ACEM Primary Examination Vivas > Pathology > CNS

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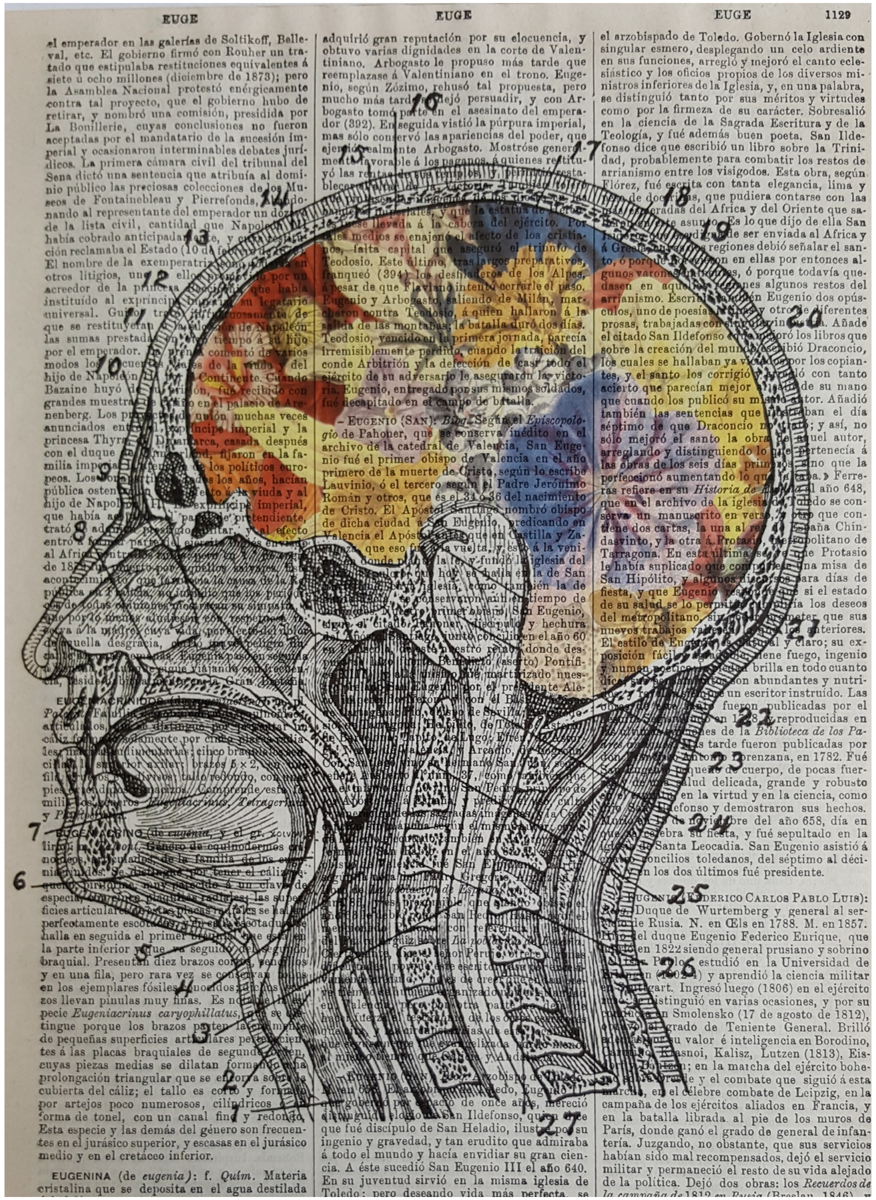

Berry Aneurysms 2012-1

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.

Berry Aneurysms 2007-2

OPENING QUESTION	What is the morphology of a berry aneurysm?	COMMENTS
POINTS REQUIRED	1. Medial muscular layer thins as approaches neck and gets thickened hyalinised intima, covered with normal adventitia	
SECOND QUESTION	**What are the common sites of berry (saccular) aneurysms?**	
POINTS REQUIRED	2. Anterior circulation around Circle of Willis at arterial junctions – internal carotid; middle cerebral; anterior cerebral and anterior communicating. 3. May be multiple (20-30%).	Prompt: Anterior or posterior circulation?
	Anterior circulation around Circle of Willis	Desired knowledge
THIRD QUESTION	**What is the natural history of a ruptured berry aneurysm?**	
	1. Acute onset of severe headache, often with loss of consciousness. 2. 25-50% die at the time. 3. Rebleeding is common. 4. Vasospasm in vessels other than the bleeding site can cause secondary ischaemic injury. 5. In the healing phase, meningeal fibrosis and scarring can cause secondary hydrocephalus.	Headache, high mortality initial and rebleeding 3 of 4

Cerebral Infarction 2017-2-B

Stem: Moving on to Pathology.			
Question 4 Cerebral infarction **Subject:** Pathology LOA: 1	(a) What are the causes of ischaemic cerebral infarction?	(a)**Arterial thrombosis** - Cerebral emboli - lacunar infarcts from small vessels - cerebral arteritis - arterial dissection - venous infarction	Thrombus + one
	(b) Where are some sources of cerebral thromboemboli?	(b)left atrium/ventricle thrombus - valvular vegetations - PFO causing paradoxical emboli - Carotid plaque	At least 2 sources

Cerebral Infarction 2016-1-C

Stem: Moving onto Pathology			
Question 3 Ischaemic Stroke / Reperfusion injury **Subject:** Path LOA: 1	1. What are the main pathological processes causing ischaemic stroke? *(Give examples for each category)*	**Thrombotic occlusion – atherosclerosis** most common **Embolism** – AMI, mural thrombus, Valvular heart disease, AF, vascular surgery and shower embolism, fat embolism, endocarditis Inflammatory process leading to luminal narrowing - Infectious vasculitis, autoimmune vasculitis, primary angiitis of the CNS	**Bold to pass** and at least 2 causes of embolism plus one other (embolic or inflammatory).
	2. What are the distinguishing pathological features of haemorrhagic and non-haemorrhagic ischaemic cerebral infarcts? Prompt: why does haemorrhagic change occur in ischaemic stroke.	Haemorrhagic (**red**) - multiple, sometimes confluent, petechial haemorrhages typically associated with **embolic** events. Thought to be secondary to **reperfusion** either via collaterals or dissolution of materials. Greater risk if anticoagulated. Non-Haemorrhagic (**pale/bland anaemic**) – usually associated with **thrombosis.**	Bold causes and concepts
	3. How are these pathological processes important in relation to stroke thrombolysis?	Complications higher with embolic/haemorrhagic CVAs. Trying to reverse injury in ischaemic penumbra. In non- haemorrhagic CVA little macroscopic change can be seen within the first 6 hours. Earlier treatment leads to better outcome and less haemorrhagic risk.	Reversible ischaemic penumbra (term or concept) Red Vs pale –Red CI

Cerebral Infarction 2014-2-D

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	**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	Both types and description
	What are the causes of focal cerebral infarction? Prompt: Give examples	**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)	3 causes plus 1 example of each
	What are the pathological effects of hypertension on the brain?		4 out of 4

Cerebral Infarction 2012-2

Fri AM Q3 CVA LOA: 1	1 What are the causes of focal cerebral infarction? 2. What are the sources of cerebral thromboemboli? (Prompt: What happens in cerebral embolism?)	**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 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 bold (arterial thrombosis, embolism) and one other (underlined) to pass. Need at least 1 cardiac and 2 sources in total to pass.
		1. Age: 25% in those >80 yo, women > men; Environmental factors	3 of 5 bolded

Cerebral Infarction 2007-2

TOPIC: Non Haemorrhagic CVA		**NUMBER: Q4**
OPENING QUESTION	What are the causes of cerebral infarction? Prompt: What causes obstruction of blood supply	**COMMENTS**
POINTS REQUIRED	Hypoxia and ischaemia – **Thrombotic / Embolic** Prompt – What are the causes of thrombosis and/or embolus	Mandatory
	Atherosclerosis	Mandatory
SECOND QUESTION	Arteritis – Syphilis, TB, PAN, Opportunistic infections (Toxoplasmosis, aspergillosis, CMV)	2/6
POINTS REQUIRED	Dissection	
	Primary angiitis of CNS	
	Hypercoagulation state	3/5
POINTS REQUIRED	Drug abuse (cocaine, amphetamine, heroin)	
	Embolic -	1/1
	cardiac (Mural: Myocardial infarction, valve, AF)	Mandatory + 2/3 examples
	Arterial, carotid	1/1
	Paradoxical and other (tumour, fat, gas, post surgical)	2/4

Cerebral Odema 2015-2-D

Stem: Moving onto Pathology. He has a head injury and a CT brain reveals gross cerebral oedema.			
Question 2 Cerebral Oedema and raised ICP **Subject:** Path LOA: 1	Describe the pathological mechanisms which cause cerebral oedema. (prompt if specific examples used – can you describe the difference between vasogenic and cytotoxic oedema?)	**Vasogenic**. BBB disruption, increased vascular permeability. Fluid shift intravascular to intercellular spaces of brain May be generalised or localised (inflammation or neoplasm) **Cytotoxic.** Increased intracellular fluid due to neuronal, glial, or endothelial injury eg generalised hypoxic/ ischaemic insult or metabolic damage Interstitial or ependymal oedema around (lateral) ventricles due to the high pressure of hydrocephalus	**Bold to pass or basic understanding of two mechanisms**
	What are the morphological findings of generalised cerebral oedema. (Prompt: What would be the CT findings?)	Flattened gyri, narrowing of sulci, compression of ventricles and/or basal cisterns, herniation	3 of 4 to pass
	Describe the major herniation locations associated with raised intracranial pressure	**Subfalcine** herniation- Asymmetric expansion of cerebrum displaces the cingulate gyrus under the falx cerebri **Transtentorial** or Uncal herniation -Medial aspect of the temporal lobe is compressed against the free margin of the tentorium **Tonsillar herniation**- Displacement of the cerebellar tonsils through the foramen magnum.	2 of 3 bold plus correct description

Dementia 2007-2

TOPIC: Dementia/Alzheimer's disease _____ **NUMBER: Q5** _____

OPENING QUESTION	What are some of the causes of dementia?	**COMMENTS**
POINTS REQUIRED	Alzheimer's disease is commonest	Essential to pass
	Frontotemporal dementia	Two others required to pass
	Multi-infarct dementia (vascular)	
POINTS REQUIRED	Parkinson's disease (Lewy bodies)	
	Creutzfeld-Jakob disease	
	Neurosyphilis, toxins etc	
SECOND QUESTION	Can you describe the pathogenesis of Alzheimer's disease?	
POINTS REQUIRED	Lysis of transmembrane protein Amyloid Precursor Protein by beta and gamma secretases produces Aβ & C-terminal portion of APP	Required amyloid generation
FOURTH QUESTION	Aβ peptides aggregate into amyloid fibrils and can be directly neurotoxic	Additional information
	C-terminal portion of APP involved in cell signalling and transcription regulation	Additional information
	Severity of Alzheimer's disease is related to loss of synapses	Important fact

Intracranial Haemorrhage 2017-2-C

Stem: Moving on to Pathology. The patient has an intracerebral haemorrhage.			
Question 5 Intracerebral haemorrhage **Subject**: Pathology LOA: 2 Robbins and Cotran's Pathologic Basis of Disease; 9th Edition; Chapter 28: The Central Nervous System; Page 1268	(a) What are the main pathophysiological causes of spontaneous intracerebral haemorrhage?	**Hypertension and cerebral amyloid** are the main causes. Other causes include systemic coagulation disorders, neoplasms, vasculitis, aneurysms, and vascular malformations.	**1 of 2 bold** and two others to pass
	(b) Which areas of the brain do hypertensive intracerebral haemorrhages most commonly occur?	Hypertensive intracerebral haemorrhage may originate in the putamen (50% to 60% of cases), thalamus, pons, cerebellar hemispheres (rarely) Accept basal ganglia, brainstem	At least two to pass.
	(c) Describe the pathophysiology of cerebral amyloid angiopathy? (Bonus question)	There is deposition of amyloidogenic peptides in the walls of medium- and small-calibre meningeal and cortical vessels. This deposition can result in weakening of the vessel wall and risk of haemorrhage	

Intracranial Haemorrhage 2013-1

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

Meningitis 2015-1-A

Stem: These are his CSF results.			
Clinical Building Block:	What is the likely diagnosis and why?	Turbid, low sugar, high protein, pleocytosis with neutrophil predominance, no bacteria **Acute bacterial meningitis**	Diagnosis + 2 reasons
Stem: Moving onto Pathology.			
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.

Meningitis 2010-1

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

Meningitis 2007-1

3. Meningitis	1. What are the different types of meningitis?	**1. Infectious: bacterial, aseptic/viral**, chronic (TB, spiro, crypto) Chemical: nonbacterial irritant Ca, lymphoma	Point 1 + 1 other
	2. What organisms commonly cause bacterial meningitis?	Neonates: E coli, **grp B strep** Children: **Strep pneu**, H flu reducing with vaccines Young adults: **Neisseria meningitidis**, Elderly: **Strep pneum**, Listeria Immunosuppressed: Klebsiella, anaerobes	3 highlighted + 1 other
	3. How do the cerebrospinal fluid findings typically differ between acute bacterial and viral meningitis?	Bact: cloudy, higher pressure, ***more neutrophils**, **raised protein**, ***reduced glucose** Viral: ***lymphocytes, moderately raised protein, *normal glucose**	Must say protein, glucose, cells

Meningitis 2003-1

TOPIC: Non Bacterial Meningitis **NUMBER:** ____

OPENING QUESTION	What are the non-bacterial causes of meningitis	**COMMENTS**
POINTS REQUIRED	1 Viruses	Viruses + 2
	2 Fungi	
	3 Chemical	
	4 Malignant	
	5	
	6	
	7	
PROMPTS		
SECOND QUESTION (if needed)	What are the typical CSF findings in Viral Meningitis	
POINTS REQUIRED	1 Lymphocytes	3 of 4
	2 Normal Glucose	
	3 Mildly elevated Protein	
	4 No bacteria seen	
	5	
	6	
PROMPTS		
THIRD QUESTION (if needed)	What are the common viral causes	
POINTS REQUIRED	1 Echo	2 of 4
	2 Coxsackie (enteroviruses)	
	3 Non paralytic Polio	
	4 Others	
	5	
	6	
PROMPTS		

Multiple Sclerosis 2009-2

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
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

Multiple Sclerosis 2007-1

4. Multiple sclerosis	1. What is multiple sclerosis?	**autoimmune demyelinating** disorder characterised by distinct episodes of neurological deficits separated in time, attributable to white matter lesions that are separated in space.	Highlighted
	2. Describe the pathogenesis of multiple sclerosis.	a. **Cellular immune response inappropriately directed against components of myelin sheath**. (Not clear how cellular reaction initiated - ? infection +/- genetics; CD4 and TH1 T-cells; Secrete cytokines…activate macrophages; Activated macrophages and products cause demyelination) b.**Genetic and environmental influences** (greater incidence in first degree relatives)	a & b
	3. What are the typical cerebrospinal fluid changes found in multiple sclerosis?	a.Elevated protein, b.Mild pleocytosis (33%), c. Increased gamma globulin, d.Oligoclonal bands (B cell proliferation)	At least 2

Parkinson's Disease 2010-2

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

Peripheral Nerve Repair 2003-1

TOPIC: Peripheral Nerve Repair **NUMBER:** _____

OPENING QUESTION	Describe the process of peripheral nerve repair following traumatic injury	**COMMENTS**
POINTS REQUIRED	1 Death of distal part (+/- some of proximal)	
	2 Axonal Cone of Growth 1-2 mm per day	
	3 Growth through Schwann cell structure	
	4 regenerating Clusters	
	5	
	6	
	7	
PROMPTS	What would you see microscopically?	

Spinal Cord Injury 2015-2-B

Stem: Moving onto Pathology			
Question 5 Spinal Cord injury including cellular injury as it relates to spinal cord **Subject:** Path LOA: 2 and 1	(a) What changes occur in the spinal cord after a traumatic injury	**Acute phase :** haemorrhage, necrosis, axonal swelling in the surrounding white matter at level of injury **Late phase :** area of neuronal destruction becomes cystic & gliotic, 2° wallerian degeneration involving long white matter tracts, liquefactive necrosis often seen in CNS	1 acute, 1 late
	(b) What are the features of irreversible injury at the cellular level	**Mitochondrial damage:** Failure of oxidative phosphorylation → **ATP depletion** → failure of energy dependent cellular functions **Membrane damage:** Plasma membrane → loss of osmotic balance Lysosomal membrane → **enzyme leakage** → cell necrosis	3 out of 4 bold
	(c) What are the acute clinical consequences of a cervical spinal cord injury Prompt: what happens in a high cervical level injury?	Complete or incomplete Spinal shock - **Quadriplegia**/flaccid paralysis, total anaesthesia, areflexia If above C4 → **respiratory compromise** (diaphragmatic paralysis) Neurogenic shock : hypotension, bradycardia, warm dry skin etc Incomplete syndromes, eg anterior cord, central cord etc	Bold

Subarachnoid Haemorrhage 2011-1

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

Subarachnoid Haemorrhage 2007-1

4. SAH	1. What is the most frequent cause of clinically significant subarachnoid haemorrhage?	rupture of a **saccular (berry) aneurysm**	
	2. Where are saccular aneurysms commonly located?	40% ant comm art, 34% middle cerebral art, 20% int carotid/PICA, 4% Basilar/Posterior Cerebral (most likely to cause problems with vasospasm)	At least anterior circulation to pass
	3. What is the aetiology of saccular aneurysms? Prompt: "What are the risk factors for saccular aneurysms?	1. **Generally unknown**, 2. Not 'congenital', 3. Some genetic risk (Polycystic kidney, Ehlers Danlos type 4, Neurofibromatosis type 1, Marfans), 4. Predisposing factors (Smoking, Hypertension)	At least 2
	4. What are the consequences of subarachnoid haemorrhage?	1. Early (Vasospasm and additional ischemic injury) 2.Later (Meningeal fibrosis & scarring, CSF obstruction)	At least 1 early, 1 late

Traumatic Brain Injury 2015-1-D

Stem: Moving onto Pathology			
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?	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.**	Bridging **veins**
	2. Which groups of patients are most at risk for SDH and why?	2. **Elderly**- veins stretched and more movement due to brain atrophy Infants- thin walled bridging veins	Elderly
	3. How does an extradural haematoma occur?	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.**	**Meningeal (often middle) artery**
	4. Define and describe diffuse axonal injury?	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.**	**Microscopic damage to deep brain white matter**