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Chapter 4

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

Neurology

Lateral medullary syndrome (Wallenberg's syndrome) Pathophysiology • Ischemic occlusion of the Posterior Inferior Cerebellar Artery (PICA) lesion to dorsolateral medulla • May caused by dissection or thrombosis of the vertebral artery, which gives rise to the posterior inferior cerebellar artery (PICA) • PICA is the largest branch of the vertebral artery and is the most common territory involved in cerebellar infarction. • The PICA supplies blood to structures of the lateral medulla (vestibular nuclei, spinal cord tracts) and the inferior cerebellar peduncle. Features • Vertigo, nausea and truncal ataxia are the most common presenting features and due to vestibular or cerebellar damage. • Hoarseness (or dysphagia, if present) is fairly specific for lateral medullary syndrome because it points to a lesion of the nucleus ambiguous (cranial nerves IX, X, XI). • Damage to the spinal trigeminal nucleus can result in loss of pain and temperature sensation in the ipsilateral face. • Damage to the lateral spinothalamic tract can result in loss of contralateral pain and temperature sensation below the neck. • Damage to the descending sympathetic fibers that also course through the lateral medulla would result in an ipsilateral Horner syndrome of ptosis, miosis, and anhidrosis. • Ataxia is probably due to infarction of the ipsilateral inferior cerebellar peduncle. • Pyramidal tract findings (weakness) are typically absent in lateral medullary lesions. Ipsilateral Contralateral • Cerebellar signs (Ataxia, dysmetria, dysdiadochokinesia, nystagmus) • Horner syndrome • Loss of pain and temperature in the face (due to 5th CN nucleus damage) • Loss of pain and temperature in the trunk and limbs Diagnosis: MRI is the imaging investigation of choice for posterior fossa lesions. Lateral medullary syndrome: • Caused by → PICA lesion • Characterised by : Ipsilateral cerebellar signs (Ataxia, Nystagmus) & Horner syndrome (ptosis, miosis, and anhidrosis). Contralateral trunk and limbs sensory loss

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Lateral pontine syndrome VS Lateral medullary syndrome Lateral pontine syndrome Lateral medullary syndrome Aetiology Anterior inferior cerebellar artery (AICA) posterior inferior cerebellar artery (PICA) Differences Facial nucleus and facial nerve involvement leads to: • Ipsilateral paralysis of the upper and lower face (lower motor neuron lesion). • Ipsilateral loss of lacrimation

and reduced salivation. • Ipsilateral loss of taste from the anterior two-thirds of the tongue. • Hyperacusis. Nucleus ambiguous involvement leads to: Dysphagia, dysarthria, dysphonia Similarities Ipsilateral Horner’s syndrome. Why? Descending hypothalamic tracts affected. Contralateral loss of pain and temperature. Why? Lateral spinothalamic tract affected. Ipsilateral cerebellar ataxia. Why? Cerebellar peduncles affected. Inferior cerebellar peduncle in medullary and middle cerebellar peduncle in pons. Nausea, nystagmus, vertigo, vomiting. Why? Vestibular nuclei involved. Ipsilateral loss of pain and temperature sensation from the face (facial hemianesthesia). Why? Spinal trigeminal nucleus and tract involved. Ipsilateral hearing loss. Why? Cochlear nuclei and nerve fibers involved.

Pontine syndromes Locked-in syndrome • Pathophysiology The locked-in syndrome is caused by destructive bilateral brainstem lesions affecting the corticospinal, corticopontine, and corticobulbar tracts. The most common cause is ischemic infarction of the ventral pons. • Features Quadriplegia and inability to speak or swallow with retained consciousness and eyes movement

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 Because the supranuclear ocular motor pathways are spared, patients can move their eyes and blink. Ventral pontine syndrome • Millard-Gubler syndrome (MGS), also known as facial abducens hemiplegia syndrome is one of the classical crossed brainstem syndromes characterized by a unilateral lesion of basal portion of the caudal pons involving fascicles of abducens (VI) and the facial (VII) cranial nerve, and the pyramidal tract fibers • Classical Raymond syndrome also has the same components as Millard-Gubler syndrome, but there is ipsilateral sixth nerve palsy along with contralateral facial paresis and hemiplegia. Components of Millard-Gubler syndrome (MGS)

1. Ipsilateral weakness of the eye on abduction (VI nerve)
2. Ipsilateral facial muscle weakness (VII nerve)
3. Contralateral hemiparesis or hemiplegia of upper and lower extremities (pyramidal tract involvement) Inferior medial pontine syndrome • Inferior medial pontine syndrome, also known as Foville syndrome, is one of the brainstem stroke syndromes occurring when there is infarction of the medial inferior aspect of the pons due to occlusion of the paramedian branches of the basilar artery. • Features corticospinal tract: contralateral hemiplegia/hemiparesis medial lemniscus: contralateral loss of proprioception and vibration middle cerebellar peduncle: ipsilateral ataxia facial nerve (CN VII) nucleus: ipsilateral facial weakness abducens nerve (CN VI) nucleus: lateral gaze paralysis and diplopia

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Weber's syndrome Overview • Weber syndrome is a midbrain stroke syndrome that involves the cerebral peduncle and the ipsilateral fascicles of the oculomotor nerve • caused by midbrain infarction as a result of occlusion of the paramedian branches of the posterior cerebral artery or of basilar bifurcation perforating arteries. Features • Ipsilateral III palsy • Contralateral weakness Weber syndrome • ipsilateral III palsy • contralateral weakness

Parinaud syndrome (Dorsal midbrain syndrome)

Pathophysiology • Results from dorsal midbrain lesion • Affected vessel : Branches of the posterior cerebral artery. Often result from compression, e.g., by tumor of the pineal gland →compresses the vertical gaze center at the rostral interstitial nucleus of medial longitudinal fasciculus →vertical gaze palsy Feature • Vertical gaze palsy (inability to move the eyes up). Downward gaze is usually preserved • Eyelid retraction (Collier sign): development of unilateral or bilateral lid retraction due to a midbrain lesion of the superior colliculi and the medial longitudinal fasciculus. • Pupillary light-near dissociation (pseudo-Argyll Robertson pupils) • Convergence-retraction nystagmus

Labyrinthine infarction Anatomy • The blood supply to the inner ear flows through only 1 main blood vessel, the internal auditory artery (or labyrinthine artery), which typically originates from the anterior inferior cerebellar artery. Features • almost always present with acute prolonged vertigo and vestibular dysfunction • sudden-onset unilateral hearing loss. Differential diagnosis • Unlike viral labyrinthine dysfunction, the most common pattern of dysfunction with labyrinthine infarction includes a combined loss of auditory and vestibular function.

Posterior communicating artery aneurysm (PCA) Posterior communicating artery aneurysm will cause →compression of the third nerve, and therefore →isolated ipsilateral painful third nerve palsy • Pupillary involvement (pupil dilation) from compression of the parasympathetic fibres that run on the outside of the third nerve • Other features of a third nerve palsy include ptosis, and a ‘down and out’ eye. • Cerebral aneurysms may be associated with polycystic kidney disease. Features • Pupillary dilatation, Ophthalmoplegia, and Ptosis. • other features suggestive of subarachnoid blood (headache, nuchal rigidity and photophobia). Investigation • Urgent CT angiogram of the cerebral vessels is required for diagnosis. Sudden-onset unilateral hearing loss → consider Labyrinthine infarction typically due to anterior cerebellar artery dissection after minor head trauma

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Neurology • Conventional angiography: the definitive procedure for the detection and characterisation of cerebral aneurysms. • Digital subtraction angiography: may be helpful in identifying an acutely ruptured aneurysm. Differential diagnosis • Features distinguishing PCA from a cavernous sinus thrombosis are absence of sinusitis or a midface infection, which are common, absence of fever or additional cranial nerve abnormalities. The differential diagnoses in a patient presenting with headaches and painful diplopia are: • posterior communicating aneurysm (PCA) • Ophthalmoplegic migraine • Pituitary adenoma • Cavernous sinus thrombosis, or • medical mononeuritis. Ischaemic Stroke management Initial management • Aspirin 300mg orally or rectally should be given as soon as possible if a haemorrhagic stroke has been excluded for the initial two weeks. Reperfusion therapy for acute ischemic stroke • Thrombolysis by Alteplase (recombinant

tissue plasminogen activator or tPA) are commonly used Mechanism of action: Alteplase → bind to fibrin in a thrombus (clot) → convert entrapped plasminogen to plasmin → plasmin breaks up the thrombus → fibrinolysis Criteria : Ischemic stroke with the onset of symptoms <4.5 hours Written consent is not required to administer alteplase Absolute contraindications Ischemic stroke, head trauma or surgery in the previous three months Previous intracranial hemorrhage Intracranial neoplasm Gastrointestinal malignancy or hemorrhage in the previous 21 days Cerebral hemorrhage Persistent High BP (systolic ≥185 mmHg or diastolic ≥110 mmHg) Haematological bleeding disorders Bleeding risk: 1 %. • Mechanical thrombectomy: a device that can remove a clot patients should undergo the procedure within 6 hours of symptom onset. Indications large vessel occlusion (usually in addition to IV thrombolytic therapy) proximal MCA or distal internal carotid artery or basilar artery occlusion. Patients who are ineligible for IV thrombolysis who present within the appropriate time-frame with large vessel occlusion.

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Surgical treatment → decompressive hemicraniectomy • Indications: Patient with middle cerebral artery infarction who meet all of the criteria below:

1. Age ≤60 years
2. Clinical deficits suggestive of infarction in the territory of the middle cerebral artery, with a score on the National Institutes of Health Stroke Scale (NIHSS) of above 15.
3. Decrease in the level of consciousness to give a score of 1 or more on item 1a of the NIHSS.
4. Signs on CT of an infarct of at least 50% of the middle cerebral artery territory For individuals aged up to 60 years who suffer an acute MCA territory ischaemic stroke complicated by massive cerebral oedema, surgical decompression by hemicraniectomy should be offered within 48 hours of stroke onset. Secondary prevention • Clopidogrel is now recommended by NICE if clopidogrel is not tolerated →Aspirin + dipyridamole lifelong • Anticoagulation treatment for other comorbidities (e.g. atrial fibrillation): Should not be started until brain imaging has excluded haemorrhage, usually after 14 days from the onset of an ischaemic stroke. ischaemic stroke + atrial fibrillation →aspirin 300 mg for the first 2 weeks before considering anticoagulation treatment. cerebral infarction in patient with prosthetic valves and who are at significant risk of haemorrhagic transformation →anticoagulation treatment should be stopped for 1 week and aspirin 300 mg substituted. ischaemic stroke and symptomatic proximal DVT or PE →should receive anticoagulation treatment in preference to treatment with aspirin haemorrhagic stroke and symptomatic DVT or PE →prevent further PE using either anticoagulation or a caval filter. Prevention of thromboembolic event in stroke patient: Patients admitted with stroke are very likely to be at high risk of developing a thromboembolic event due to reduced mobility. NICE advises that patients admitted within three days of an acute stroke should have intermittent pneumatic compression considered. This should be provided for 30 days or until the patient is mobile or discharged. • Statin All patients who are diagnosed with stroke or TIA should be commenced on statin therapy irrespective of the cholesterol level. If the patient is unable to tolerate a statin (Stroke guidelines by Royal college of physicians 2016): try

alternative methods to improve the tolerability of a statin such as a reduced dose, alternate day dosing or a lower-intensity statin Do not use fibrates, ezetimibe, bile acid sequestrants, nicotinic acid or omega3 fatty acids for cholesterol-lowering after stroke • Management of hyperglycaemia The European Stroke Initiative guidelines recommend treatment for glucose

> 180 mg/dL (>10 mmol/L)

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Neurology The Joint British Diabetes Society 2012 guidelines recommend a target BM of between 6 and 12 mmol/l for hyperglycaemic patients on NG feed with insulin to be started when BM over 12 mmol/l. The insulin regime of choice is a biphasic insulin with a mixture of intermediate and short acting insulin twice daily • Management of blood pressure in acute stroke If thrombolytic therapy is indicated → BP should be maintained ≤185/110 mmHg, before thrombolytic therapy (Labetalol, Nicardipine or Clevidipine are used as first line) If thrombolytic therapy is not indicated: treat high BP only if the systolic BP >220 mmHg or diastolic BP >120 mmHg cautious lowering of BP by approximately 15% during the first 24 hours. Top Tips Hypertension should not be treated in the initial period following a stroke unless complications develops Stroke thrombolysis - only consider if less than 4.5 hours and haemorrhage excluded

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Haemorrhagic stroke Spontaneous Intracerebral haemorrhage (ICH) Epidemiology • Hemorrhagic stroke (~15%) The putamen is the commonest site for hypertensive intracerebral haemorrhage Risk factors • Hypertension (the most common risk factor), older age , haemophilia, cerebral amyloid angiopathy, anticoagulation, use of illicit sympathomimetic drugs, history of heavy alcohol, and vascular malformations. Classification by location • Lobar ICH commonly due to cerebral amyloid angiopathy (CAA). Amyloid deposition in small-sized to medium-sized cortical perforators may lead to the rupture of these vessels, • Non-lobar ICH Time window for : • Thrombolysis (IV Alteplase): < 4.5 hours • Mechanical thrombectomy: < 24 hours

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 commonly due to long-standing high blood pressure resulting in lipohyalinosis of small perforating arteries of the basal ganglia, thalamus, pons and cerebellum, leading to deep haemorrhages, often with extension into the ventricles. Feature • ICH should be suspected in any patient with severe headache, vomiting, elevated systolic blood pressures or decreased level of consciousness. • Fever is common Sustained fever after ICH is an independent prognostic factor for worse outcome. Pontine haemorrhage commonly presents with reduced GCS, paralysis and bilateral pin point pupils Diagnosis • Non-contrast head CT is highly sensitive and specific Treatment • Stabilisation of airway, breathing and circulation (ABCs) . Intubation for airway protection is indicated in patient with GCS ≤8 or significant respiratory distress. • Intensive

lowering of systolic blood pressure to <140 mm Hg Intravenous calcium channel blockers (eg, nicardipine) and β-blockers (eg, labetalol) are the treatment of choice for early BP reduction, given their short halflife and ease of titration. During acute phase, patients may have resistant HTN due to sympathetic surge. A few weeks later, they may require fewer medications and be at risk of hypotension unless the doses of medications are adjusted promptly • Hyperosmolar therapy (Mannitol or hypertonic saline) Peri-haematoma oedema (PHE) develops within the first few days after ICH and may cause elevated ICP, mass effect, midline shift and brain herniation asymptomatic PHE require no specific treatment except maintaining a normal sodium goal →Observe symptomatic cerebral oedema and elevated ICP →mannitol and hypertonic saline (HTS) are the first-line Mannitol is an osmotic diuretic. It increases water excretion by the kidneys and reduces cerebral oedema and ICP. HTS increases plasma osmolarity and the flow of excess water from cerebral tissue to the blood via the osmotic gradient. hypertonic saline is slightly more effective than mannitol for the treatment of elevated ICP. keep serum sodium level at 140–150 mEq/L for 7–10 days to minimise oedema expansion and mass effect.

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Neurology • Reversal of coagulopathies intracerebral haemorrhage in association with Vitamin K antagonist (warfarin) combination of prothrombin complex concentrates (PCC) and intravenous vitamin K. If not PCC not available → fresh frozen plasma (FFP). Dabigatran Idarucizumab Factor Xa inhibitors (eg, rivaroxaban, apixaban and edoxaban) OR other Direct thrombin inhibitors apart from Dabigatran. Low-molecular-weight heparin Protamine Thrombolytic (eg, recombinant tissue plasminogen activator (rtPA)) • Neurosurgery: Patient with a decreased level of consciousness from intraventricular haemorrhage with hydrocephalus, mass effect or brainstem herniation should receive ventriculostomy. • Venous thromboembolism (VTE) prophylaxis with intermittent pneumatic compression (IPC) devices Top tips Haemorrhage associated with dabigatran →Idarucizumab

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Cerebral venous thrombosis (CVT) Patients with a hypercoagulable state (e.g. pregnancy) and papilloedema with neurological signs should be investigated for cerebral venous thrombosis. Basics • Structure: reflections in dura matter where meningeal and periosteal layers split • Function: return blood from cerebral veins to internal jugular vein • veins contain NO valves Epidemiology • more common in young women. Sex: ♀> ♂, 3:1. Age of onset: ≤ 40 years Types • Superior sagittal sinus thrombosis (SSST) is the most common type of dural venous sinus thrombosis • Cavernous sinus thrombosis (CST) →3rd, 4th and ophthalmic (V1) and maxillary (V2) divisions of the 5th cranial nerve affected Notes & Notes for MRCP
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reversed with Prothrombin complex concentrate (PCC) 1st line →cryoprecipitate administration. 2nd line (If cryoprecipitate is contraindicated or unavailable) →tranexamic acid (antifibrinolytic agent)

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• Superior orbital fissure syndrome: similar to the cavernous sinus syndrome except for the presence of proptosis. • Lateral sinus thrombosis → 6th and 7th cranial nerve palsies Risk factors • Local infection : e.g. Sinusitis, furuncle (Staphylococcus aureus is the most common) • Diabetes mellitus • Hypercoagulable states: pregnancy, post-partum period • Malignancy • Clotting disorders (e.g., factor V Leiden, protein C and S deficiencies, antiphospholipid syndrome) • Polycythemia • Medications: Oral contraceptive pill, Corticosteroids Features • Raised intracranial pressure (ICP) Headache: the most common presenting symptom Nausea & vomiting: Vomiting in the morning is characteristic of raised ICP as it follows a period of lying flat. visual disturbance. Papilloedema. • Peri-orbital oedema may be the earliest physical finding Chemosis, oedema and cyanosis of the upper face occur due to obstruction of the ophthalmic vein. Eye swelling begins as a unilateral process and spreads to the other eye within 24-48 hours via the intercavernous sinuses. This is pathognomonic for cavernous sinus thrombosis (CST). • Cranial nerve symptoms (e.g. Ophthalmoplegia): CN VI →Lateral gaze palsy (patient cannot abduct eye) is usually seen first. CN III → Ptosis, mydriasis, and eye muscle weakness CN V → Hyperaesthesia of upper face and eye pain • Seizures (focal or generalized) Investigations • If CVT is suspected, D-dimer levels and imaging studies are first steps of diagnosis • Contrast-enhanced CT scan is the test of choice to confirm the diagnosis shows a filling defect in a vein or sinus, the reverse delta sign (that is, empty triangle sign). On contrast CT → empty delta sign (is a specific to the superior sagittal sinus) Plain CT/MRI help detect only edema and/or infarcts, but the thrombus itself can be visualized by means of venography. • Evaluation for possible causes (e.g. CBC, CRP, Thrombophilia screen, antibody studies) Treatment • Anticoagulants (full-dose heparin then warfarin). • Dexamethasone can be used to reduce cerebral oedema. • Surgical therapy (e.g. Shunt or clot removal) indications:

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Neurology Progressive neurologic worsening (despite adequate anticoagulation) Acute rise in intracranial pressure Impending herniation Cavernous sinus contents Which group of nerves run through the cavernous sinus? III, IV, (V-1, V-2), and VI MRCPUK-part-1-September 2012: Left-sided eye pain & diplopia for the past 2 days + 6th & 3rd cranial nerve palsy on the left side + hyperaesthesia of the upper face on the left side. Where is the likely lesion? Cavernous sinus
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Empty delta sign indicating a superior sagittal sinus thrombosis • CT with contrast demonstrating a superior sagittal sinus thrombosis showing the typical empty delta sign. • Look at the 'bottom' of the scan for the triangular shaped dural sinus. • This should normally be white due to it being filled with contrast. • The empty delta sign occurs when the thrombus fails to enhance within the dural sinus and is outlined by enhanced collateral channels in the falx. • This sign is seen in only about 25%-30% of cases but is highly diagnostic for sagittal sinus thrombosis Structures passes through the cavernous sinus: • Internal carotid artery • Cranial nerves: third, fourth, and sixth cranial nerves Ophthalmic (V1) and maxillary (V2) divisions of the fifth cranial nerve The mandibular branch of the trigeminal nerve (V3) does not travel in the cavernous sinus and would therefore not be affected. (NO lower face symptoms)

Cervical vascular dissection (Carotid and Vertebral artery dissection) Overview • Important causes of stroke in young patients • The two commonest causes of young onset stroke (less than 40 years) are

1. Cardio-embolism and
2. Cervical artery dissection. • Dissection of the internal carotid artery can occur intracranially or extracranially Extracranially is more common. 75% of carotid dissections affect the internal carotid artery (that is, extracranially) Mechanism of ischaemia • Emboli from the site of the dissection (85-95%) of cases → ischaemic symptoms • vessel narrowing (5-15%): subintimal tears →intramural haematomas protrude inward and narrow the vessel lumen. Causes • Mechanical forces (eg, trauma, blunt injury, and stretching) • Arteriopathies (eg, Ehlers-Danlos syndrome, Marfan syndrome and other connective tissue disorders) Features • Pain (Headache, neck or facial pain): is the initial common symptom, ipsilateral to the dissected artery. • Ischaemic neurological features (transient or completed strokes): Sudden-onset • Partial Horner syndrome (Ptosis with miosis) usually painful when caused by internal carotid artery dissections The term partial Horner syndrome is used for the oculosympathetic palsy because anhydrosis is absent. Because the sympathetic fibers innervating the facial sweat glands are anatomically located on the external rather than the internal carotid artery Diagnosis • Plain CT head first to exclude haemorrhagic stroke • Computed tomography angiography (CTA) head and neck the diagnostic modality of choice. Stroke provoked by minimal trauma (e.g. exercise) is likely due to Cervical vascular dissection until proven otherwise Triad of carotid dissection:
3. unilateral (ipsilateral) headache
4. ipsilateral Horner's syndrome and
5. contralateral hemisphere signs (aphasia, neglect, visual disturbance,

Neurology Carotid dissection • Younger age group <50 years. • Neck pain. • Associated with vigorous exercise or event that sustains severe neck movement (e.g., roller coaster ride, motor vehicle accident). • May have Horner's syndrome or history of genetic collagen abnormality. Vertebral artery dissection: The typical presentation of vertebral artery dissection is a young person (average age 40 years) with severe occipital headache and neck pain following a recent head or neck injury. The trauma is often trivial but is usually associated with some form of cervical distortion. Vertebral artery dissection presents with: • Brainstem Stroke or transient ischaemic attack • Pain in the ipsilateral neck, face or head. • Commonly occurring in young people following a recent head or neck injury.

Carotid artery stenosis Epidemiology • Carotid artery stenosis causes 10% to 15% of all ischaemic strokes. • the annual risk of stroke in patients with asymptomatic carotid disease is between 1%

and 2% Pathophysiology • Atherosclerotic plaque in the cervical carotid artery is the most common cause. • Plaque disruption and athero-embolisation into the intracranial circulation is the most common mechanism for stroke. • The most common site of carotid Atherosclerosis: usually at the fork where the common carotid artery divides into the internal and external carotid artery. Features • The majority are asymptomatic. • TIA or stroke Plaques rupture →embolism to intracranial arteries →(TIA or stroke) or embolism to retinal arteries →(amaurosis fugax or retinal strokes). • Cervical bruit Diagnosis • Duplex ultrasonography is the preferred initial mode of diagnosis and screening. sensitivity of 99%, specificity of 86% If the stenosis is less than 50% →no further imaging is needed. Carotid endarterectomy for stroke or TIA in the carotid territory only indicated if:

1. ≥50% ipsilateral carotid stenosis
2.

The patient not severely disabled

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 If the stenosis > 50% →CTA or MRA for more detailed plaque characterization. • CT or Magnetic resonance angiography (CTA or MRA) helps to define the anatomy if intervention is indicated. Management • Initial management →Antiplatelet therapy, Statin and risk factor modification. • Carotid revascularization → Endarterectomy Indications: Significant stenosis: Carotid stenosis > 70% according ECST criteria (European Carotid Surgery Trial' Collaborative Group) or > 50% according to NASCET (North American Symptomatic Carotid Endarterectomy Trial) criteria. TIA or resolving stroke (The patient not severely disabled): The benefit of endarterectomy is prevention of future stroke, with dense strokes, if there is no recovery, the benefits are greatly reduced due to end-organ damage. Asymptomatic carotid stenosis ≥ 70% : 1st line → antiplatelet therapy and cardiovascular risk reduction. the best course of action? → Discharge and outpatient follow up Contraindications: 100% carotid stenosis usually requires a bypass procedure, as risk of endarterectomy outweighs benefit. previous stroke with persistent neurological symptoms Timing of surgery: should be performed within two weeks. ("urgent" endarterectomy within 2 weeks) Benefit: It reduces the risk of disabling stroke or death by 48% in a person with severe symptomatic carotid stenosis (>70%) who has had a TIA. • Carotid stenting used as an alternative to endarterectomy: Indications Restenosis. Previous radiotherapy to the neck may make endarterectomy difficult, and stenting may be preferred. MRCPI-part-2-april-2018: left-sided hemiparesis of more than 8 hours’ duration. carotid ultrasound scan, shows 80% stenosis of the left carotid artery, 50% stenosis of the right carotid artery. What is the most appropriate treatment for long-term stroke prevention? Clopidogrel endarterectomy is not recommended in: significant stenosis but asymptomatic side (left carotid in this case) symptomatic side but there is less than 70% (right carotid in his case). Carotid artery atherosclerosis is an important cause of ipsilateral amaurosis fugax.

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Localisation of speech problems Overview • The speech area is in the left, dominant side of the brain in about 99% of right-handed people • Thus, impairment of the speech area with a stroke, causing left-sided weakness, is rare. It will occur in virtually no right-handers and in only 30% of left-handers. • As a general rule, a lesion of the left hemisphere will cause dysphasia whilst, in the right hemisphere, it will cause neglect, visuo-spatial and cognitive problems • Wernicke's aphasia and pure aphasia (that is, without alexia) are middle cerebral artery. • Comprehension, fluency and repetition are the three main variables that allow for localisation of speech problems • The three, general, areas are:

1. Wernicke's area (posterior, superior temporal lobe) - lesions produce normal fluency, impaired comprehension, impaired repetition receptive aphasia They are unaware of their language difficulties
2. conduction (arcuate fasciculus) - lesions produce normal fluency, normal comprehension, diminished repetition
3. Broca's area (inferior frontal lobe) lesions produce impaired fluency, intact comprehension, impaired repetition. Unlike Wernicke's aphasia, Broca's patients are aware of their language difficulties. Only 1 out of 3 features of speech are affected: • poor comprehension with normal fluency and repetition →Transcortical sensory aphasia • poor fluency with normal comprehension and repetition →Transcortical motor aphasia • poor repetition with normal fluency and comprehension →Conduction aphasia 2 out of 3 features of speech are affected: • Poor comprehension and fluency with normal repletion →Transcortical mixed aphasia • Poor comprehension and repetition with normal fluency →Wernicke (receptive) aphasia • Poor fluency and repetition with normal comprehension →Broca's (expressive) aphasia. All 3 features of speech are affected: • Poor fluency, comprehension and repetition →global aphasia

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Aphasia Syndromes Aphasia Lesion Fluency Comprehension Repetition Broca's (expressive) Broca's area (inferior frontal lobe) superior division of the left middle cerebral artery (MCA) lesion No Yes No Wernicke (receptive) (superior temporal lobe) inferior division of the left MCA lesion Yes No No Conduction arcuate fasciculus peri-Sylvian area Yes Yes No Transcortical motor anterior cerebral artery (ACA)-MCA watershed infarct No Yes Yes Transcortical sensory posterior cerebral artery (PCA)-MCA watershed infarct Yes No Yes Transcortical mixed Can be secondary to both an ACA-MCA and PCA-MCA infarct No No Yes Global proximal MCA occlusion affecting both superior and inferior division of the MCA No No No • Mixed aphasia (or transcortical mixed aphasia) patients can often repeat words but not understand commands. Not specific for stroke , commonly caused by Alzheimer's disease, bilateral cerebral damage, tumours, and thalamic lesions. • Transcortical sensory aphasia The main problem lies within the brain in a region known as the temporal-occipitalparietal junction, located behind Wernicke's area. The patient has intact repetition but is unable to follow verbal commands. He has fluent grammatical speech. • Anomic aphasia or nominal aphasia results in word finding difficulties. • Aphemia is a type of aphasia in which there is severe dysarthria and impairment of verbal output. There is intact comprehension. MRCPUK-part-1-January 2008 exam: H/O difficulty in finding the right words whilst speaking. With normal comprehension. Where is the likely lesion? Posterior frontal lobe (expressive aphasia due to a

lesion in Broca's area, located on the posterior aspect of the frontal lobe, in the inferior frontal gyrus)

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Pupil conditions Pupillary control • Oculomotor nerve carries parasympathetic efferent to the sphincter pupillae muscle. • Optic nerve carries sympathetic postganglionic fibres to the dilator pupillae muscle. Pupillary control • Parasympathetic fibers lead to pupillary constriction (miosis) light enters the eye →retinal ganglion →optic nerve → optic chiasm → optic tract → pretectal nucleus→ Edinger-Westphal nucleus →ciliary ganglion →pupillary constrictor muscles →causing uniform bilateral miosis • Sympathetic fibers lead to pupillary dilation (mydriasis) hypothalamic nuclei →T1 and T2 spinal cord levels →paravertebral sympathetic chain (via the white ramus) →superior cervical ganglion →pupillary dilator muscle • Causes of small pupils include: Horner's syndrome Old age Pontine haemorrhage Argyll Robertson pupil Drugs, and Poisons (opiates, organophosphates). • Causes of dilated pupils include: Holmes-Adie (myotonic) pupil Third nerve palsy Drugs, and Poisons (atropine, CO, ethylene glycol). Pupil Lesion Slightly smaller but reactive Early stage of thalamic damage Fixed dilatated (7 mm) nonreactive Oculomotor nerve lesion Fixed midsized pupil (5 mm) Midbrain lesion Pinpoint pupil (1 – 1.5 mm) Pontine lesion/ opioid overdose Asymmetrical pupils Normal in 20% of population but reactive. If one pupil is sluggish to react than the other think of midbrain or oculomotor • Equality of pupils diameter Afferent pupillary defect (e.g. optic neuritis) → pupils are isocoric (equal diameter) Efferent (impulse transmission to the iris sphincter muscle) pupillary defect (i.e. impairment of the pupillary reflex) → pupils are anisocoric (unequal diameter)

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Hippus • Hippus is papillary athetosis.(athetosis →abnormal muscle contraction causes involuntary writhing movements). • It is a spasmodic rhythmical dilation and contraction of the pupil. • It is typically a benign finding.

Tonic pupil (Holmes-Adie pupil) Holmes-Adie →dilated pupil Definition • Tonic pupil or Holmes-Adie pupil is a dilated pupil caused by parasympathetic damage. Pathophysiology • parasympathetic denervation at the level of the ciliary ganglion and postganglionic nerves. Causes • Idiopathic (Most cases) • Local causes: infections , trauma • Systemic autonomic neuropathies Ross syndrome is characterized by a triad of tonic pupil, hyporeflexia, and segmental anhidrosis Horner syndrome Features • Anisocoria (unequal pupil diameter) Although the tonic pupil is usually larger than the uninvolved fellow eye, over time the pupil tends to become smaller (the "little old Adie" pupil). • Hypersensitivity to dilute pilocarpine drops. Diagnosis • Clinically : poor pupillary reaction to light + normal test for a pupillary near response (lightnear dissociation). • The usual diagnostic test is to use weak pilocarpine eye drops, which induce vigorous pupil contraction on the affected side, but only weak contraction of the pupil on the unaffected side. • Patients with

unexplained bilateral tonic pupils should have serologic testing for syphilis Treatment • benign condition → observe Ross’s syndrome: The triad of

1. abnormal pupil size,
2. loss of deep tendon reflexes, and
3. excessive sweating. Although some doctors will still diagnose the condition as a variant of Holmes-Adie pupil.

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Argyll Robertson Pupil (ARP) Argyll-Robertson Bilateral small irregular pupils that do not react to light but react to accommodation. Referred to as the "Whore's Eye" because of the association with tertiary syphilis and because of the convenient mnemonic that, like a prostitute, they “accommodate but do not react” Causes : neurosyphilis, Multiple Sclerosis, Sarcoidosis, DM Accommodation Reflex Present ARP Pupillary Reflex Absent Argyll Robertson Pupil (ARP) • Bilateral small pupils • Prostitute's pupils →reduce in size on a near object (they “accommodate”), but do not constrict when exposed to bright light (they do not “react” to light). • They are a highly specific sign of neurosyphilis and might also be a sign of diabetic neuropathy. Pupillary Defect Comments Argyll Robertson pupil • Pupils accommodate but do not react to direct or indirect light A type of light-near dissociation where Bilateral miosis the eye does not constrict in response to light as much as it does with accommodation pupil has an absent light reflex • Associated with neurosyphilis Adie's myotonic pupil • A type of light-near dissociation where the eye does not constrict in response to light as much as it does with accommodation light reflex is merely reduced Affected eye is dilated usually • Secondary to degeneration of the ciliary ganglion

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Visual field defects Visual field defects: • left homonymous hemianopia means visual field defect to the left, i.e. lesion of right optic tract • homonymous quadrantanopias: PITS (Parietal-Inferior, Temporal-Superior) • incongruous defects = optic tract lesion; congruous defects= optic radiation lesion or occipital cortex

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The main points for the exam are: • left homonymous hemianopia means visual field defect to the left, i.e. Lesion of right optic tract • homonymous quadrantanopias: PITS (Parietal-Inferior, Temporal-Superior) • incongruous defects = optic tract lesion; congruous defects = optic radiation lesion or occipital cortex • A congruous defect simply means complete or symmetrical visual field loss and conversely an incongruous defect is incomplete or asymmetric. Please see the link for an excellent diagram. Homonymous hemianopia • incongruous defects: lesion of optic tract • congruous defects: lesion of optic radiation or occipital cortex • macula sparing: lesion of occipital cortex Homonymous quadrantanopias • superior: lesion of temporal lobe • inferior: lesion of parietal lobe • mnemonic = PITS (Parietal-Inferior, Temporal-Superior) Bitemporal hemianopia •

lesion of optic chiasm • upper quadrant defect > lower quadrant defect = inferior chiasmal compression, commonly a pituitary tumour • lower quadrant defect > upper quadrant defect = superior chiasmal compression, commonly a craniopharyngioma Cortical blindness • Patients with cortical blindness frequently have visual hallucinations and occasionally deny that they are blind. • Pupillary reactions and fundoscopy are normal. Bitemporal hemianopia: • lesion of optic chiasm • upper quadrant defect > lower quadrant defect = inferior chiasmal compression, commonly a pituitary tumour • lower quadrant defect > upper quadrant defect = superior chiasmal compression, commonly a craniopharyngioma

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Neurology Visual field defects Bilateral internal carotid artery displacement can cause binasal incongruous hemianopia if the optic nerves are compressed. MRCP-part-1-September 2012 exam: What sort of visual field defect is expected following an operation to remove a meningioma in left temporal lobe? □ Right superior homonymous quadrantanopia Mrcpuk-part-1-January 2009 exam: In a left congruous homonymous hemianopia. Where is the lesion most likely to be? □ Right occipital cortex

Cranial nerves The major characteristics of the 12 cranial nerves Nerve Functions Clinical I (Olfactory) Smell Cribriform plate II (Optic) Vision Optic canal III (Oculomotor) Eye movement (MR, IO, SR, IR) Pupil constriction Accommodation Eyelid opening IV (Trochlear) Supplies superior oblique (SO) →(depresses eye, moves inward) V (Trigeminal) Facial sensation Mastication VI (Abducens) Eye movement (LR) Palsy results in defective abduction → horizontal diplopia VII (Facial) Facial movement Taste (anterior 2/3rds of tongue) Lacrimation Salivation VIII (Vestibulocochlear) Hearing, balance Hearing loss Vertigo, nystagmus Acoustic neuromas are Schwann cell tumours of the cochlear nerve IX (Glossopharyngeal) Taste (posterior 1/3rd of tongue) Salivation supplies the parotid salivary gland controlling salivary secretions. Swallowing Mediates input from carotid body & sinus Notes & Notes for MRCP
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Pathway/ foramen Palsy results in Superior orbital fissure (SOF) • ptosis • 'down and out' eye • dilated, fixed pupil Palsy results in defective downward gaze → vertical diplopia SOF Lesions may cause: V1: SOF, V2: Foramen rotundum, V3: Foramen ovale • trigeminal neuralgia • loss of corneal reflex (afferent) • loss of facial sensation • paralysis of mastication muscles • deviation of jaw to weak side SOF Lesions may result in: Internal auditory meatus • flaccid paralysis of upper + lower face • loss of corneal reflex (efferent) • loss of taste • hyperacusis Internal auditory meatus Lesions may result in; Jugular foramen • hypersensitive carotid sinus reflex • loss of gag reflex (afferent)

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Neurology Nerve Functions Clinical X (Vagus) Phonation Swallowing Innervates viscera supplies the palatal muscles XI (Accessory) Head and shoulder movement XII (Hypoglossal) Tongue movement Tongue deviates towards side of lesion The fourth cranial nerve palsy →superior oblique palsy → vertical diplopia (eg: missing steps when walking down the stairs, bumping head when trying to get out of a car) Cranial nerve locations • 4 CN are above pons (I,II,III,IV). • 4 CN exit the pons

(V,VI,VII,VIII). • 4 CN are in medulla (IX,X,XI,XII).

Cranial nerve reflexes Reflex Afferent limb Efferent limb Corneal Ophthalmic nerve (V1) Facial nerve (VII) Jaw jerk Mandibular nerve (V3) Mandibular nerve (V3) Gag Glossopharyngeal nerve (IX) Vagal nerve (X) Carotid sinus Glossopharyngeal nerve (IX) Vagal nerve (X) Pupillary light Optic nerve (II) Oculomotor nerve (III) Lacrimation Ophthalmic nerve (V1) Facial nerve (VII) Brain stem (Mid brain, Pons, Medulla Oblongata) lesions are typically characterized by ipsilateral cranial nerve involvement and contralateral body involvement. Petrous apex lesion • Features Abducens nerve palsy →horizontal diplopia Trigeminal nerve involvement at the Meckel cave → ipsilateral facial pain or sensory disturbance (numbness) • Causes →Meningioma or nasopharyngeal carcinoma of the petrous apex is the most common cause now Lesions of the cerebellopontine angle causes compression of cranial nerves V (trigeminal), VII (facial) and VIII (vestibulocochlear). Notes & Notes for MRCP
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Pathway/ foramen Lesions may result in; Jugular foramen • uvula deviates away from site of lesion • loss of gag reflex (efferent) Lesions may result in; Jugular foramen • weakness turning head to contralateral side Hypogloss al canal

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Optic nerve palsy Anatomy • The optic nerve is part of the central nervous system; hence its myelin sheaths are derived from oligodendrocytes, not Schwann cells. • Accordingly, diseases of the peripheral nervous system and radiculopathies don’t target the optic nerve. • The physiological blind spot results from absence of photoreceptors in the area of the retina where the optic nerve leaves the eye. Causes • Ischemic optic neuropathy • multiple sclerosis • optic nerve glioma • ethambutol Features • Complete transection → ipsilateral blindness and loss of direct pupillary light reflex • Compression (e.g., tumor) → optic atrophy • Pituitary adenoma → compression to the optic chiasm → bitemporal hemianopia

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Oculomotor (third nerve) palsy Features • Divergent squint - affected eye deviated 'down and out'. Downward displacement result from unopposed action of the superior oblique (innervated by the fourth cranial or trochlear nerve). due to paralysis of superior rectus, inferior rectus and inferior oblique. outward displacement results from unopposed action of the lateral rectus (innervated by the sixth cranial nerve). due to paralysis of the medial rectus muscle. • Ptosis • Dilated pupil (mydriasis), sometimes called a 'surgical' third nerve palsy. the parasympathetic fibers run on the outside of the nerve. Therefore, 3rd nerve compression →mydriasis appear before ptosis and "down and out" position are seen. pupillary abnormalities are more commonly associated with trauma than with ischemia. Compression of the oculomotor nerve can cause isolated pupillary dilation due to injury of the parasympathetic fibers. Microangiopathy (e.g., due to diabetes mellitus) typically affects the deeper somatic fibers first, causing ophthalmoplegia without pupillary dilation Ipsilateral 3rd CN palsy + contralateral hemiplegia →Weber's syndrome Ipsilateral 3rd CN palsy + contralateral hemiataxia →Benedikt syndrome Ipsilateral 3rd CN palsy + ipsilateral hemiparesis + Contralateral homonymous hemianopsia → Uncal herniation

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 A patient with a third nerve palsy with pupillary involvement should be considered to have a posterior communicating artery aneurysm until proven otherwise → requires urgent neurosurgical evaluation. • Unreactive pupil to light: Lesions of the autonomic (parasympathetic) portion → absence of the pupillary reaction Causes • Vascular causes (usually does not affect the pupil): Diabetic neuropathy, vasculitis, Weber's syndrome • Compressive lesions: Posterior communicating artery aneurysm (painful, pupil dilated) → Urgent CT angiogram of the cerebral vessels is required for diagnosis. • Cavernous sinus thrombosis • Others causes: amyloid, multiple sclerosis Parasympathetic fibers are located more superficially than motor fibers, causing the following features: • Prominent motor dysfunction and sparing of the pupil in ischemic lesions due to vascular disease (e.g., vasculitis, diabetes): parasympathetic fibers are less affected by decreased diffusion of nutrients from the vasa nervorum • Severely impaired pupillary reaction with relatively spared motor function in compressive lesions (e.g., uncal herniation, aneurysm of the posterior communicating artery): parasympathetic fibers are affected by compression first Painful third nerve palsy = posterior communicating artery aneurysm Raised ICP can cause a third nerve palsy due to herniation

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