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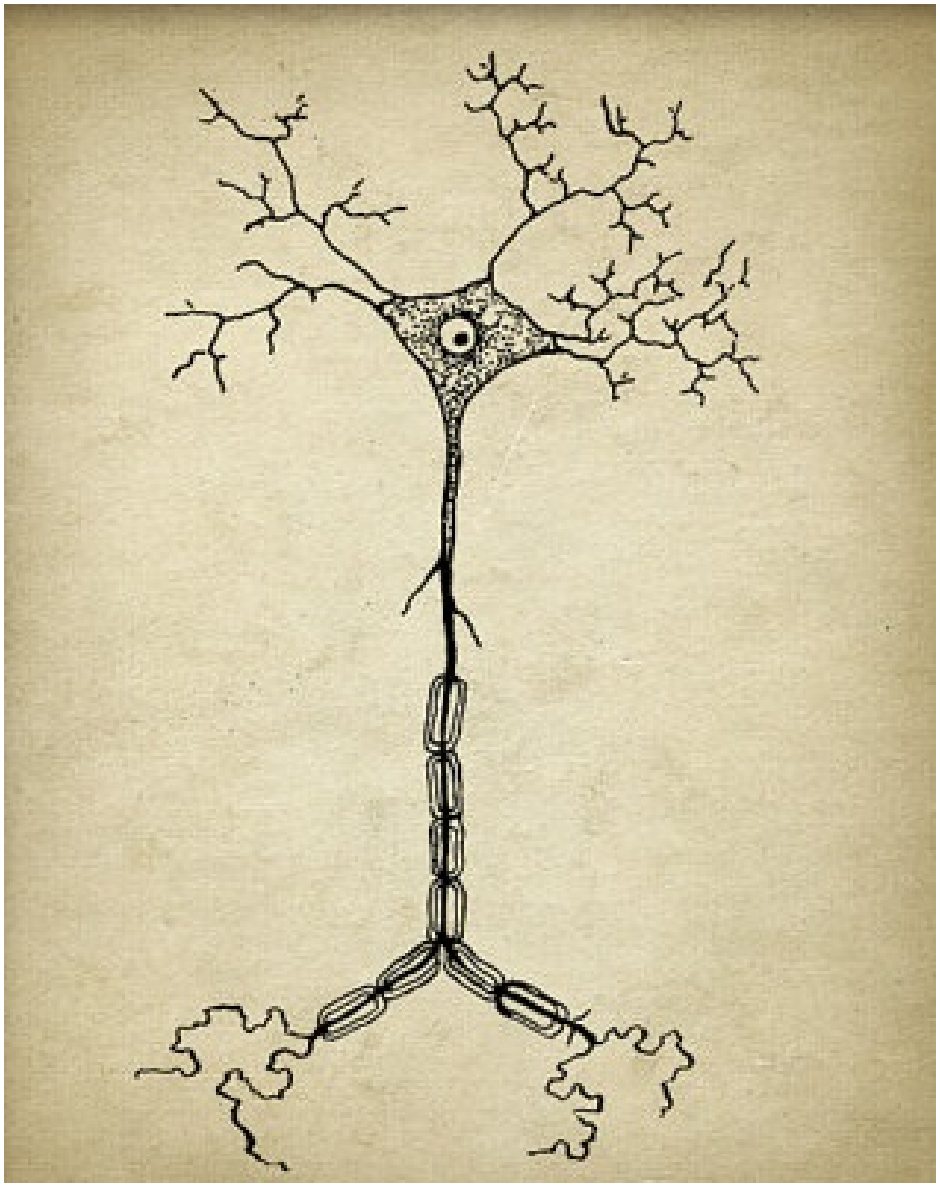

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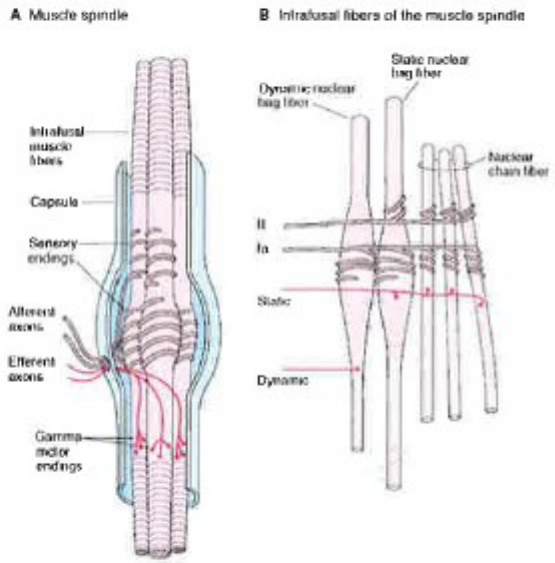
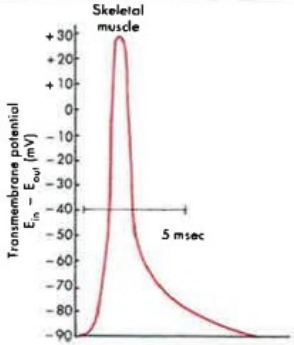
Muscle Spindle 2012-2

Question 4 Stretch rflx LOA: 2	1. Describe or draw the components of a muscle spindle.	Various psychological stimuli **In parallel intrafusal muscle fibers** (3 types – dynamic nuclear bag, static nuclear bag and nuclear chain); **sensory nerve endings** (Group Ia afferent to all and efferent axons, Group II to nuclear chain and static nuclear bag); dynamic gamma **motor nerves** to dynamic bag fibers, static gamma motor nerves (to static nuclear bag and chain fibers).  	**Bold to pass** **Must mention 3 of 5 bold**
	2. Describe the sequence of events involved in producing a stretch reflex.	Sequence: stimulus (muscle **stretch**); muscle; sensory organ (muscle **spindle**) within the muscle body; efferent **sensory** nerve; **synapse** in spinal cord to **motor** neuron supplying same muscle. Transmitter (glutamate).	

Skeletal Muscle Contraction 2015-2-D

Stem: Moving onto Physiology.			
Question 3 Skeletal Muscle action potential **Subject:** Phys LOA: 1	Draw a skeletal muscle action potential **(**Prompt if draw cardiac musc AP**)**		**Correct shape, axes, resting membrane potentials and durations (+/- 25%).**
	What is the sequence of events in the contraction of a skeletal muscle fibre, starting at the motor end-plate?	1.Discharge of motor neuron 2.Release of transmitter (acetylcholine) at motor endplate 3.Binding of ACh to Nicotinic Ach receptors 4.Increased Na^+ and K^+ conductance in end plate membrane 5.Generation of end plate potential 6.Generation of action potential in muscle fibers 7.Inward spread of depolarisation along T tubules 8.Releases of Ca^{2+} from terminal cisterns of sarcoplasmic reticulum and diffusion to thick and thin filaments 9.Binding of Ca^{2+} to troponin C, uncovering myosin-binding sites on actin 10.Formation of cross-linkages between actin and myosin and sliding of thin on thick filaments, producing movement	**5/10** to pass
	What is the sequence of events in the relaxation of a skeletal muscle fibre?	1.**Ca^{2+} pumped back into sarcoplasmic reticulum** 2.Release of Ca^{2+} from troponin 3.**Cessation of interaction between actin and myosin**	**Bold to pass**

Skeletal Muscle Contraction 2012-1

Question 5 LOA: 1	What is the sequence of events in skeletal muscle excitation contraction coupling?	**Discharge of motor neuron.** **Release of transmitter (acetylcholine) at motor end-plate.** **Binding of acetylcholine to nicotinic acetylcholine receptors.** Increased Na+ and K+ conductance in end-plate membrane. **Generation of end-plate potential.** **Generation of action potential in muscle fibers.** Inward spread of depolarization along T tubules. **Release of Ca2+ from terminal cisterns of sarcoplasmic reticulum and diffusion to thick and thin filaments.** **Binding of Ca2+ to troponin C, uncovering myosin-binding sites on actin.** ATP dependent **Formation of cross-linkages between actin and myosin and sliding of thin on thick filaments, producing movement.**	Need bold to pass

Skeletal Muscle Contraction 2009-2

Question 4:	**What are the sequence of events in contraction and relaxation of a skeletal muscle?** **Prompt: what about relaxation**	1)Motor neurone d/c + Ach presyn release 2)AcH to post syn- Nicotinic receptors ↑ Na/K in end plate generates AP along muscle fibre 3) T tubules spread depolarisation releases Ca++ from sarcoplasmic reticulum (terminal cisterns) 4) ↑ Ca around myosin/actin filaments, to TropC uncovers myosin binding sites on Actin 5) 5) X-links form thin/ thick – shorten as slide **Relaxation** Ca pumped out, trop C reactivated and blocks actin/myosin bind.	Pass /FailShould have 3 /5 steps mentioned with some detail and know active Ca ++ reverses for relation

FIRST QUESTION	**Describe the sequence of events in contraction and relaxation of skeletal muscle.**	
POINTS REQUIRED	Steps in contraction **(1) Discharge of motor neuron.** **(2) Release of transmitter (acetylcholine) at motor end-plate.** **(3) Binding of acetylcholine to nicotinic acetylcholine receptors.** (4) Increased Na+ and K+ conductance in end-plate membrane. **(5) Generation of end-plate potential.** **(6) Generation of action potential in muscle fibers.** (7) Inward spread of depolarization along T tubules. **(8) Release of Ca2+ from terminal cisterns of sarcoplasmic reticulum and diffusion to thick and thin filaments.** **(9) Binding of Ca2+ to troponin C, uncovering myosin-binding sites on actin.** **(10) Formation of cross-linkages between actin and myosin and sliding of thin on thick filaments, producing movement.**	Bolded at least?
	Steps in relaxation **(1) Ca2+ pumped back into sarcoplasmic reticulum.** (2) Release of Ca2+ from troponin. **(3) Cessation of interaction between actin and myosin.**	Bolded at least
PROMPTS		

SECOND QUESTION	**What is summation of contractions?**	COMMENTS
POINTS REQUIRED	1. The electrical response of a muscle fibre to repeated stimulation.	
	2. Contractile mechanism does not have a refractory period, so repeated stimulation before relaxation has occurred produces additional activation and a response added to the contraction already present.	
	3. With rapidly repeated stimulation, individual responses fuse into one continuous contraction (tetanus; tetanic contraction).	
	4. Complete tetanus: no relaxation between stimuli; tension developed ~ 4 times that of an individual twitch contraction	
	5. Incomplete tetanus: periods of incomplete relaxation between summated stimuli	
PROMPTS	Describe the response of a muscle fibre to repeated stimulation. What is a tetanic contraction?	

Skeletal Muscle Contraction 2007-2

Question	***Required response [Key items marked with*]***	***To Pass***
Describe the sequence of events in the contraction of skeletal muscle after discharge of the motor neurone.	*1 Discharge of motor neuron* *2 Release of ACh at motor endplate *** *3 ACh binds nicotinic ACh receptors* *4 Increase in Na and K conductance in end plate membrane* *5 end plate potential* *6 Muscle action potential* *7 Depolarization along T tubules* *8 Ca release at SR* *9 Ca binds Trop C and uncovers myosin binding sites on actin *** *10 actin myosin cross links and thin filaments slide on thick ***	Release Ach Ca release and Trop C bind actin myosin link and slide
How does tetanic contraction occur? *How does this differ from Treppe ? (Differentiation)*	*Contractile mechanism has **no refractory period**.*** *Repeated stimulation before relaxation has occurred - **summation** of contractions* *Fast repeated stimulation causes a fused continuous tetanic contraction. Can be complete or incomplete.* *Series of maximal stimuli at a frequency just below tetanizing causes increasing tension between each twitch. Due to increased calcium availability.*	*Describe tetanic contraction.*
What are the major differences in types of skeletal muscle?	*Type 1* *slow oxidative red,* *Moderate Ca2+ pumping,diameter and glycolytic capacity* *Slow myosin ATPase rate. High oxidative capacity.* *Type 11* *fast glycolytic white.* *High Ca2+ pumping,diameter and glycolytic capacity* *Fast myosin ATPase rate. Low oxidative capacity*	*Know two types and three differences*

TOPIC: Smooth muscle contraction _____ **NUMBER:** _____

OPENING QUESTION	Describe the sequence of events in contraction and relaxation of visceral smooth muscle.	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Binding of ACh to muscarinic receptors	Is there a difference between smooth muscle and other muscle?	
	2 Increased influx of Ca^{2+} into the cell		Essential
	3 Activation of calmodulin-dependent myosin light chain kinase		Essential
	4 Phosphorylation of myosin		
	5 Increased myosin ATPase activity and binding of myosin to actin		
	6 Contraction		
	7 Dephosphorylation of myosin light chain phosphatase		
	8 Relaxation or sustained contraction due to latch bridge and other mechanisms		
SECOND QUESTION	What factors influence intestinal smooth muscle contraction?		
POINTS REQUIRED	1 Stretch of visceral smooth muscle causes contraction in the absence of innervation		Essential
	2 Cold increases activity		
	3 ACh decreases smooth muscle potential and increases spike frequency so resulting in more active muscle		
	4 Adrenaline and noradrenaline increase smooth muscle potential and decrease spike frequency causing decreased muscle activity		
	5 Neural		

Acetylcholine 2017-1-B

<table>
<tr><td colspan="4">Stem: Moving onto Physiology. He is tachycardic and has a dry mouth.</td></tr>
<tr><td>Question 3
Cholinergic neurotransmission

Subject: Phys

LOA 1</td><td>a) Please describe the synthesis, release and action of acetylcholine at a nerve synapse.
You may draw a diagram

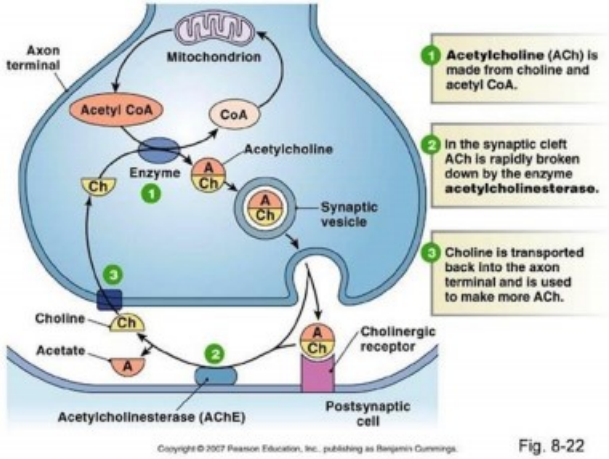
b) Once acetylcholine is released into the synaptic cleft, how is its effect terminated?</td><td> 

Fig. 8-22

Diffusion
Acetylcholine esterase
Re-uptake of choline into presynaptic nerve terminal</td><td>Key sequence to pass.
- Synthesis: acetyl CoA and choline
- Release from the synaptic vesicle
- Bind to post-synaptic receptor

Bold to pass.</td></tr>
</table>

Acetylcholine 2011-2

Question 5 LOA: 1	1) What happens to acetylcholine when released into a synapse?	• Binds to post-synaptic cholinergic receptors • Catabolism by acetylcholinesterase at the postsynaptic membrane • Reuptake of choline • No acetylcholine reuptake • Catabolism by pseudocholinesterase in the circulation	3/5 to pass
	2) Describe the differences between the two types of acetylcholine receptors.	• Divided on basis of pharmacological properties into muscarinic and nicotinic • Muscarinic–actions mimicked by muscarine and blocked by atropine. Found in smooth muscle, glands and brain. G-protein coupled to adenylyl cyclase and/or phospholipase. • Nicotinic–actions mimicked by nicotine. Found in neuromuscular junction, autonomic ganglia and the central nervous system. Ligand-gated sodium ion channels.	Necessary to pass Two sites to pass Two sites to pass

Acetylcholine 2006-2

TOPIC: Physiology of acetylcholine **NUMBER: 3**

OPENING QUESTION	Describe the biochemical events involving acetylcholine at a cholinergic nerve ending?	**PROMPTS CO**	**MMENTS**
POINTS REQUIRED	1 Choline - synthesized in neurons - active uptake into cholinergic neuron via a transporter	1	Pass = 3 bold plus 1
	2 Acetate activated by combination with coenzyme A	2	
	3 Acetyl-coenzyme + Choline forms ACh	3	
	4 **Catalysed by Choline acetyltransferase** found in high concentrations in cytoplasm of cholinergic nerve endings.	4	
	5 ACh then taken up into synaptic vesicles via transporter (VAChT)	5	
	6 Release ACh	6	
	7 ACH rapidly removed from synapse to allow repolarisation	7	
	8 **ACH to Choline + Acetate by acetyl cholinesterase** (clustered in post synaptic membrane of cholinergic synapse)	8	
SECOND QUESTION (if needed)	Describe the differences between the main types of acetylcholine receptors?		Pass = bold plus 1
POINTS REQUIRED	1 **Divided on basis of pharmacological properties into Muscurinic and Nicotinic**	1	
	2 **Muscurinic** **- mimic by muscurine** **- block by atropine** - smooth muscle, glands and brain (both) - 5 types (5 genes) - serpentine receptors coupled via G protein - M1 brain, M 2 heart, M 3 and 4 smooth muscle, M 4 pancreas, M 5 ?	2	
	3 **Nicotinic** **- mimic by nicotine** - 2 types – NMJ and autonomic ganglia & CNS - 5 subunits (16 genes) - alpha, beta, gamma, delta, epsilon - binding site Ach on alpha subunit - member superfamily ligand gated ion channels - ACH binding opens Na channel - subunit structure differs location (gangli vs brain) & age (foetus vs adult) -	3	

SUBJECT: PHYSIOLOGY Session 2 Question 3

TOPIC: _____ **NUMBER:** _____

OPENING QUESTION	Please describe the synthesis and release of acetyl choline at a nerve synapse. You may draw a diagram	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1	1	
	2	2	
	3	3	
	4	4	
	5	5	
	6	6	
SECOND QUESTION (if needed)	Once it is released, how is the effect terminated?		
POINTS REQUIRED	1 Diffusion	1	
	2 Acetylcholinesterase	2	
	3	3	
	4	4	
	5	5	
	6	6	
	7		
THIRD QUESTION (if needed)	What types of cholinergic receptors are there?		
POINTS REQUIRED	1	1	
	2	2	
	3	3	
	4	4	
	5	5	
	6	6	
	7		

Catecholamines 2011-2

Question 4 LOA: 1	1) Outline the biosynthesis of adrenaline. 2) How is the action of noradrenaline terminated?	**Tyrosine**-tyrosine *hydroxylase* – **DOPA** (dihydroxy-phenylalanine)-amino acid *decarboxylase*- **Dopamine**-dopamine *hydroxylase*- **Noradrenaline**- phenylethalonamine *methyltransferase* **Adrenaline** a) **reuptake** presynaptic neuron than metabolised by MAO to inactive deaminated derivates or recycled b) **catabolised synaptic cleft** by COMT (catechol methyltransferase) to Normetanephrine	Bold to pass Prompt: How is adrenaline synthesised from tyrosine? Bold to pass

Catecholamines 2010-1

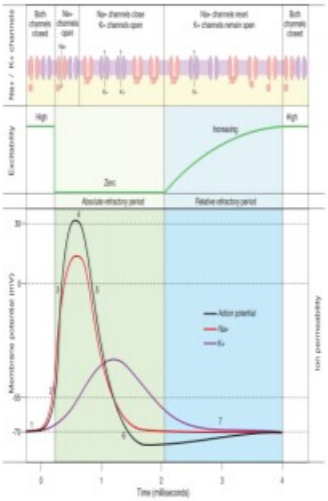
4 a).Outline the steps in the synthesis of catecholamines	**TyOHylase** **Tyrosine→ DOPA** **↓ DOPA** **Decarboxylase** **Dopamine** **↓ Dopamine** **βhydroxylase** **Adrenaline← Nor Adrenaline** **PNMT (adrenal medulla, some central)** **Adrenaline/Noradrenaline**	**Tyrosine to dopamine to noradrenaline, plus one of the synthesis enzymes**
4 b).What happens to noradrenaline after it is released into the synaptic cleft?	**Removed by post-synaptic and pre-synaptic binding, reuptake and catabolism** **IC) MAO ↓ COMT (EC)** **VMA**	**Three out of four processes**
5. Describe the renal response to		

Catecholamines 2007-2

QUESTION: 5. Catecholamines

Question	Required response [Key items marked with*]	To Pass
Which catecholamines act as neurotransmitters?	*Noradrenaline, *Adrenaline and Dopamine	* to pass
Describe the sequence of events at a **noradrenergic synapse**, following stimulation of a sympathetic nerve. *Prompts:* *How is noradrenaline released?* *How is noradrenaline removed from the synaptic cleft?* *What enzymes are involved in the breakdown of noradrenaline?*	Noradrenaline, which has been **stored in granulated** ***vesicles**, is released into the synaptic cleft by ***exocytosis.** Noradrenaline acts on postsynaptic and to a lesser extent presynaptic and glial receptors. In addition to binding to receptors, Noradrenaline is also removed from the synaptic cleft by: • ***Reuptake** into presynaptic neuron (via a Neuro Transmitter Transporter (NTT)) and then is broken down to inactive product by Monoamine Oxidase (MAO) located on mitochondria • **Broken down** to inactive product by Catechol-O-methyl transferase (COMT) located on the postsynaptic membrane	* to pass

Nerve Action Potential Conduction 2017-1-D

Stem: Moving onto Physiology. She has a nerve injury.			
Question 4 Nerve action potential and excitation **Subject:** Phys: LOA: 1	a) Please draw a nerve action potential and indicate the sequence of events that occur	It depends on the change in conductance of Na and K ions. 1. When a **depolarising stimulus** occurs, the voltage-gated **Na channels** become active, Na enters the cell 2. When the **threshold potential** is reached the voltage-gated Na channels overwhelms the K channels. 3. Entry of Na causes opening of more voltage-gated Na channels and further depolarisation (positive feedback loop) resulting in the upstroke of AP 4. The membrane potential moves close to the equilibrium potential for Na (+60mV). 5. The voltage gated Na channels then enter an inactivated state for a few milliseconds before returning to the resting state 6. Reversal of membrane potential limiting further Na influx and **opening of voltage-gated K channels** results in **repolarisation** and end of AP 7. Slow return of K channels results in hyperpolarization 8. Returns to resting membrane potential	Bold to pass 

Nerve Action Potential Conduction 2007-2

QUESTION: 4. Nerve excitation / conduction

Question	**Required response [Key items marked with*]**	**To Pass**
Draw and label an action potential of a neuron. Prompt: What are the phases of a nerve action potential?	1 Latent period at −70 mV then upslope until firing level is reached at −55mV. 2 Spike potential with overshoot to +35 mV. 3 Rapid repolarization then slow after-depolarization. 4 After-hyperpolarization beyond −70 mV. 5 Return to latent period.	Shape From negative to positive
What ionic fluxes occur during the action potential? Prompt: What ions are involved in nerve conduction?	1 At firing level, rapid influx of Na towards equilibrium (+ 60 mV). 2 Na channels rapidly close (inactivated state) and Na inhibits further Na influx. 3 Voltage-gated K channels open. 4 Slow K efflux completes repolarization. 5 Decrease (increase) in extracellular Ca decreases (increases) the Na and K conductance required for an action potential.	Na influx (depol) K efflux (repol)
Where are ion channels distributed in myelinated neurons?	1 Voltage-gated Na channels concentrated in **node of Ranvier** and initial segment. 2 Na channels flanked by K channels	Nodes of Ranvier

Nerve Action Potential Conduction 2005-1

Nerve action potential	Draw a nerve action potential. What are the ion fluxes that occur during an action potential?	Resting membrane potential (-70mV); firing potential (-55mV); depolarises to positive level (+35mV) (Concept, not exact figures) Fast sodium influx; slow potassium efflux	

Nerve Action Potential Conduction 2003-1

TOPIC: Conduction along a nerve cell _____

OPENING QUESTION	Draw & label the action potential in a nerve cell.	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. Resting membrane potential	1	
	2. Movement of sodium potassium ions.	2	
	3.	3	
SECOND QUESTION (if needed)	Discuss the factors that affect conduction.		2/4
POINTS REQUIRED	1. Myelinated vs demyelinated	1	
	2. Saltatory vs non-saltatory	2	
	3. Size	3	
	4. Direction of the conduction	4	

Nerve Fibres 2011-1

Question 5	5.1 What are the different types of nerve fibres? PROMPT – What classifications are there?	• Diameter & speed of conduction • Function Large, fast – proprioception, conscious touch, somatic motor Small, slow – pain , temperature, autonomic • Gasser ABC (A – αβγδ) • Numerical Ia, Ib, II,III IV	One system or concept of system
	5.2 What is the clinical relevance to emergency medicine?	Pain fibres are smaller and better penetrated by local anaesthetic leading to loss of pain before loss of touch or proprioception	One example

Neuromuscular Transmission 2005-1

Neuromuscular excitation contraction coupling	Describe the sequence of events in transmission of a motor nerve impulse to a muscle How does the muscle them become depolarised?	Motor neurone action potential; end-plate potential; Acetylcholine release; Ach binding to nicotinic receptors; muscle end-plate potential. T tubules and release of Ca^{2+} from sarcoplasmic reticulum.	

TOPIC: Neuromuscular transmission _____ **NUMBER:** _____

OPENING QUESTION	Describe the synthesis and release of acetyl choline at the neuro-muscular junction? You may draw a diagram.	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Acetyl choline formed From acetyl coenzyme A and choline, by enzyme Choline acetyl transferase at Presynaptic terminal Stored in synaptic vesicles With ATP and proteoglycan	1	
	2	2	
	3	3	
	4	4	
	5	5	
	6	6	
	7	7	
	8		
SECOND QUESTION (if needed)	Once it is released, how is the effect terminated?		
POINTS REQUIRED	1 Diffusion	1	
	2 Acetylcholinesterase	2	
	3	3	
	4	4	
	5	5	
	6	6	
	7		
THIRD QUESTION (if needed)			
POINTS REQUIRED	1	1	
	2	2	
	3	3	
	4	4	

Neuronal Inhibition 2010-1

5. a) In the synapse, where can inhibition occur? b)What are the mechanisms involved?	Post-synaptic: direct or indirect (refractory periods, after-hyperpolarisations) Pre-synaptic: mediated by neurons that end on excitatory endings (axo-axonal synapses). i Increased Cl^- conductance – reduces Ca^{2+} influx and amount of excitatory transmitter released ii. Voltage-gated K^+ channels – K^+ also decreases Ca^{2+} entry iii. Direct inhibition of excitatory transmitter release, independent of Ca^{2+} influx	Must give pre-synaptic and post-synaptic Must give reduction in Ca^{2+} influx

Neuronal Inhibition 2006-2

TOPIC: Neuronal inhibition _____ NUMBER: 5 _____

OPENING QUESTION	What are types of neuronal inhibition?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Presynaptic	1	Pass = 2/2
	2 Postsynaptic	2	
SECOND QUESTION (if needed)	What are types of postsynaptic neuronal inhibition?		Pass = 1/2
POINTS REQUIRED	1 Direct during the course of an IPSP and not the consequence of a previous discharge	1	
	2 Indirect due to the effect of a previous postsynaptic neuron discharge	2	
THIRD QUESTION (if needed)	What is Presynaptic inhibition?		
POINTS REQUIRED	1 neurons that end on an excitatory ending forming an axo-axonal connection	1	Pass = 1/2
	Calcium, chloride and gabba are transmitters involved		

TOPIC: Synthesis and fate of catecholamines at synaptic junction **NUMBER:** _____

OPENING QUESTION	**Describe the biosynthesis and storage of norepinephrine at the synaptic junction.**	**COMMENTS**
POINTS REQUIRED	1. dietary tyrosine mostly (some formed from phenylalanine)	
	2. tyrosine transported into catecholamine-secreting neurones by concentrating mechanism	
	3. tyrosine → dopa by tyrosine hydroxylase [this is the rate-limiting step & is subject to feedback inhibition by dopamine and norepinephrine] → dopamine by dopa decarboxylase in cytoplasm	At least 4 in correct order
	4. dopamine enters granulated vesicles → norepinephrine by dopamine β-hydroxylase (DBH)	
	5. norepinephrine stored bound to ATP, with protein chromogranin A	
PROMPTS		
SECOND QUESTION (if needed)	**How is Norepinephrine removed from the synaptic junction?**	
POINTS REQUIRED	1. norepinephrine is removed from the synaptic junction by: i. binding to postsynaptic receptors ii. binding to presynaptic receptors iii. reuptake into presynaptic neurons iv. catabolism (MAO)	Bolded to pass
	2. catabolism at noradrenergic nerve endings is catalysed by MAO (monoamine oxidase) and COMT (COMT mainly in liver, also at postsynaptic noradrenergic nerve endings)	
	3. norepinephrine → DOMA (3,4-dihydroxymandelic acid) & DHPG (3,4-dihydroxymandelic aldehyde) → VMA (vanillylmandelic acid) & MHPG (3-methoxy-4-hydroxyphenylglycol) by systemic COMT. These deaminated derivatives are physiologically inactive.	
PROMPTS		

COMMENTS

Noradrenergic Neurotransmission 2007-2

QUESTION: 4. CHEMICAL TRANSMISSION OF SYNAPTIC ACTIVITY

Question	Required response [Key items marked with*]	To Pass
What are the steps in synthesis of noradrenaline at a nerve ending? *Prompt:* *What is noradrenaline made from?*	1 **Tyrosine transported in and converted to Dopa** by tyrosine hydroxylase **(rate-limiting)** in presence of tetrahydrobiopterin 2 **Dopa converted to Dopamine** by dopa decarboxylase 3 **Dopamine** enters granulated vesicles and **converted to Noradrenaline** by dopamine beta hydroxylase 4 Noradrenaline inhibits tyrosine hydroxylase (feedback inhibition)	
What happens to noradrenaline released into a synapse? *Prompt:* *How is the effect terminated?*	1 Binds to post-synaptic receptors 2 Binds to pre-synaptic receptors 3 **Reuptake** into pre-synaptic neurons 4 **Catabolism** by monoamine oxidase (A or B) (nerve endings) and catechol-o-methyl transferase (post synaptic membrane, liver, kidneys, muscles)	
What happens to acetylcholine released into a synapse? *Prompt:* *How does it differ from noradrenaline?*	1 **No acetylcholine reuptake** 2 **Catabolism by acetyl cholinesterase** 3 Reuptake of choline 4 Catabolism by pseudocholinesterase	SPARE

Noradrenergic Neurotransmission 2004-2

TOPIC: Noradrenergic neurotransmission _____ **NUMBER:** _____

OPENING QUESTION	Please describe the synthesis and release of noradrenaline at a synapse? You may draw a diagram.	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Draw synaptic nerve ending or describe	1	
	2	2	
	3	3	
	4	4	
	5	5	
	6	6	
SECOND QUESTION (if needed)	Once it is released, how is the effect terminated?		
POINTS REQUIRED	1 Diffusion	1	
	2 Reuptake	2	
	3 MAO	3	
	4 COMT	4	
	5	5	
	6	6	
	7		
THIRD QUESTION (if needed)	What types of noradrenergic receptors are there?		
POINTS REQUIRED	1 Alpha and beta	1	
	2	2	
	3	3	
	4	4	
	5	5	
	6	6	
	7		

Resting Membrane Potential 2015-2-B

Stem: Moving onto Physiology.			
Question 2 Resting Membrane Potential **Subject:** Phys LOA: 1	(a) Define resting membrane potential of a neuron	**Potential difference** across cell at rest, as a result of separation of positive and negative electronic charges across cell membrane (**inside negative** relative to outside of cell). Normal RMP of neuron = -70mV	Bold
	(b) Explain how resting membrane potential is created Prompt: Why is RMP negative on the inside of a cell?	Main ions involved – **Na^+ & K^+** **Na^+-K^+-ATPase pump** creates **electrochemical gradient** by pumping out 3 Na^+ for every 2 K^+ pumped in **Na^+ & K^+ diffuse down concentration gradient** across permeable cell membrane (K^+ diffuses from inside to outside of cell; opposite for Na^+) Cell membrane **more permeable to K^+** at rest → that's why RMP is close to equilibrium potential for K^+ RMP represents an equilibrium state; driving force for ions down concentration gradient = driving force down electrical gradient	Bold
	(c) Why is a cell more excitable in hyperkalaemia	RMP moves **closer to threshold potential for eliciting action potential** (becomes less negative on the inside of cell).	Bold

Resting Membrane Potential 2011-2

Question 5 LOA: 1	1.Describe the resting membrane potential of a cell.	There is difference in electronic charge across a cell membrane. The inside is negative compared to the outside. Resting MP results from separation of positive and negative charges across a cell membrane. Neuron average RMP -70 mV.	Bold to pass
	2.What conditions are required to create a resting membrane potential?	Lipid bilayer, unequal distribution of ions, membrane must be permeable to ions, concentration gradient.	2 out 4
	3. In a neuron what ions are involved and how is the concentration gradient produced?	Na and K. Na is primarily extracellular and K intracellular. Passive movement of ions occur via selective ion channels. Na-K ATPase actively move ions against their electrochemical gradient.	Bold to pass.

Resting Membrane Potential 2010-1

3a). What is the basis of the resting membrane potential?	a). Potassium – more open Potassium channels at rest therefore intracellular/extracellular Potassium concentrations are prime determinants of resting membrane potential b).Sodium c). Separated by the cellular membrane 1.Na actively transported out of cells 2.K actively transported into cells 3.Activity of Na-K ATPase pump	Na, K, ATPase and correct directions.
3b). Describe the ionic fluxes during the action potential	Voltage gated Na channels open (short lived), Na channels overwhelm K once threshold reached. Memb potential approaches Na (+60mV) . Na stops as short open phase, then they close/become inactivated, then resting state again. Also electrical gradient reversed. Then opening of volt gated K channels-slower and more prolonged than Na. Slow return to closed state causes after hyperpolarization	Na and K and sequence.
4 a). What is the normal	285-295mosm/l	

Serotonin 2009-1

Question 5: Serotonin (Ganong pp 106-107, 262-263)	i) What are the functions of serotonin?	a) Regulation of **vomiting reflex** b) Regulation of **mood** c) Control of respiration d) Platelet aggregation and smooth muscle contraction e) Facilitate GI secretion and peristalsis f) Regulation of circadian rhythms	Core knowledge in bold
	ii) What are the steps in synthesis and catabolism of serotonin?	i) Hydroxylation and decarboxylation of tryptophan to form serotonin ii) Released serotonin from serotonergic neurones is recaptured by an **active re-uptake mechanism and inactivated by MAO** to form 5HIAA iii) (5-hydroxyindoleacetic acid) iv) 5HIAA is excreted as a urinary metabolite	Core knowledge in bold

Synapse Depolarisation 2017-2-C

Stem: Moving on to Physiology. He has painful quadriceps muscle cramping in the injured limb.			
Question 5 Resting membrane potential **Subject**: Phys LOA: 1 *Ganong 25th Pp90, 130, 131, 14*	Define the resting membrane potential of a nerve	**Potential difference across the membrane** at rest with inside negative relative to outside In nerves, it is – 70 mV	**Bold**
	How is this resting membrane potential maintained?	The gradients are actively maintained by **NA^+/K^+ ATPase** NA^+/K^+ ATPase actively pumps Na out and K into the cell using ATPase for energy. Na then passively flows back into the cell via channels down concentration gradient, and K passively flows out of cell via K channels down concentration gradient. BUT at rest, there are more open K channels than Na channels, so the permeability to K is greater (passively).	**Bold** + concept Na out and K in Passive flow in opposite direction
	Describe the sequence of events that occur at the motor end plate following discharge of a motor neuron	Activation of voltage gated Ca2+ channels in presynaptic membrane Calcium influx into the cell Exocytosis of preformed ACh into synaptic cleft Diffusion of ACh across synaptic cleft Binds to post synaptic nicotinic receptor Increase Na+ and K+ conductance in end plate membrane (muscle) Generation of end plate potential. Generation of action potential in muscle fibres. Spread of depolarisation along T tubules. Ca++ released from Sarcoplasmic Reticulum (diffusion to thick and thin filaments). Binding of Ca++ to trop C uncovering myosin-binding sites on actin. Actin-myosin binding and sliding of thin on thick filaments producing movement	**6 of 12** steps
	Describe what occurs to the ACh released at the motor end plate?	ACh removed from synaptic cleft by Acetylcholinesterase Choline re-uptake Acetate to liver and metabolised	

CNS Autoregulation 2015-1-D

Stem: Her GCS has fallen to 8. We will now move onto Physiology			
Question 3 CNS Autoregulation / Cushing response Subject: Phys LOA: 1	1. What factors affect cerebral blood flow?	1. MAP at brain level MVP at brain level ICP Viscosity of the blood Local constriction/dilatation of cerebral arterioles	Pass in bold **3/5**
	2. What is the mechanism of the Cushing response?	2. Increase in ICP results in Decr CBF – ischaemia of VMA – SNS output incr - Incr systemic BP – stimulation of baroreceptors – stimulation of vagal outflow – decr HR and RR	**Explains concept**
	3. What is the Monro-Kellie doctrine?	3. The volume of blood (75mL), CSF (75mL) and brain (1400g) in cranium must be relatively constant. Negative effects on these therefore if additional intracranial volume eg SDH / EDH occurs	**Explains concept**

Deafness 2013-1

Question 5 Hearing LOA: 2	a. What are the two major mechanisms of deafness?	**Conductive deafness** – due to impaired sound transmission in external or middle ear, affects all frequencies. **Sensorineural deafness** – due to loss of cochlear hair cells (commonest), or problems with CN VIII or within central auditory pathways, affects some frequencies.	**Bold** Explain both and 2 examples of each to pass
	b. Explain these causes in physiological terms and give examples.	**Examples** Conductive – blockage of extl canals (e.g. wax, FBs), otitis ext or media, perforated eardrum, osteosclerosis Sensorineural – degeneration (presbycusis), damage to outer hair cells (prolonged noise exposure), aminoglycoside antibiotics, CN VIII tumours or cerebellopontine angle, CVA in medulla.	
	Bonus: How can one differentiate between the two forms using a tuning fork?	Weber/ Rinne : 256 tuning fork	Bonus if have time

Fever 2016-1-C

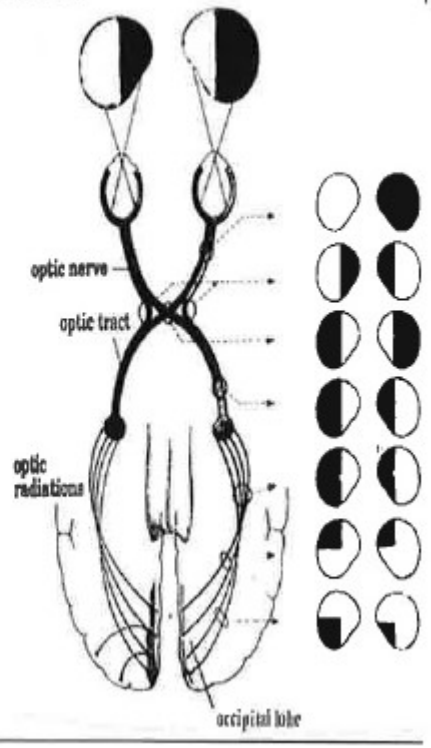
Stem: Moving onto Physiology. The patient is febrile.			
Question 4 Temperature regulation **Subject:** Phys LOA: 1	1. What is the pathogenesis of fever (Prompt: What causes a febrile response?) (Prompt: Which area of the brain is activated in a febrile response?)	Bacterial **toxins** eg endotoxin act on monocytes, macrophages, and Kupffer cells to produce cytokines that act as **endogenous pyrogens** IL-1β, IL-6, IFN-β, IFN-γ, and TNF-α can act independently to produce fever. Cytokines also produced by cells in CNS when these are stimulated by infection -may act directly on the thermoregulatory centres. Activates the preoptic area of the **hypothalamus**. Causes release of **prostaglandins** eg PGE2. This causes a raise in **temp set point** resulting in fever	Concept to pass
	2. What is the body's response to hot and cold environments? (Prompt: What happens to heat production & loss?)	Mechanisms activated by **cold:** (post hypothalamus) **Inc Heat Production:** Shivering, Hunger, Voluntary activity, NA/A release **Dec Heat Loss,** Skin vasoconstriction, Curling up, Horripilation. Mechanisms activated by **heat**: (ant hypothalamus) **Dec Heat Production** Anorexia, Apathy & Inertia. **Inc Heat Loss,** Cutaneous vasodilation, Sweating, Respiration.	1 mechanism for each of heat production & loss for cold 1 mechanism for each of heat production & loss for hot environment

Nystagmus 2007-2

QUESTION: 5. VESTIBULAR FUNCTION

Question	Required response [Key items marked with*]	To Pass
What is nystagmus ? *Prompt:* *Are there different types ?*	Characteristic jerky movement of the eye seen at start & end of period of rotation * Different types * - horizontal (eyes move horizontal plane) - vertical (head tipped sidewise in rotation) - rotatory (head tipped forward) Direction of eye movement is identified by the direction of the quick component.	Definition Horizontal plus one other
Why does nystagmus occur ? *Prompt:* *Why do the eyes move ?*	Reflex that maintains visual fixation on stationary points while the body rotates, although not initiated by visual impulses. - When rotation starts, the eyes move slowly in a direction opposite to the direction of rotation, maintaining visual fixation * (vestibulo-ocular reflex VOR). - When the limit of this movement is reached, the eyes quickly snap back to a new fixation point and then again move slowly in the other direction. *	General relationship of eye movements in relation to head movement
How is nystagmus mediated ?	Slow component is initiated by impulses from the labyrinths * Quick component is triggered by a centre in the brain stem. *	

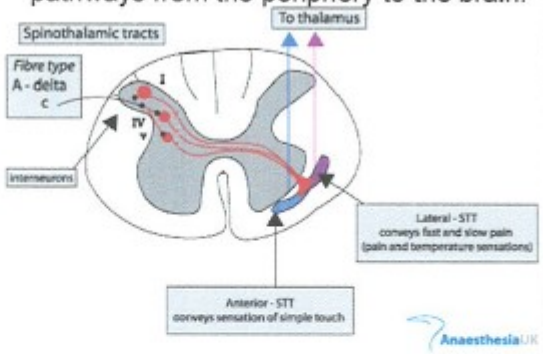
Optic Pathways 2012-2

Question 4 LOA: 2	4.1 Describe the neural connections of the visual pathways?	1.Retina – **optic n – optic chiasm –optic tract** - lateral geniculate body (thalamus) – geniculocalcarine tract – **primary visual cortex (occipital lobe,** Brodmann 17) **(Bold to pass)** Other connections a) lat geniculate nucleus to pretectal midbrain and sup colliculus (papillary refexes, eye movement) b) to frontal cortex (refined eye movement-vergence, near point response c) optic chiasm to thalamic suprachiasmatic nucleus (endocrine and circadian responses to day/night cycle)	Visual Pathway Diaphragm – looking from above, R side lesions 
	4.2 Describe the visual field defects of nerve sectioning at optic chiasm and optic tract on the right.	2. See diagram. **Both to pass**	

Pain Conduction 2012-2

Question 5 Pain and its Modulation LOA: 2	5.1 Describe how pain is transmitted from the periphery to the brain	a. sense organ = **naked nerve endings** b. transmission via 2 fibre types - small, fast myelinated **A-delta fibres** - large slow unmyelinated **C fibres** c. spinal cord: both fibre groups end in **dorsal horn of spinal cord ("gate")** - A-delta fibres on neurons in laminas 1&4 -C fibres on laminas 1&2 d. from spinal cord to brain via **ventrolateral system – second order)** (including lateral spinothalamic tract) to **thalamus** and then third order neurons on to **cerebral cortex**	Must mention dorsal horn of spinal cord and at least 3 others of bold to pass
	5.2 How can acute pain be modulated?	a. "gate theory" : eg stimulation of large touch/pressure afferents causes inhibition of pain pathways in dorsal horn of spinal cord b. Stress-induced analgesia c. Drugs (eg opioids) d. Higher centre interpretation	Must get 'gate theory' + 1 other
	5.3 What sites do opioid peptides act on?	a. receptors in afferent nerve fibres b. dorsal horn region of spinal cord c. periaqueductal grey matter in brain	Supplementary Question if answers above

Pain Conduction 2009-1

Question 5: Ganong pp 139-147	i) Describe the route followed by pain pathways from the periphery to the brain.  ii) What are the characteristics of the different types of pain fibres?	i) Primary efferent fibres - naked nerve endings peripherally - cell bodies in dorsal root ganglia (or equivalent in cranial nn.) - terminate on neurons in **dorsal horns** (Aδ fibres in laminae I and V, C fires in laminae I and II) Axons from dorsal horns travel in **anterolateral system** (**lateral spinothalamic tract**) to ventricular posterior nuclei (specific sensory relay nuclei of thalamus) and thence to cerebral cortex. ii) Aδ: **Myelinated** Large diameter (2-5 microm) **Fast** conduction rates (12-30 m/s) Modulate "fast" pain C: **Unmyelinated** Small diameter (0.4-1.2 microm) **Slow** conduction rates (0.5-2 m/s) Modulate "slow" pain	Core knowledge in bold.

TOPIC: Physiology of pain **NUMBER: 3**

OPENING QUESTION	Describe the characteristics of nerve fibers responsible for transmission of "fast pain"?	**PROMPTS**	**COM MENTS**
POINTS REQUIRED	1 **myelinated** A delta fibers	1 what types of nerve fibers transmit painful stimuli ?	Pass = myelinated plus 2/4
	2 2-5 um diameter	2	
	3 conduction rates 12-30 m/s	3	
	4 end in dorsal horn (lamina 1 and 5)	4	
	5 neurotransmitter is glutamate	5	
SECOND QUESTION (if needed)	What differences are there between these nerve fibers and those responsible for transmission of "slow" or second pain?		
POINTS REQUIRED	1 **unmyelinated C fibers**	1	Pass = 4 bolded issues
	2 **smaller** 0.4 – 1.2 um diameter	2	
	3 **slower** 0.5 – 2.0 m/s	3	
	4 also dorsal horn but lamina 1 and 2	4	
	5 neurotransmitter is substance P	5	
	6 **different sensation – dull / intense / diffuse**	6	
	7 different locations as less A delta fibers in deeper structures	7	
THIRD QUESTION (if needed)	What do you understand by the term referred pain?	Prompt for example	
POINTS REQUIRED	1 Same embryonic segment or dermatome	1	Pass = 1/2
	2 Example	2	

Pain, Referred 2013-2-D

Stem: Moving on to Physiology: The patient complains of shoulder tip pain that is thought to be referred from his abdomen.			
PHYSIOLOGY Question 4 LOA: 2	1. Define the term 'referred pain" 2. From which structure is pain referred to the shoulder? 3. Explain this relationship 4. Can you give another example of referred pain? 5. (EXTRA if good candidate) What is the physiological basis/theory for referred pain	1. **Irritation of a visceral organ causing pain in a distant somatic structure** 2. **Diaphragm** 3. Dermatome rule. Referred pain is usually to a structure that **developed from the same embryonic segment or dermatome** as the structure from which the pain originates 4. Cardiac pain to arm. Ureteric pain to testicle. 5. Convergence-Projection Theory. Somatic and visceral pain fibres converge on the same second-order neurons in dorsal horn that then go on to thalamus and sensory cortex via common path. Sensory cortex cannot determine whether the stimulus came from viscera or are of referral	Bold to pass Bold to pass Bold to pass 1 to Pass

Reticular Activating System 2007-1

TOPIC: Reticular activating system _____ **NUMBER:** _____

OPENING QUESTION	What is the function of the reticular activating system?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Centres within network regulate respiratory, cardiovascular, vegetative and endocrine functions		
	2 Non-specific activation from any modality		
	3 Sends signals mostly to the thalamus		
	4 Increases cortical electrical activity		
	5 Increased consciousness, alert state, heightened sensory perception		Essential
SECOND QUESTION	Describe its location and structure.	What are its connections?	
POINTS REQUIRED	1 Complex polysynaptic network	Where is the RAS located?	Essential
	2 Mid ventral portion of medulla + midbrain		
	3 Converging sensory fibres from long tracts and cranial nerves		

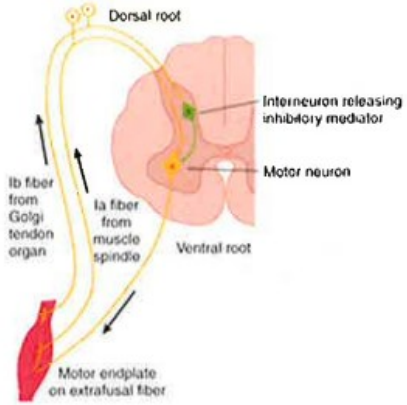
Spinal Tracts 2015-1-B

Stem: We will now move to Physiology.			
Question 4 Spinal Tracts (pp 228-229) **Subject:** Phys LOA: 2	What are upper motor neurons?	Upper motor neurons usually refer to **corticospinal neurons that innervate spinal motor neuron**s (also include brain stem neurons that control spinal motor neurons).	Bold
	What clinical features are seen when they are injured?	Damage **initially causes muscles to become weak and flaccid** but eventually leads to **spasticity, hypertonia, hyperactive stretch reflexes and an abnormal plantar extensor** reflex (upwards))	2 of bold findings
	What is the physiological basis to clonus?	Loss of descending cortical input to inhibitory neurons called Renshaw cells, and therefore **loss of inhibition of antagonists,** resulting in repetitive sequential contractions of ankle flexors and extensors.	Bold
	List the long term complications of spinal cord injury	Ulcers Protein /muscle degradation Hypercalcaemia Renal stones (calcium) Urinary tract infection	2

Stretch Reflex 2017-2-C

Stem: Moving on to Physiology. As part of your assessment of the nerve injury, his upper limb reflexes are assessed			
Question 4 Reflexes **Subject:** Physiology LOA: 1	Describe the components of the stretch reflex. How is it different from the withdrawal reflex? *Prompt – describe a polysynaptic reflex*	Sensor (muscle spindle), **afferent** nerve, integrator (**monosynapse** on motor neurone), **efferent** nerve, effector (intrafusal fibres). Withdrawal reflex is a Polysynaptic reflex. Also has afferent and efferent limbs, but sensory organ is **nociceptor (painful stimulus)**. Central integrator consists of **polysynaptic connections in the spinal cord** i.e. one or more interneurons and interposed between afferent and efferent neurons. Efferent limbs are motor nerves to effector muscles on the ipsilateral and contralateral sides. **Flexion and withdrawal of the ipsilateral limb** and extension of the contralateral limb.	Bold to pass Bold to pass

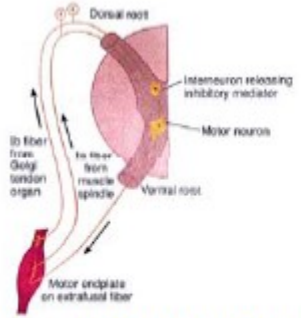
Stretch Reflex 2016-2-C

Stem: A 4-year-old girl presents following a febrile convulsion. She is hyperreflexic. We will start with Physiology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	
Question 1 Stretch reflexes **Subject:** Physiology LOA: 2	1. Please describe the components of the stretch reflex: 2. What is a typical example of this?	1. **Monosynaptic**; skeletal muscle stretched with contraction muscle as the response Stretch receptor sensory neuron makes an excitatory connection with the extensor motor neuron of the same muscle and an inhibitory interneuron projecting to the antagonistic muscle. Sense organ is muscle spindle. Impulses from spindle are transmitted to the CNS by fast sensory fibres that pass directly to the motor neurons that supply the same muscle. The neurotransmitter at the central synapse is glutamate. Eg The Knee **jerk**  	Simple explanation +/- diagram Bold

Stretch Reflex 2012-1

Question 5 LOA: 2	5.1 What is clonus?	**Regular, repetitive, rhythmic contractions of a muscle subjected to sudden, sustained stretch.**	Bold to pass
	5.2 Why does ankle clonus occur with upper motor neuron lesions?	Loss of descending cortical input to inhibitory neurons called Renshaw cells, and therefore **loss of inhibition of antagonists**, resulting in repetitive sequential contractions of ankle flexors and extensors.	
	5.3 What are the components of the stretch reflex?	**Sensor, afferent nerve, Monosynaptic at spinal level, efferent nerve, effector**	

Stretch Reflex 2010-2

Question 4 Monosynaptic reflex	**Please describe a monosynaptic stretch reflex**	 Muscle spindle and its reflex connections are involved in proprioception	Essential to pass Monosynaptic, sensory organ, effector

Stretch Reflex 2006-1

TOPIC: Stretch and inverse reflexes **NUMBER: 4**

OPENING QUESTION	Can you give an example of a stretch reflex?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. Knee jerk 2. Ankle jerk		Need 1 example to pass
SECOND QUESTION (if needed)	Describe the elements of the stretch reflex..		
POINTS REQUIRED	1. Sensor from afferent limb (1a fibre from muscle spindle, monosynaptic neurone & excite the motor neurone, so the muscle contract.		Need to say all to pass
THIRD QUESTION (if needed)	What is an inverse stretch reflex?		
POINTS REQUIRED	Following prolonged stretch or muscle contraction, the contracted muscles suddenly relax. Stimulate the Golgi tendon organ , integrator (synapse on motor neurone for stretch and on inhibitory interneuron for inverse stretch), efferent limb (ventral root for both) and effector (extrafusal fibres muscle fibres)		Need definition, sensor, synapse, inhibitory effect.

COMMENTS

Stretch Reflex 2005-1

Stretch reflex	Describe the elements that make up a stretch reflex	Sensor (muscle spindle); afferent limb; integrator (synapse on motor neurone); efferent limb; effector (intrafusal fibres)	
	How do the muscle spindles function?	Parallel intrafusal fibres responds to stretch with different dynamic and static responses.	

Thermoregulation 2016-2-D

Stem: His temperature is 38°C. Moving to Physiology.			
Question 2 Thermoregulation **Subject:** Phys LOA: 1	a. How is heat lost from the body? b. How is fever produced in the body? *Prompt: Outline the pathophysiological mechanism of fever.*	**Radiation and conduction** (70%), **vaporisation of sweat** (27%), respiration (2%), urination and defaecation **(**1%). Endotoxins, inflammation and other **pyrogens** act on monocytes, macrophages and Kupffer cells to **produce cytokines** (eg. interleukins and TNF). Cytokines act on circumventricular organs (eg. OVLT) which activate the pre-optic area of the **hypothalamus**. Local release of prostaglandins **raises the temperature set point**.	Bold to pass 3 of 4 bold points

Thermoregulation 2014-2-D

Stem:Moving on to physiology			
Question 2 Hypothermia / thermoregulation (pp 316-320) **Subject:** Phys LOA: 1	By what processes does the body lose heat? How does the body produce heat? What temperature-regulating mechanisms are activated by the cold? What part of the brain controls the reflex responses activated by cold?	**Radiation & Conduction** (70% of loss at 21 °C) **Vaporization of sweat** (27%) Respiration (2%) Urination & defecation (1%) **Basal metabolic processes** Food intake **Muscular activity** Shivering Hunger Increased voluntary activity Increased secretion of Adr + NorAdr Decreased heat loss mechanism Cutaneous vasoconstriction Curling up Horripilation The posterior **hypothalamus**	Bold to pass Bold to pass 4 to pass bold

Thermoregulation 2012-1

Question 4 LOA: 1	4.1 Describe the body's response to cold? 4.2 Outline the pathogenesis of fever.	shivering, hunger, ↑voluntary activity, ↑NA, A, ↓ heat loss, curling up, behaviour change, cutaneous vasoconstriction, horripilation Toxins from infective agents act on monocytes, macrophages and Kupffer cells to produce cytokines which act as endogenous pyrogens (EPs), *also IL-1β, IL-6, β-IFN, γ-IFN, TNF act on the OVLT, which in turn activates pre-optic hypothalamus through local release of PGs.*	Give 4 EPs indirect action on hypothalamus to reset

Thermoregulation 2011-1

Question 4 Thermoregulation	4.1 What mechanisms does the body use to regulate temperature? PROMPT: What mechanisms are activated by cold? PROMPT: Are any voluntary?	a) **Activated by cold:** Shivering, Hunger, Increased voluntary activity, adrenaline and noradrenaline secretion, decreased heat loss, cutaneous vasoconstriction, curling up, horripilation	4 to pass
		b) **Activated by heat:** Increased heat loss, cutaneous vasodilation, sweating, increased respiration, decreased metabolic heat production, anorexia, apathy & inertia	4 to pass
	4.2 How are these temperature regulating mechanisms controlled?	Reflex responses activated by cold controlled from posterior **hypothalamus** Those activated by warmth are controlled primarily from the anterior **hypothalamus**	Bold to pass

Thermoregulation 2010-2

Question 4 thermoregulation	What factors are responsible for heat production and heat loss?	**Heat Production** Basic metabolic process Specific dynamic action of food Muscular activity **Heat is lost by** Radiation and conduction Vaporisation of sweat Respiration Urination and defaecation	To Pass 2 of each
	Describe the body's adaptive response to a cold environment	**Mechanisms activated by cold** Shivering Hunger ↑ Voluntary activity ↑ Secretion of adrenaline/nor adrenaline ↓ Heat loss Cutaneous vaso constriction Curling up Horripilatian (Controlled from posterior hypothalamus)	3 to pass

Thermoregulation 2009-1

Question 4: Temperature regulation Ganong 251-5	i) How does the body generate heat? ii) How does the body lose heat? iii) What is the thermo-regulatory response to cold?	a) Heat production: **basal metabolic processes, muscular activity**, food intake b) Heat loss: conduction, convection and **radiation** (70%), **sweat vaporisation** (27%), respiration (2%), urine and defaecation (1%) i. **Increased heat production** such as shivering, muscular activity, hunger (eating), hormonal increase in adrenalin/ NA. **Decreased heat loss** with cutaneous vasoconstriction, curling up, horripilation (goose pimples – erection of hairs to ii. Decrease conduction/convection.	Core knowledge in bold

Thermoregulation 2007-2

QUESTION: 5. THERMOREGULATION

Question	**Required response [Key items marked with*]**	**To Pass**
What are the thermoregulatory responses to cold ?	Autonomic, Somatic, Endocrine and Behavioural Response to Cold: Increase heat production **Shivering** Hunger Increased voluntary activity Increased secretion Adrenaline and NA Decrease heat loss **Cutaneous vasoconstriction** Curling up Horripilation (pilo erection)	Shivering Cutaneous vasoconstriction
What are the thermoregulatory responses to heat ?	Response to Heat: Increase heat loss Cutaneous vasodilation Sweating Increased respiration Decrease heat production Anorexia Apathy and inertia	Any 2 from list
Where are these responses regulated?	Reflex response to cold controlled in post hypothalamus Reflex response to heat controlled in ant hypothalamus Afferents come from sensory receptors in skin, deep tissue, spinal cord, extrahypothalamic parts of brain and hypothalamus itself.	Hypothalamus

Thermoregulation 2005-2

2.5 Regulation of temperature	Describe the regulation of normal body temperature. How is fever generated?	**Hypothalamus** controls range; circadian and ovulatory variations. Posterior responds to cold, anterior to heat. **Heat production**: activity, feeding, adrenaline drive, thyroid activity; shivering, vasoconstriction. **Heat losses**: conduction, radiation, convection, vaporisation; sweating, vasodilation, increased respiration, Pyrogens etc 'reset the thermostat' Cytokines are endogenous pyrogens	4/6 5/7

Thirst 2015-1-B

Stem: A 70 year old lady with a history of bipolar disorder is found on the floor many hours after a fall. On arrival in ED, she is tachycardiac and complaining of severe thirst and thigh pain. Starting with Physiology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Thirst **Subject:** Phys LOA: 1	Q1. Where is thirst regulated?	**Hypothalamus** - diencephalon	Bold
	Q2. What factors increase thirst?	1. **Increase in osmotic pressure in plasma** sensed by osmoreceptors in the anterior hypothalamus 2. **Decrease in ECF volume** (e.g. haemorrhage) a) Sensed by baroreceptors in heart and blood vessels – increases thirst b) Increase in renin – causes increase AT II – acts on the diencephalon neurons – increases thirst 3. Psychological – e.g. acute psychosis 4. Others a) Increase liquids during eating (prandial drinking) b) Other poorly understood mechanisms such as increased osmolality as food absorbed and GI hormones acting on the hypothalamus	Bold with understanding
	Q3. In what situations may thirst sensation be blunted?	hypothalamic disease direct damage to the diencephalon **altered mental state** psychosis lesion of the anterior communicating artery (supplies the hypothalamus) diet high in protein (products of protein metabolism cause water diuresis)	Bold + 1 other

Vision 2017-1-A

Stem: Moving onto Physiology. He also reports pre-existing blurred vision.			
Question 4 Visual pathways **Subject:** Phys LOA: 1	a) Describe the neural connections of the visual pathways.	a) **Retina, optic nerve, optic chiasm, optic tract**, lateral geniculate body (thalamus), geniculocalcarine tract, primary visual cortex **(occipital lobe). At optic chiasm, nasal fibres decussate to the contralateral side.** Other connections: - Optic tract (via superior colliculus) to pretectal midbrain, then to Edinger-Westphal nuclie in oculomotor nerve (pupillary reflexes, eye movement) - Frontal cortex (refined eye movement - vergence, near point response) - Retinal ganglion cells to suprachiasmatic nucleus hypothalamus (endocrine & circadian responses to day/night cycle)	**Bold** to pass
	b) Why is the fovea important for visual acuity?	b) Point where **VA greatest**; fovea is the centre of the macula, a thinned out rod-free portion of the retina where the **cones are densely packed** & each synapses on a single bipolar cell, which, in turn, synapses on a single ganglion cell, providing a direct pathway to brain	One of **bold plus one** other to pass
	c) What ocular factors influence visual acuity?	c) **Optical factors**: state of the image-forming mechanisms eg cataracts, keratitis, astigmatism, myopia, hyperopia **Retinal factors** eg the state of the cones, retinopathies, optic neuritis **Stimulus factors** eg illumination; brightness of the stimulus; contrast between stimulus and background; length time exposed to stimulus)	**3 factors**

Visual Acuity 2014-2-D

Stem: Moving on to physiology You assess his visual acuity as 6/24.			
Question 2 Eye / Acuity / Vision (pp 178 -183) **Subject**: Phys LOA: 2	How is visual acuity measured? What does the fractions of a VA of 6/24 represent? What factors influence visual acuity? Why is the fovea important for visual acquity	Measurement from **Snellen chart** viewed at a distance of **6m** or 20 feet; **6/24 indicates reduced VA** **Optical factors** The state of the image forming mechanisms/sharpness of focus **Retinal factors** the state of the cones **Stimulus factors (**illumination, brightness of the stimulus, contrast between stimulus and background, length time exposed to stimulus); sensitivity and interpretative ability of the brain Resolving power of the eye, property of the cones fovea is the point where **VA is greatest**; fovea is the centre of the macula, a thinned out rod free portion of the retina where the **cones** are densely packed & each synapses on a single bipolar cell	numerator is the distance at which the chart is read; the denominator is the **smallest line** that can be read; 6/6 indicates normal vision; 2/3 to pass One of bold

Withdrawal Reflex 2012-2

Question 4 Withdrawal LOA: 2	Describe the withdrawal reflex. (Prompt – what are the components?)	A **polysynaptic** reflex occurring in response to a painful stimulus to skin/subcut tissue and muscle. **Survival/protective** basis. A pre-potent reflex (takes priority of all other concurrent reflex activity) The "**crossed"** response is flexor muscle contraction and extensor muscle inhibition, so the body part is flexed and withdrawn from stimulus. ALSO extension of opposite limb. 'Irradiation of stimulus' up and down spinal cord results →recruitment of motor units' Reflex is enhanced by abolition of brain modulation.	Need the bold concepts to pass

Withdrawal Reflex 2009-1

TOPIC: Withdrawal Reflex_____ **NUMBER: 3** _____

OPENING QUESTION	Describe the withdrawal reflex?	**PROMPTS COMMENTS**	
POINTS REQUIRED	1 Reflex arc consisting of sense organ afferent and efferent nerve and effector	1	Pass = 3/5
	2 Noxious stimulus to skin or sub cut	2	
	3 Response of flexor muscle contraction and extensor relaxation	3	
	4 Result in withdrawal of limb from stimulus	4	
	5 Cross extensor response	5	
SECOND QUESTION (if needed)	What is meant by the term polysynaptic reflex?	Prompt if necessary	
POINTS REQUIRED	1 One or more interneurons and interposed between the afferent and efferent neurons	1	
THIRD QUESTION (if needed)	What are the effects of a polysynaptic reflex?		Pass = 1/2
POINTS REQUIRED	1 Prolonged effect as different time for stimulus to reach effector	1	
	2 Reverberation circuit as some interneurons turn back on themselves further prolonging the effect.	2	
	3	3	
	4	4	
	5	5	
	6	6	
	7		

Withdrawal Reflex 2006-2

TOPIC: Withdrawal Reflex_____ NUMBER: 3 _____

OPENING QUESTION	Describe the withdrawal reflex?	PROMPTS	COMMENTS
POINTS REQUIRED	1 Reflex arc consisting of sense organ afferent and efferent nerve and effector	1	Pass = 3/5
	2 Noxious stimulus to skin or sub cut	2	
	3 Response of flexor muscle contraction and extensor relaxation	3	
	4 Result in withdrawal of limb from stimulus	4	
	5 Cross extensor response	5	
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	7		