Neurology VIVAs (Pathology)

shakEM

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2015.1.A.2

Question 3 Meningitis **Subject:** Path LOA: 1	What are the other types of meningitis? What organisms commonly cause bacterial meningitis in the different age groups?	**Viral**, chronic (tuberculosis), fungal, chemical / drug induced, carcinomatous Neonates: Escherichia Coli and Group B Streptococcus Children: **Streptococcus Pneumoniae**, Haemophilus Influenza (now less common) Adolescent / young adult: **Neisseria Meningiditis, Streptococcus Pneumoniae** Older adults: Strep Pneum, Listeria	3 out of 5 including bold 1 per age group, must mention Bold.

2015.1.D.3

Question 2 Traumatic Brain Injury Subject: Path LOA: 1	1. Which type of vessels have been damaged to produce the subdural blood seen on this CT? 2. Which groups of patients are most at risk for SDH and why? 3. How does an extradural haematoma occur? 4. Define and describe diffuse axonal injury?	1. Subdural blood comes from damage to **bridging veins between the brain and the venous sinuses** (displacement of the brain with in trauma can tear the veins at the point where they penetrate the dura to enter the sinuses) -> **blood between the dura and the arachnoid.** 2. **Elderly-** veins stretched and more movement due to brain atrophy Infants- thin walled bridging veins 3. Extradural hematoma occurs with **rupture of a meningeal artery**, usually associated with a skull fracture, leads to accumulation of arterial blood **between the dura and the skull.** 4. **Axonal microscopic injury** Micro findings include **axonal swelling and focal haemorrhagic** lesions. Believed to **damage the integrity of the axon at the node of Ranvier, -> alterations in axoplasmic flow. Commonly found with 'coma' but no cerebral contusions.**	Bridging veins Elderly **Meningeal (often middle) artery** **Microscopic damage to deep brain white matter**

2014.2.D.2

Question 3 Cerebrovascular Disease (pp 1290-1295) **Subject:** Path LOA: 1	What are the types of cerebral ischemic injury? Prompt: Describe the patterns cerebral ischemic injury What are the causes of focal cerebral infarction? Prompt: Give examples What are the pathological effects of hypertension on the brain?	**Global cerebral ischemia** (ischemic/ hypoxic encephalopathy) when there is a **generalised reduction** of cerebral perfusion **Focal cerebral ischemia** follows reduction of blood flow to a **localised** area of the brain **Embolic** (from cardiac mural thrombi; thromboemboli from arteries, esp. carotid; paradoxical assoc with cardiac anomalies; tumour, fat or air), **thrombotic arterial occlusion/** in situ thrombosis (large vessel disease); **Vasculitis** (small vessel disease) infectious (immunosuppression and aspergillus, CMV encephalitis, syphilis, TB); non-infectious eg PAN, primary angiitis; **Others** eg amphetamines, cocaine, heroin; dissecting aneurysm extracranial arteries; hypercoaguable states Lacumar infarcts (in lenticular nucleus, thalamus, internal capsule, deep white matter, caudate nucleus, pons); slit haemorrhages; hypertensive encephalopathy; massive intracerebral haemorrhage)	Both types and description 3 causes plus 1 example of each 4 out of 4

2013.1.3

Question 5 Traumatic CNS Injury LOA: 1	1/ What types of intracranial bleeding can be seen in a patient with a head injury?	1/ **Extradural** **Subdural** **Subarachnoid (including intraventricular)** **Intra-parenchymal**	3 of 4
	2/What sequence of events occur in an extradural haemorrhage	2/Dural **artery** (eg. middle meningeal) tear**, usually associated with a skull fracture** Strips off the dura from the skull May be a lucid period before ALOC	Must get bold
	3/Define concussion and what are its clinical features?	3/**Altered consciousness secondary to a head injury** **Transient** neurological dysfunction Transient resp arrest Transient loss of reflexes (pathogenesis is unclear, may be dysregulation of RAS)	Must get bold
		Features inc headache, amnesia, N&V, Concentration and Memory issues, perseveration, irritability, behaviour/personality changes, dexterity loss, neuropsychiatric syndromes	3 features

2012.2.1

Thurs AM Q4 Acute meningitis LOA 2	1. What are the types of meningitis? *Prompt: What other type?*	Infectious meningitis: **acute pyogenic, aseptic (inflammatory) viral, parasitic, chronic (TB)** chemical meningeal carcinomatosis	**Bacterial, viral + 1 other**
	2. What bacteria cause meningitis in different patient groups?	Neonates: E. Coli; Gp B Strep Infants: HIB (less with immunisation) Strep Young adults: N. meningitidis Elderly: Strep pneumoniae; Listeria Immunosuppressed: Klebsiella; anaerobe;	**3 bacterial causes including N. meningitidis in right age range**
	3. How do the CSF findings differ between bacterial and viral meningitis?	BACTERIAL / VIRAL Increased pressure / May be normal/slight inc Cloudy or purulent / Often clear **Increased white cells - neutrophils** / **Less increase white cells - lymphocytes** Raised protein / Only moderate increase Reduced glucose / Nearly always normal Bacteria on smear / (PCR)	**White cell differences x2 + 1 other**

2012.2.3

Fri AM Q3 CVA LOA: 1	1 What are the causes of focal cerebral infarction?	**1. Arterial thrombosis, Cerebral embolism** Lacunar- arteriosclerosis of the vessels in the lenticular nucleus, thalamus, internal capsule, deep white matter, caudate nucleus, and pons Arteritis – giant cell (temporal arteritis), PAN, SLE, infectious (CMV, aspergillosis, TB, Syphilis) Arterial dissection Venous infarction – hanging, - venous sinus thrombosis	Need bold (arterial thrombosis, embolism) and one other (underlined) to pass.
	2. What are the sources of cerebral thromboemboli? (Prompt: What happens in cerebral embolism?)	2.Source (s) - usually from heart (**LAA, mural thrombus, valvular vegetations**) - **plaques from carotid bifurcation**; - **paradoxical emboli** in patent foramen ovale Precipitant (not specifically in text) – Afib / cardioversion Consequence – most commonly **lodges in MCA** , often at branch points, **causes ischaemia** due to poor collateral flow	Need at least 1 cardiac and 2 sources in total to pass.

2012.1.1

Question 3 Subarachnoid haemorrhage	**Where in the cerebral circulation are saccular (berry) aneurysms commonly located?** ***Prompt:*** At what part of these vessels are they most likely to arise? **What factors increase the likelihood of rupture of these aneurysms?** **What are the pathological sequelae of subarachnoid haemorrhage?**	90% near major arterial **branch points – Anterior** Cerebral A / ACoA (40%); MCA / AChoroidalA (34%); ICA / PCoA (20%); Basilar A / PCoA. Multiple in 20% – 30% cases at autopsy. Increased likelihood with **size** (> 10mm) – 50% risk of rupture per year. May occur at anytime but in about 1/3 associated with **acute increases in ICP** (e.g. straining at stool; orgasm). Acute events (hours to days) – **ischaemic injury (stroke)** from **vasospasm** (especially basal SAH). Late events (healing process) – meningeal fibrosis and **scarring**; may lead to obstruction to **CSF** flow and /or to CSF absorption. Death	Mention of branch points and anterior circulation to pass. **Bold** to pass. **Two of bold** to pass.

2011.1.2

Question 5. **Subarachnoid Haemorrhage**	1. What is the **most frequent cause of subarachnoid haemorrhage?**	• **Rupture of an aneurysm** • (less common causes include ext of traumatic haem, H/T intracerebral bleed into ventricular system, AVM, bleeding disorders, tumour)	Rupture of aneurysm to pass
	2. **Where are saccular aneurysms commonly located?**	• **Most** near major arterial branch points along the circle of Willis or a major vessel just beyond (= **anterior cerebral circulation**) • 40% ant comm art • 34% middle cerebral art • 20% int carotid/PICA • 4% Basilar/Posterior Cerebral	At least anterior circulation and 1 other to pass
	3. What are the genetic risk factors for saccular aneurysms?	• Generally unknown, not 'congenital' • Some genetic risk o Polycystic kidney o Ehlers Danlos type 4 o Neurofibromatosis type 1 o Marfan's) o Fibromuscular dysplasia o Aortic coarctation	2/6
	4. What are the pathological **consequences of subarachnoid haemorrhage?** Prompt for "Late"	• **Early** - vasospasm and additional ischemic injury - increased intracranial pressure • **Late** - meningeal fibrosis & scarring - CSF obstruction	Need 2

2010.2.2

Question 2.5 Parkinsonism	1. Describe the clinical features of Parkinsonism. (Prompt: How do Parkinsonian patients look?) 2. What are the causes of Parkinsonism? (Prompt: what part of the brain is affected?) 3. Outline the possible pathogenesis of Parkinson's Disease.	1. Diminished facial expression, stooped posture, slowness of voluntary movement, festinating gait (progressively shortened, accelerated steps), rigidity and a "pill-rolling" tremor. **2. Conditions that cause damage to the <u>nigrostriatal dopaminergic system</u>** 2.1. Parkinson disease 2.2. Post-encephalitic 2.3. Familial forms (rare – auto dominant & recessive) 2.4. trauma/ injuries 2.5. Drugs – dopamine antagonists/toxins/pesticides 2.6. Multiple system atrophy, progressive supranuclear palsy 3. Possible pathogenesis – no unifying pathogenic mechanism identified 3.1. Misfolded protein/stress response triggered by α-synuclein aggregation 3.2. Defective proteosomal function due to the loss of the E3 ubiquitin ligase parkin 3.3. Altered mitochondrial function caused by the loss of DJ-1 and PINK1 3.4. Genetic variants with gene defects 3.5. Possible damage to dopaminergic cells from toxins drugs/AI conditions	1. 3 of 6 2. Bold + 2

2010.1.1

Question 5: Meningitis	a) Classify meningitis with examples of important causes.	- Acute pyogenic: bacterial - Aseptic: viral, chemical - Chronic: infection: TB, infiltration: carcinomatous	Must have bacterial and viral and at least one other
	b) What are the likely organisms causing acute bacterial meningitis in different age groups?	• E coli/Group B strep: neonates • Pneumococci: infants/older(all ages beyond neonates really) • Meningococci: All ages beyond neonates esp. young adults • Haemophilus: Children but decreased incidence with Immunisation • Listeria. extremes of age • Unusual orgs e.g staph aureus post N/surg. Immuno compromised eg gram negatives.	3 of 6
	c) What are the typical CSF findings in acute bacterial meningitis?	• Raised pressure • Turbid • Raised protein • Lower glucose • *Raised neutrophils • *+ve bacteria on gram stain or culture	* and one other

2009.2

Friday 18th Morning Question 5: MS p1383	What are clinical features of Multiple Sclerosis	Distinct episodes of neurological **deficits separated by time**. Myriad of presentations as lesions separated by space. Unilateral visual impairment (optic neuritis) is common, brainstem, cord lesions	Bold to pass
	What is the pathogenesis of Multiple Sclerosis?	Exact etiology not established **Autoimmune, demyelinating** disorder, to white matter lesions separated in space. Genetic linkage, ?microbial / viral triggers. CD4+ Th1 T cells react against myelin antigens, release cytokines, activate macrophages. Inflammatory cells create **plaques**.	Need bold to pass
	What might be found in CSF of a patient with MS?	Mildly **elevated protein**; moderate pleocytosis; increased proportion of gamma globulin, oligoclonal bands – reflects B cells	Bold to pass

2008.2

5. Pituitary Adenomas:	1. How are pituitary adenomas classified? Prompt: Name two cell types involved.	Classification based on **hormone cell-type**: prolactin cell, growth hormone cell (densely or sparsely granulated), thyroid stimulating cell, ACTH cell, gonadotroph cell (including silent and oncocytic), mixed GH-prolactin cell, Other plurihormonal cell, hormone negative.	Highlighted & 2 cell types to pass. If describe "functional" or "silent" adenomas – move to prompt
	2. What clinical syndromes may they produce?	Prolactinoma: amenorrhea, galactorrhea, loss of libido, and infertility Somatotroph (GH): gigantism or acromegaly ACTH: Cushing's syndrome Gonadotroph: local effects (headaches, visual impairment, diplopia, pituitary apoplexy), hypogonadism (lethargy, loss of libido, amenorrhoea)	