... Hereditary elliptocytosis (HE) ..... Glucose-6-phosphate dehydrogenase (G6PD) deficiency Paroxysmal nocturnal haemoglobinuria ..... Splenectomy ..... Blood products ..... Blood product transfusion complications ..... Transfusion Related Acute Lung Injury (TRALI) Plasma exchange / Deep vein thrombosis (DVT) Pregnancy: DVT/PE / Post-thrombotic syndrome Venous thromboembolism: prophylaxis in patients admitted to hospital Superficial thrombophlebitis / Thrombophilia: causes Factor V Leiden ..... Protein C deficiency ..... Antithrombin III deficiency ..... Hereditary haemorrhagic telangiectasia (HHT) Idiopathic thrombocytopenic purpura (ITP) Langerhans cell histiocytosis ..... Myelofibrosis / Myelodysplastic syndrome (MDS) Leuco-erythroblastic anaemia / Polycythaemia Myelofibrosis / Disseminated intravascular coagulation (DIC) Thrombocytopenia ..... Thrombocytosis / Essential thrombocytosis (ET): Thrombotic thrombocytopenic purpura (TTP) Von Willebrand›s disease ..... Haemophilia ..... Methemoglobinemia ..... Cyanosis without hypoxia / Heparin ..... Novel oral anticoagulants (NOACs) ..... Dabigatran ..... Warfarin ..... Porphyrias ..... Non-Hodgkin›s lymphoma (NHL) ..... Haematological malignancies: genetics ..... Haematological malignancies: infections .....

Burkitt›s lymphoma .....

Cancer in the UK / Acute lymphoblastic leukaemia (ALL) Chronic lymphocytic leukaemia (CLL) ..... Acute myeloid leukaemia (AML) ..... Acute promyelocytic leukaemia (APML) Chronic myeloid leukaemia (CML) ..... Allogenic bone marrow transplant ..... Hairy cell leukaemia / Paraproteinaemia Multiple myelom ..... Monoclonal gammopathy of undetermined significance (MGUS) Thymoma ..... Tumour lysis syndrome (TLS).Waldenstrom›s macroglobulinaemia ..... ECOG score / Tumour markers ..... Neutropenic sepsis (Febrile neutropenia) ..... Assessment of neutropenia ..... Systemic mastocytosis / Cervical cancer ..... Ovarian tumours / Breast cancer ..... Paget›s disease of the breast ..... Radiotherapy/ Chemotherapy ..... Salivary Gland Tumors / Palliative care prescribing: pain Opioid toxicity in palliative care ..... Palliative care prescribing: nausea and vomiting Palliative care prescribing: hiccups ..... Palliative care prescribing: Constipation ..... Palliative care

Third edition Notes & Notes For physician By Dr. Yousif Abdallah Hamad Neurology Updated 2022

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad Chapter 4

Neurology

CNS anatomy

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad

ocalisation of a brain lesion L The following neurological disorders/features may allow localisation of a brain lesion Lobes lesions Lobe lesion Features Frontal lobes lesions • Difficulties with task sequencing and executive skills • Expressive (Broca's) aphasia: (located on the posterior aspect of the frontal lobe, in the inferior frontal gyrus). • Disinhibition • Perseveration • Anosmia • primitive reflexes (positive grasp, pout and palmomental reflexes) • Inability to generate a list • Changes in personality. Parietal lobes lesions • Sensory inattention (contralateral hemihypesthesia) • Apraxia • Astereognosis (tactile agnosia) • Inferior homonymous quadrantanopia • Neglect • Mild hemiparesis • Parietal ataxia • Acalculia (inability to perform mental arithmetic). • Gerstmann's syndrome (lesion of dominant parietal): Alexia (inability to read), acalculia, finger agnosia and right-left disorientation • unilateral impairment of optokinetic nystagmus: a nystagmus that occurs in response to a rotation movement. It is present normally. Temporal lobes lesions • Wernicke's aphasia: this area 'forms' the speech before 'sending it' to Broca’s area. Lesions result in word substitution, neologisms but speech remains fluent • superior homonymous quadrantanopia • auditory agnosia • prosopagnosia (difficulty recognising faces) • Memory impairment. Occipital lobes lesions • homonymous hemianopia (with macula sparing). may present as Anton syndrome where there is blindness, but the patient is unaware or denies blindness. • cortical blindness • visual agnosia • visual illusions and elementary visual hallucinations. Visual- spatial awareness deficit • Due to the Damage to the right parietal lobe • Patient unable to navigate around locations, specially places that are new to him , but also familiar locations Homonymous quadrantanopias • PITS (Parietal-Inferior, Temporal-Superior)

More specific areas Area Associated conditions Medial thalamus and mammillary bodies of the hypothalamus Wernicke and Korsakoff’s syndrome Subthalamic nucleus of the basal ganglia Hemiballism Striatum (caudate nucleus) of the basal ganglia Huntington chorea Substantia nigra of the basal ganglia Parkinson's disease Amygdala Kluver-Bucy syndrome: • hypersexuality, • hyperorality (insertion of inappropriate objects in the mouth) • hyperphagia, • visual agnosia increased activation to the amygdala is associated with depression Hippocampus pathology Short term memory impairment (for example, Alzheimer's disease). Lateral geniculate nucleus pathology visual field defect. Red nucleus (located in the midbrain). • Tremor, which is present both at rest and during action (for example, multiple sclerosis tremor). • A lesion in this area would cause problems with arm swing and motor co-ordination of the upper limbs, not chorea. Prefrontal cortex damage disinhibition and problems with social interaction and judgement and has been implicated in schizophrenia. Left prefrontal cortex Depression Anterior hypothalamic nucleus • Plays a crucial role in thermoregulation and circadian rhythms • Situated at the inferior border of the paraventricular nucleus

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MRCPUK-part-1-September 2012 exam: (SLE) presents with continuous jerky, irregular movements, which move from one limb to another. Where is the lesion most likely to be? Caudate nucleus Crossed neurological signs (ipsilateral motor and sensory cranial nerve signs and contralateral hemiplegia) localise to the brainstem (midbrain, pons or medulla). • Midbrain (ipsilateral oculomotor nerve palsy , contralateral hemiplegia) • Pons (ipsilateral abducens and facial nerves palsy, contralateral hemiplegia) Stroke and pupils: • Midbrain lesions typically cause fixed, midpoint pupils. • Pontine haemorrhage typically cause bilateral pin point pupils Lesions at the jugular foramen • Nasopharyngeal carcinoma is the commonest cause. • Affected CN →9,10,11 CN IX (Glossopharyngeal nerve) & CN X (Vagus nerve) → palatal weakness and swallowing difficulties , Laryngeal muscle paralysis would result in bovine cough and husky voice. CN XI (Accessory nerve) → shoulder and sternocleidomastoid weakness

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Cerebellar lesions A history of vertigo, nystagmus, Slurred speech, intention tremor and past pointing, as well as ataxia, suggest the cerebellum as the site of injury. Oppenheim's sign is seen when scratching of the inner side of leg leads to extension of the toes. It is a sign of cerebral irritation Chorea is caused by damage to the basal ganglia, in particular the caudate nucleus Cerebellum lesions: Charcot's neurological triad: scanning speech, nystagmus, and intention tremors Cerebellar lesion localization: • Lesions to the vermis results in → truncal ataxia and nystagmus. • Cerebellar lesions cause neurological deficits on the ipsilateral side • Lesions to the cerebellar hemispheres results in → ipsilateral dysmetria, dysdiadochokinesis, ipsilateral limb ataxia and fast-beat nystagmus towards the lesion.

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Neurology Transient ischaemic attack (TIA) Definition • Temporary cerebral ischemia that results in brief neurologic deficits lasting < 24 hours Investigations (NICE guidelines. Last updated: March 2019) • MRI is the first choice , identifies ischemia earlier than CT, determine the territory of ischaemia, and detect alternative pathologies. • Do not offer CT brain unless there is clinical suspicion of an alternative diagnosis. • Duplex ultrasound for carotid stenosis urgent for possible carotid endarterectomy. the most appropriate next step if bruits in the neck are heard upon auscultation. If ultrasound is not available, a CTA or MRA may be used. Treatment • Immediate therapy (after initial assessment) Aspirin 300 mg immediately unless contraindicated To be seen within 24 hours of onset of symptoms for specialist assessment Do not use scoring systems, such as ABCD2, to assess risk of subsequent stroke or to inform urgency of referral. • Secondary prevention (introduced as soon as the diagnosis is confirmed) Clopidogrel 300 mg loading dose followed by 75 mg daily. aspirin + dipyridamole should be given to patients who cannot tolerate clopidogrel High intensity (e.g. atorvastatin 20-80 mg daily) started immediately (as per Royal College guideline 2016) Immediate initiation of statin is not recommended as per (NICE guideline 2019) Carotid endarterectomy: for people with non-disabling stroke or TIA: carotid stenosis of 50 to 99%: referred urgently for carotid endarterectomy medical treatment (BP control, antiplatelets, Statin, lifestyle advice). carotid stenosis of less than 50%: No surgery Medical treatment (BP control, antiplatelets, Statin, lifestyle advice). Control BP: antihypertensive If associated AF →Anticoagulation Top Tips

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Brain imaging for TIA and stroke • MRI brain with diffusion-weighted imaging is the preferred modality in patients with suspected TIA. • Non-contrast cranial CT (gold standard and most important initial imaging in stroke): detects acute hemorrhage but cannot reliably identify early ischemia

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Ischaemic stroke: Overview Definition • Stroke is an acute neurological deficit lasting more than 24 hours due to occlusion or critical stenosis of a cerebral artery. Epidemiology • Ischemic stroke (~85%) Risk factors • older age, hypertension, smoking, diabetes mellitus, dyslipidaemia, atrial fibrillation, sickle cell disease, and history of TIA or stroke. Mechanisms • Thrombotic strokes (most common) Atherosclerosis of the extracranial vessels (carotid atheroma) is the most common cause • Embolic strokes Cardiac emboli e.g. Atrial fibrillation Paradoxical embolisation: For a right-sided thrombus (e.g. DVT) to cause a leftsided embolism (e.g. stroke) it must obviously pass from the right-to-left side of the heart. Causes patent foramen ovale : present in 20% of the population. Transoesophageal echocardiography (TOE) is the investigation of choice for diagnosis atrial septal defect - a much less common cause Assessment • ROSIER score (Recognition Of Stroke In the Emergency Room). It is validated tool recommended by the Royal College of Physicians. useful for medical professionals. Exclude hypoglycaemia first, then assess the following:

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Neurology Assessment Scoring Loss of consciousness or syncope

- 1 point Seizure activity
- 1 point New, acute onset of: • asymmetric facial weakness

- 1 point • asymmetric arm weakness
- 1 point • asymmetric leg weakness
- 1 point • speech disturbance
- 1 point • visual field defect
- 1 point stroke is likely if > 0 Imaging • CT without contrast for acute presentation (the best initial test) → to rule out hemorrhage • MRI : if the CT is negative → Diffusion weighted imaging (DWI) MRI is the most sensitive and specific imaging modality for diagnosing acute ischaemic stroke. • Duplex ultrasound for carotid stenosis Diagnostic evaluation • Glucose: the only lab test which should be done before thrombolysis • ECG : to look for cardiogenic thrombus (Atrial fibrillation) • If an embolic stroke is suspected → Echocardiography • Thrombophilia screening: if patient is < 50 years old and/or has a history of thrombosis Only glucose test and CT head are required before thrombolytic therapy. Do not delay treatment to complete the diagnostic evaluation. Management • Exclude hypoglycaemia because it is a stroke mimic • Admission to a stroke unit → improve the overall prognosis. • presentation within 4.5 hours AND thrombolysis not contraindicated → thrombolysis with iv alteplase • presentation after 4.5 hours OR thrombolysis contraindicated → Supportive care • Deep vein thrombosis prophylaxis with subcutaneous heparin or low molecular weight heparin Pulmonary embolism from DVT is the most common cause of early death in patients with acute stroke. Vitamin D has a role as a neuroprotective agent towards large artery atherosclerosis. Many patients with ischemic stroke have vitamin D deficiency.

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Stroke: Clinical features Affected cerebral vessels and associated features Site of the lesion Associated effects Anterior cerebral artery (ACA) • Contralateral weakness and sensory loss more marked in the lower limbs than in upper limbs • Urinary incontinence • Dysarthria Middle cerebral artery • Contralateral weakness and sensory loss more marked in the upper limbs than in lower limbs • Contralateral homonymous hemianopia • Aphasia if in dominant hemisphere (usually left MCA territory (global aphasia) • Hemineglect if in nondominant hemisphere (usually right MCA territory • Gaze deviates toward the side of infarction Posterior cerebral artery • Contralateral homonymous hemianopia with macular sparing • Visual agnosia • Cortical blindness • Visual hallucinations Weber's syndrome (branches of the posterior cerebral artery that supply the midbrain) Or branches of the basilar artery Ipsilateral CN III palsy Contralateral weakness Posterior inferior cerebellar artery
(PICA)(lateral medullary syndrome, Wallenberg syndrome) lesion to dorsolateral medulla • Ipsilateral: facial pain and temperature loss • Contralateral: limb/torso pain and temperature loss. • Ataxia, nystagmus Anterior inferior cerebellar artery (lateral pontine syndrome) Symptoms are similar to Wallenberg's (see above), but: Ipsilateral: facial paralysis and deafness Retinal/ophthalmic artery Amaurosis fugax Basilar artery 'Locked-in' syndrome