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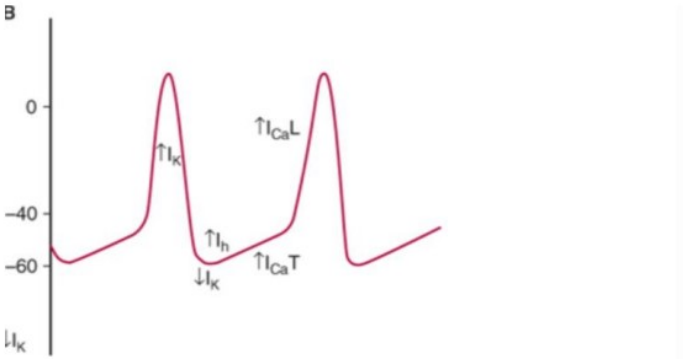
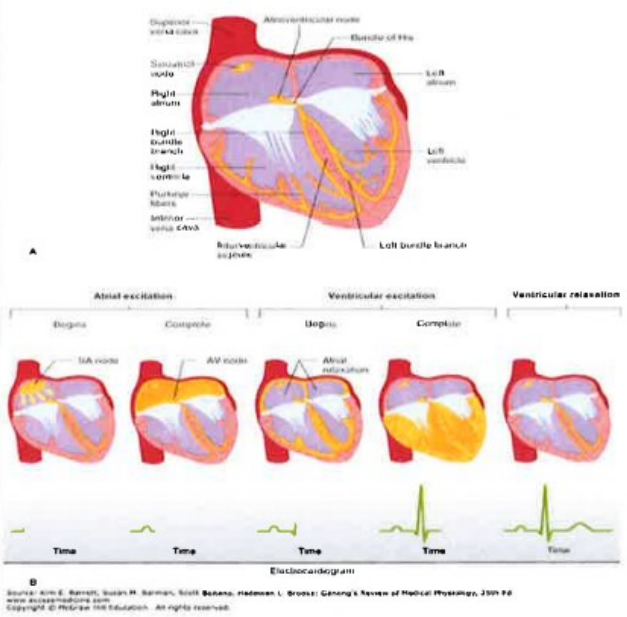
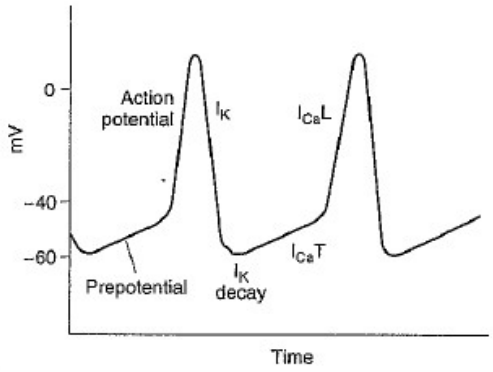
Cardiac Action Potential 2017-2-A

Stem: Moving on to Physiology			
Question 3 Conducting system **Subject:** Physiology LOA: 1	a) Describe the normal cardiac conduction pathway	**a) SA node** (pacemaker) **Atria** (3 internodal pathways) **AV node; Bundle of His; Right and left bundle branch** **Anterior and posterior fascicles on left** **Purkinje fibres** **Ventricular muscle:** left side of IV septum first, spread down septum to apex, Up AV grooves, spread from endocardial to epicardial surfaces **- P wave: Atrial depolarisation** **- QRS complex: Ventricular depolarisation** **- T wave: Ventricular repolarisation** NB: atrial repolarisation is not represented because it is buried in the QRS complex.	6 of 8 Bold to pass Prompt: what is happening electrically?
	b) This is a normal ECG complex. What does each wave of the ECG represent? c) Draw and describe the action potential of a cardiac pacemaker cell	 1. **Pre-potential is initially due to a decrease in K^+ efflux**, then completed by Ca^{2+} influx through CaT channels (prepotential). 2. The **action potential is due to influx of Ca^{2+}** via CaL channels. 3. **Repolarisation is due to K^+ efflux**	Bold Need approx. correct shape of curve and approx. potential values

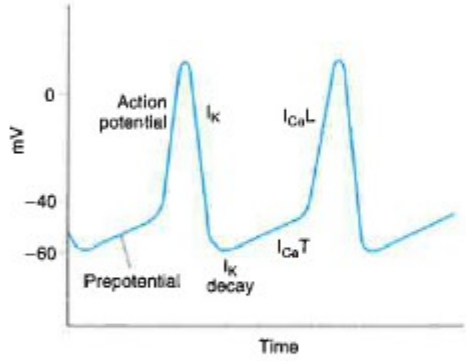
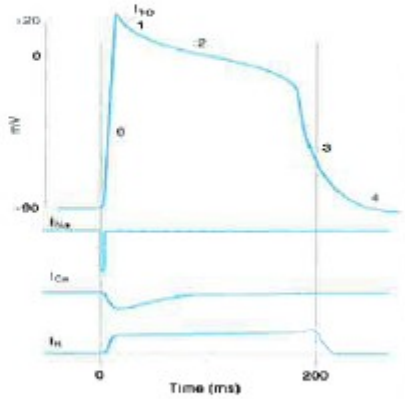
Cardiac Action Potential 2016-2-B

Stem: Moving onto Physiology.			
Question 2 Electrical activity of the heart / atrial arrhythmias **Subject:** Physiology **LOA:** 1	Please draw and describe a normal ECG complex. Describe the normal sequence of electrical excitation of the cardiac conduction system and cardiac muscle	 **P** = Depolarization initiated in the **SA node** Spreads radially through the atria, Converges on the AV node. (Atrial depolarization 0.1 s.) **PR** = **Atrial depolarization & AV nodal delay (**Delay of about 0.1 s) **QRS** = Bundle of His, R&L Bundles & Purkinje fibers (**ventricles** 0.08-0.1 s) (L) to (R) across IV septum then down septum to apex along ventricular walls to AV groove from endocardial to epicardial surface last parts to be depolarized are posterobasal portion of LV, pulmonary conus, and uppermost septum	Draw ECG and accurately identify all waves and intervals (**P,PR, QRS, T, QT & ST**) Prompt – Intervals? Bold to pass 2 depolarization directions to pass

Cardiac Action Potential 2014-2-B

Stem: We will now move on to Physiology. The patient has reverted to sinus rhythm.			
Question 4 **Subject:** Phys LOA: 1	1. Describe the normal sequence of electrical excitation of the cardiac conduction system and cardiac muscle?	Normal sequence of depolarisation: **SA Node** **Atria** **AV Node** Bundle of His **Major bundles (Right and left)** Purkinje fibres **Ventricular muscle**	Bold to pass.
	2. What are the common mechanisms that cause abnormalities of cardiac conduction?	Abnormal pacemakers Re-entry circuits Conduction deficits Prolonged repolarisation Accessory pathways Electrolyte disturbance	4 to pass
	3. Please draw and explain the action potential of a cardiac pacemaker cell Prompt: Which electrolytes are responsible for each phase of the action potential?	Pre-potential is initially due to a decrease in K+ efflux, then completed by Ca2+ influx through CaT channels. The action potential is due to influx of Ca2+ via CaL channels. Repolarisation is due to K+ efflux  	To pass: Correct shape of graph Know ion fluxes: • Pre-potential decrease K+ efflux/Ca2+ influx • Action potential Influx Ca2+ • Repolarisation K+ efflux

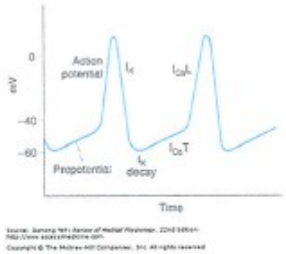
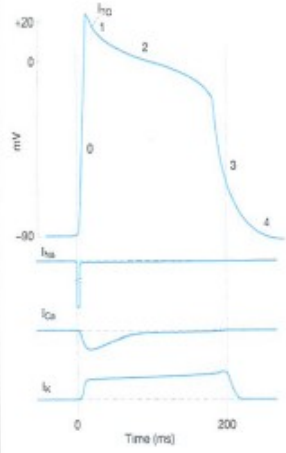
Cardiac Action Potential 2013-1

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOA: 1 Cardiac Muscle Action Potential (- incl difference to pacemaker action potential)	a. Please draw and explain the action potential in a cardiac pacemaker cell. Prompt: "What electrolytes are responsible for each phase of the action potential?"	Pre-potential is initially due to a decrease in K^+ efflux, then completed by Ca^{2+} influx through CaT channels The action potential is due to influx of Ca^{2+} via CaL channels Repolarisation is due to K^+ efflux  	Must have the shape to pass and know the ion fluxes. 
	b. Describe the major differences between a ventricular muscle action potential and a pacemaker cell potential.	Greater negative RMP. Fast depolarisation via Na^+ versus slower Ca^{2+} dependent. No prepotential and no automaticity. Plateau phase.	Clear contrast to the above graph, No prepotential as no leaking Ca^{2+} and plateau due to Ca^{2+}.

Cardiac Action Potential 2011-1

Question 1	1.1 Describe the normal sequence of electrical excitation of the cardiac conduction system and cardiac muscle?	Normal sequence of depolarisation: **SA node** **Atria** (pathways) **AV node** Bundle of His **Major bundles** (Right & Left) Purkinje fibres **Ventricular muscle** Left side of IV septum first Spread down septum to apex Then up to AV grooves Spread from endocardial to epicardial surfaces	All of bold to pass
	1.2 What are the common mechanisms which cause abnormalities of cardiac conduction?	**Abnormal pacemakers** **Re-entry circuits** **Conduction defects** Prolonged repolarisation Accessory pathways	2/3 bold to pass
	1.3 What are the possible clinical consequences of these conduction abnormalities? (Flexibility between 1.2 & 1.3)	Abnormal pacemakers • **ectopic beats** • pacemaker failure (sinus arrest) • **fibrillation (atrial or ventricular)** Re-entry circuits • leading to tachyarrhythmias Conduction delays • **heart block** • bundle branch blocks Prolonged repolarisation • Long QTc Accessory pathways • WPW or LGL	2 bold + 2 others to pass

Cardiac Action Potential 2009-2

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1a:	**Draw and explain the action potential in a cardiac pacemaker cell.**	 PROMPTS: What electrolytes are responsible for each phase of the AP?	**Pass-fail** **Must have shape to pass and know ion fluxes (↓effK + infCa T)- InfCa L – Eff K)** **1 Pre-potential initially due to decrease in efflux K^+, then completed by influx Ca^{2+} through T channels** **2 AP due to influx Ca^{2+} via L channels** **3 Repolarisation due to efflux K, no plateau**
Question 1b:	**Describe the major differences between a cardiac myocyte AP and the pacemaker**		1. Resting membrane potential, , –90mV rapid depolarisation voltage gated Na (overshoots) 2. Phase 1 rapid repolarisation = closure of Na channels.(inner v outer gates) 3. Plateau phase 2 voltage gated Ca2+ channels open (slower L type) 4. Phase 3 repolarisation Ca2+ ch close 5. Phase 4 due to various K+ efflux Differences- 1) Na fast v Ca dependent, 2) automaticity due to rising prepotential (K+/ Ca+), 3) plateau phase, 4) > resting potentials **Pass-Fail: Need correct shape + some knowledge of different channels (partic Na v Ca), no automaticity (no-prepotential, as no leaking K/ Ca) and plateau due to Ca ++ (>er inactive phase)**

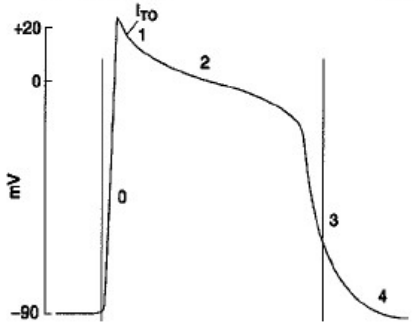
Cardiac Action Potential 2008-2

OPENING QUESTION	**Draw the action potential in a cardiac pacemaker cell, and explain the ionic fluxes.**	**COMMENTS**
POINTS REQUIRED	**1 Prepotential initially due to decrease in efflux K^+ , then completed by influx Ca^{2+} through T channels**	Suggested Pass/Fail Criteria in Bolded Type
	2 AP due to influx Ca^{2+} via L channels	
	3 Repolarisation due to efflux K, no plateau	All essential
PROMPTS	What electrolytes are responsible for each phase of the AP?	Stress cardiac pacemaker cell
SECOND QUESTION	**How do sympathetic and parasympathetic stimulation change the prepotential?**	
POINTS REQUIRED	1 Noradrenaline binds to Beta 1 receptor and raises cAMP, resulting in increased opening of L channels and Ca^{2+} influx. **Thus increased slope of prepotential and firing rate**	
	2 ACh binds to M2 receptor and decreases cAMP, resulting in both slowing of Ca channel opening and opening of special K channels (counters decay of K efflux) leading to greater fall in prepotential **Thus decreased slope of prepotential and firing rate**	
PROMPTS	What does noradrenaline do? What does vagal stimulation do?	

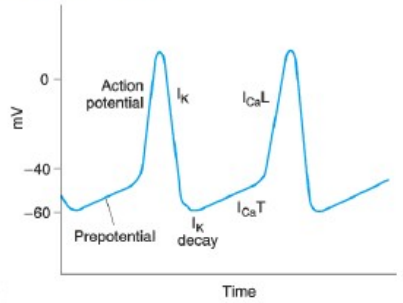
COMMENTS

Cardiac Action Potential 2007-2

QUESTION: 2. ACTION POTENTIAL IN CARDIAC CELLS

Question	Required response [Key items marked with*]	To Pass
Please describe or draw an action potential in ventricular muscle.	• RMP -90 mV (+/- 20) • No prepotential • Phase 0 rapid upstroke to +20 mV (+/- 20) • Phase 1 short-lived rapid depolarisation to around 0 mV. • Phase 2 prolonged plateau. • Phase 3 moderately fast repolarisation to RMP. • Phase 4 is the RMP. 	Shape including plateau General voltages (negative to positive)
What are the ion fluxes that produce this action potential?	• Phase 0 - opening of voltage-gated Na^+ channels allows Na^+ influx. • Phase 1 - due to closure of Na^+ channels and transient K^+ efflux. • Phase 2 - due to slower but prolonged opening of voltage-gated Ca^{2+} channels with Ca^{2+} influx. • Phase 3 - due to closure of Ca^{2+} channels and opening of various types of K^+ channels allowing K^+ efflux • Phase 4 - RMP is due to membrane permeability at rest being much higher for K^+ than for Na^+.	Na influx (depol) Ca influx (plateau) K efflux (repol)
How does the ECG relate to the ventricular muscle action potential?	**Upstroke on QRS** Plateau occupies QT interval **Repolarisation at T wave**	

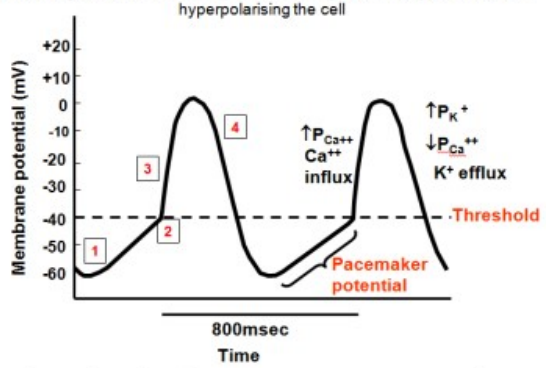
Cardiac Action Potential 2006-2

OPENING QUESTION	Please describe or draw a cardiac pacemaker potential arising from the sino-atrial node?	**PROMPTS COMMENTS**	
POINTS REQUIRED	 1	1	Pass = 3/5
	2 RMP –60 mV	2	
	3 Prepotential to –40 mV	3	
	4 Leisurely upstroke to +ve value	4	
	5 Symmetrical downstroke to RMP	5	
SECOND QUESTION (if needed)	What are the ion fluxes that produce this pacemaker potential?		Pass = 2/4
POINTS REQUIRED	1 The decline in I_K efflux permits the prepotential	1	
	2 $I_{Ca}T$ (transient) influx completes the prepotential	2	
	3 $I_{Ca}L$ (long) influx produces the upstroke	3	
	4 I_K efflux produces the downstroke	4	
THIRD QUESTION (if needed)	Please describe how a pacemaker potential is conducted throughout the myocardium?		3/5 = pass
POINTS REQUIRED	1 Specialised conduction tissue	1	
	2 SA node—atrial pathways—AV node—Bundle of His—Purkinje system (bundles)	2	

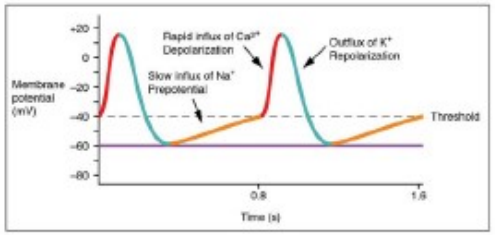
Cardiac Action Potential 2005-2

2.3 Action potential in cardiac cells	Describe the action potential in cardiac muscle fibre Why does tetany not occur in cardiac muscle?	Diagram. **–90 mv. Voltage gated Na channels -> rapid depolarisation (phase 0); closure of channels -> initial rapid repolarisation (phase 1); slower but prolonged opening of Ca channels -> plateau (phase 2); closure of Ca channels and opening of K channels -> final repolarisation (phase 3) to resting potential (phase 4).** Muscle still contracting in relative refractory period and beyond the duration of AP so cannot develop tetany.	4/5

Cardiac Pacemaker Action Potential 2017-1-C

Stem: Moving on to Physiology				
Question 5 Cardiac pacemaker **Subject**: Physiology LOA: 1	a) Draw the action potential in a **cardiac pacemaker** cell and explain the ionic fluxes.	1. 'Funny' sodium channels (I_f channels) are open ($\uparrow P_{Na}{}^{+}$); and closing K^+ channels. 2. Transient Ca^{2+} (T-type) channels open, pushing the membrane potential to threshold. 3. Long-lasting Ca^{2+} (L-type) channels open, giving rise to the action potential. 4. Opening of K^+ channels, ($\uparrow P_{K}{}^{+}$), and closing of Ca^{2+} (L-type) channels, hyperpolarising the cell  	i) Pre potential due to increased influx of Na via 'funny channels' (open in response to hyperpolarisation), **decrease of K efflux**, then **completed by influx of Ca** through T channels ii) **Action potential due to influx of calcium** via L channels iii) **Repolarisation due to efflux of K,** no plateau	Correctly drawn action potential curve and 2 out of 3 bold sections to pass
	b) What is the effect of sympathetic and parasympathetic stimulation on the prepotential?	**i)** Noradrenaline binds to Beta 1 receptors and raises cAMP, resulting in opening of L channels and Ca influx. **Increased slope of prepotential** and increased firing rate. **ii)** Acetylcholine binds to M2 receptor and decreases cAMP resulting in both slowing of calcium channel opening and opening of special K channels (counter decay of K efflux) leading to greater fall in prepotential. Leads to **decreased slope of prepotential** and firing rate.		Bold to pass

Cardiac Pacemaker Action Potential 2015-1-D

Stem: She has a history of palpitations. Moving onto Physiology.			
Question 3 Ventricular Tachycardia **Subject:** Phys LOA: 1	Draw and label the membrane potential of normal pacemaker tissue		Must identify fast upslope being due to **Ca influx** and repolarisation due to **K efflux plus presence of pre-potential**
	By what mechanisms can tachyarrhythmias be generated?	Increased automaticity (AT,VT) Accessory pathways (WPW) Re-entry loops (VT) Early afterdepolarisations (torsade de pointes) Delayed afterdepolarisations (as in digoxin toxicity)	Automaticity plus one other
	What conditions may predispose to increased automaticity?	IHD Previous repair of congenital heart disease (scar tissue) Structural heart disease Channelopathies (congen or acquired) Electrolyte imbalances (K, Mg, Ca) Sympathomimetic agents Infiltrative cardiac diseases	Mention at least one condition
Stem: She is hypotensive and this ECG is performed.			
Clinical Building Block – ECG	What rhythm does it show?	Broad complex regular tachycardia consistent with VT. Rate approximately 180bpm.	Must identify that broad complex, regular tachycardia or VT

Cardiac Pacemaker Action Potential 2007-1

TOPIC: Pacemaker potential _____ **NUMBER:** _____

OPENING QUESTION	Describe the features of the action potential in cardiac pacemaker tissue	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Prepotential initially due to decrease in inward K^+ movement then inward Ca^{2+} through T channels	Compare it to vent muscle	All essential
	2 Action potential due to inward Ca^{2+} through L channels		
	3 Repolarization due to inward K^+ movement		
	4 No plateau		
SECOND QUESTION	How do autonomic factors alter the slope of the prepotential?	What does noradrenaline do?	
POINTS REQUIRED	1 Noradrenaline from sympathetic endings raises intracellular cAMP		
	2 Facilitates opening of L channels		
	3 Increased Ca^{2+} influx		
	4 Increased heart rate		
	5 ACh acts via muscarinic receptors and G protein to open K^+ channels and decrease rate		

ECG Physiology 2015-2-A

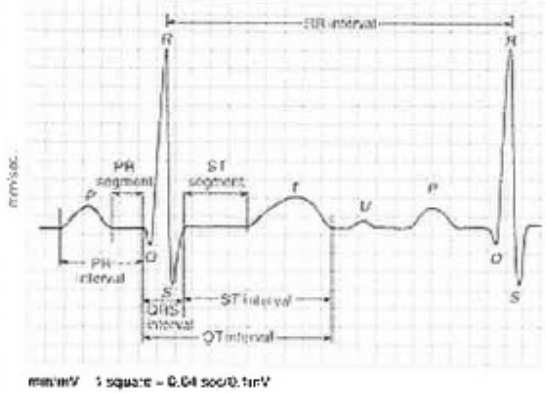
This woman also has chronic renal failure. An ECG is obtained.			
Question 2 **Clinical Building Block:** ECG – hyperkalaemia	What are the abnormalities on the ECG? What is the likely diagnosis?	**Widespread peaked T waves**, mild tachycardia, some inverted T waves, ST elevation **Suggestive of hyperkalaemia**	Bold
Stem: Moving onto Physiology			
Question 3 ECG and hyperkalaemia & Renal handling of K+ **Subject:** Phys LOA: 1	What are the ECG changes in Hyperkalaemia? Describe the way the kidney handles K^+ Prompt: What happens to K^+ in Proximal Tubule? Prompt: What happens to K^+ in distal tubule?	Peaked T waves (repolarisation abnormality) P wave flattening, Loss of P waves (progressive atrial paralysis) QRS widening / bizarre QRS (Conduction abnormality) Sinusoidal ECG Ventricular arrhythmias Asystole K^+ is filtered at the glomerulus. **Most filtered K^+ is actively reabsorbed at the proximal tubules.** **K^+ is then secreted into the fluid by the distal tubules.** (Rate of K^+ secretion is proportionate to distal tubular fluid flow) At Distal tubule: K+ secretion is passive and Na^+ is reabsorbed. In a healthy person: the amount of K^+ secreted = K^+ intake and K balance is maintained Normally > 93% K^+ is reabsorbed by the kidneys. K^+ secretion & excretion alter depending on serum K^+ and H^+	3/6 to pass Bold to pass

ECG Physiology 2015-1-C

Stem: A 60 year old woman with a history of hypertension presents with chest pain radiating into her back. An ECG is done.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Clinical Building Block ECG with AMI	Please describe and interpret the significant abnormalities in this ECG.	• Sinus, rate ~100/min, normal axis • **ST elevation (STEMI)** • **Inferior leads** • ST depression and inverted T waves in I, aVL, V2, V3 (Reciprocal changes)	Bold
Stem: We will now move to Physiology.			
Question 1 ECG – myocardial infarction **Subject:** Phys LOA: 1	Explain the electrophysiological changes that cause the ST segment elevation seen in a myocardial infarction? *Prompt for time course* *Second prompt for cellular mechanism*	• Abnormally rapid repolarisation of the infarcted muscle (accelerated opening of K+ channels). Current flow out of infarct (normal region negative relative to infarct). Occurs within seconds of infarction and last a few minutes. • Decreased resting membrane potential (due to loss of intracellular K+). Begins in first few minutes secondary to process above. Current flow into infarct during diastole (ECG configured to record as ST elevation). • Slowed depolarisation of affected cells cf normal cells. Occurs @ 30 minutes into infarct process. Current flow out of infarct.	**2 of 3 to pass**

ECG Physiology 2013-2-C

Stem: A 65 yr old man presents with an inferior myocardial infarction
We are starting with Physiology

TOPIC	QUESTIONS	*KNOWLEDGE **(essential in bold)***	NOTES
Question 1 **PHYSIOLOGY** **ECG including MI changes** LOA: 1 Ganong 24th ed pp 524-529, 534-537	1. Draw and describe an ECG tracing of a single normal heart beat Prompt: What produces the waves and segments?	 **P wave- atrial depolarization, PR AV conduction** **QRS- ventricular depolarization, ST- plateau of Vent depolarization, (QT- Ventricular Action potential),** **T wave- Vent repolarization**	Bold 5/6
	2. What features would appear different in this patient's ECG?	**2. ST segment elevation in inferior leads** **ST segment depression in the reciprocal leads**	both
	3. At the myocardial cell membrane level, what causes these changes?	3. Abnormally rapid depolarisation in early phase (accelerated opening of K+ channels) Decreased resting membrane potential (due to loss of intracellular K+) Slowed depolarization of affected cells (cf normal cells)	1 of 3 to pass

ECG Physiology 2012-2

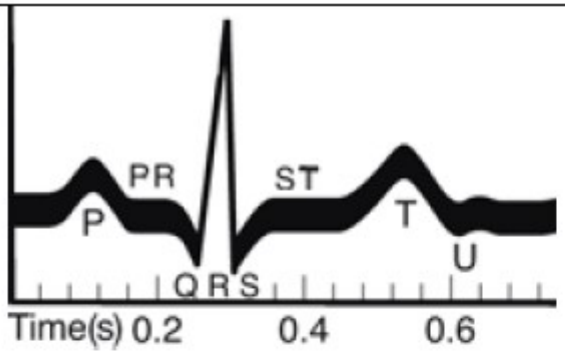
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOA: 1	1.1 Please draw and label the intervals and segments of a normal ECG including times? 1.2 What electrophysiological event occurs during these periods?	**P wave, PR interval, QRS complex, ST segment, T wave (** U wave optional) and **QT segment** **PR interval: 0.12-0.2 sec. Atrial depolarisation and conduction through AV node** **QRS duration: 0.08 – 0.12 sec. Ventricular depolarisation** and atrial repolarisation. **QT interval: 0.40-0.43 sec. Ventricular depolarisation plus ventricular repolarisation** **ST interval** (QT minus QRS) **0.32 sec. Ventricular repolarisation**	Successfully draw an ECG tracing and label all of it + correctly identify the duration of 2 of the 4 intervals to pass 3 of 4 events

ECG Physiology 2010-2

<table>
<tr>
<td>Question 1:

Normal ECG and cardiac membrane polarisation changes/ECG findings in MI</td>
<td>Please draw a normal ECG tracing

Describe the cardiac events that relate to each of the intervals

What is the electrophysiological basis for ST elevation in acute MI?</td>
<td> 

The ST segment represents the plateau (Phase 2) of the AP and the T wave is repolarisation (Phase 3).

ST segment elevation concave upwards.

<table>
<tr><th>Polarisation anomalies in infarcted Cells</th><th>Current flow</th><th>ECG Change in leads over the Infarct</th></tr>
<tr><td>Rapid repolarisation</td><td>Out of infarct</td><td>ST segment elevation</td></tr>
<tr><td>Decreased resting membrane potential</td><td>Into infarct</td><td>TQ segment depression (manifested as ST segment elevation)</td></tr>
<tr><td>Delayed depolarisation</td><td>Out of infarct</td><td>ST segment elevation</td></tr>
</table>
</td>
<td>Recognisable shape with QRS

Name intervals.

Describe the ST segment representing plateau (Phase 2) to pass this subsection.

One of three</td>
</tr>
</table>

ECG Physiology 2008-2

OPENING QUESTION	**Please draw a normal ECG tracing, showing the durations of the major intervals**	**COMMENTS**
POINTS REQUIRED	**1. correct shape**	All
	2. **times PR 0.16 QRS 0.12 QT 0.4**	
PROMPTS		
SECOND QUESTION (if needed)	**How does the ECG change with hyperkalaemia?**	
POINTS REQUIRED	1. progression	
	2 Initial **tall peaked T waves. Intervals normal K 7.0**	At least 3
	3. **later no atrial activity, QRS broad/slurred**	
	4 **ventricular arrhythmias then fibres eventually** unexcitable, sine wave appearance	
PROMPTS		
THIRD QUESTION (if needed)	**How does it change with hypokalaemia?**	
POINTS REQUIRED	1. **long PR, ST depression, inverted T,**	Both required
	2. **U wave**	
PROMPTS		

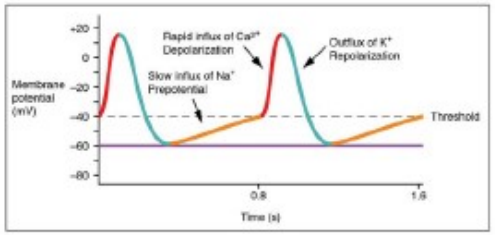
COMMENTS

ECG Physiology 2006-1

TOPIC: Physiology of ECG NUMBER: 1

OPENING QUESTION	Please draw a normal electrocardiogram. What do the different waves & segments represent?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	P-wave=atrial depol, PR interval=atrial depol & AV conduction (0.18 sec) QRS=ventricular depol (& atrial repol 0.08 sec) ST segment & T wave = ventricular repol 0.32 sec QT interval = ventricular depol & repol 0.4 sec. U wave=slow repol papillary muscle	Ask for duration if candidate is doing really well.	Need to name the different segments. Know what PR, QRS, QT mean to pass.

Pacemaker 2015-1-D

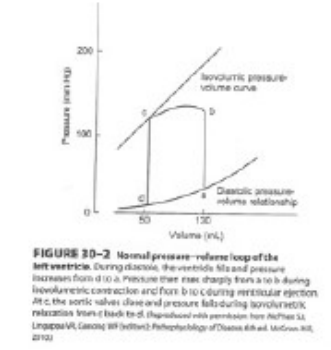
Stem: She has a history of palpitations. Moving onto Physiology.			
Question 3 Ventricular Tachycardia **Subject:** Phys LOA: 1	Draw and label the membrane potential of normal pacemaker tissue		Must identify fast upslope being due to **Ca influx** and repolarisation due to **K efflux plus presence of pre-potential**
	By what mechanisms can tachyarrhythmias be generated?	Increased automaticity (AT,VT) Accessory pathways (WPW) Re-entry loops (VT) Early afterdepolarisations (torsade de pointes) Delayed afterdepolarisations (as in digoxin toxicity)	Automaticity plus one other
	What conditions may predispose to increased automaticity?	IHD Previous repair of congenital heart disease (scar tissue) Structural heart disease Channelopathies (congen or acquired) Electrolyte imbalances (K, Mg, Ca) Sympathomimetic agents Infiltrative cardiac diseases	Mention at least one condition
Stem: She is hypotensive and this ECG is performed.			
Clinical Building Block – ECG	What rhythm does it show?	Broad complex regular tachycardia consistent with VT. Rate approximately 180bpm.	Must identify that broad complex, regular tachycardia or VT

Cardiac Cycle 2016-2-B

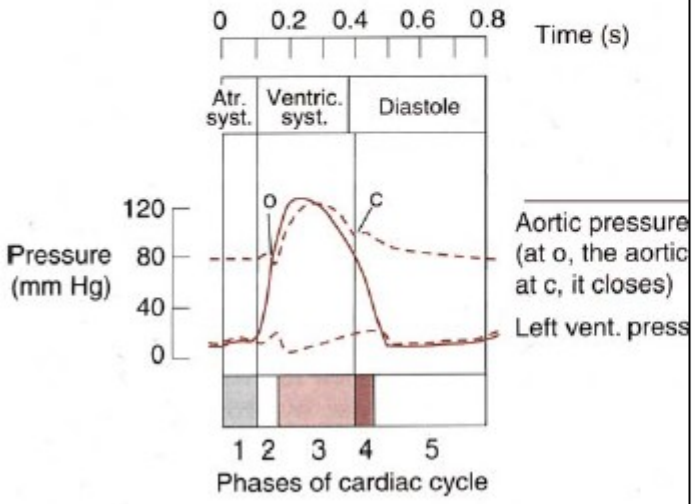
Stem: Moving onto Physiology			

Question 3
Cardiac cycle - ventricular volume relationship to ECG
 	
Subject: Physiology
LOA: 1
	Describe how the waveforms of an ECG relate to the cardiac cycle
	
	Describe the changes in left ventricular volume through the cardiac cycle starting from atrial systole
		Atrial systole starts just after the **P wave**
		Ventricular systole starts near the end of the R wave and ends just after the T wave
		
		Atrial systole
		• Phase 1 – P to R wave
		  o **Small** amount of increased **ventricular filling** due to atrial contraction
		Ventricular systole:
		• Phase 2 = **isovolumetric contraction** – R wave to ST segment (130ml)
		  o Mitral valve closes
		  o Ventricular contraction occurs with no change to volume
		• Phase 3 = **ventricular ejection** – ST segment to end T wave (65ml at end)
		  o Aortic valve opens
		  o Ventricular systole
		Diastole:
		• Phase 4 = **isovolumetric ventricular relaxation**
		  o Aortic valve closes
		• Phase 5 = **ventricular filling**
		Mitral valve opens
			Bold concepts to pass
			Prompt – how do the waveforms relate to atrial and ventricular systole
			
			Bold concepts to pass
			
			Prompt – During atrial systole, what happens to ventricular volume

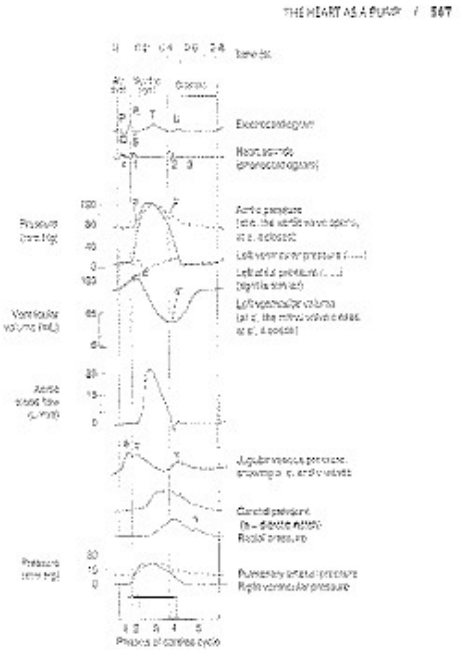
Cardiac Cycle 2015-1-A

Stem: Moving onto Physiology. His blood pressure is 100/60			
Question 2 Cardiac cycle; pressure / volume Subject: Phys LOA: 1	Please draw and label the pressure volume curve of the left ventricle Describe the pressure and volume changes in the left ventricle at the onset of systole *Prompt : What is meant by isovolumetric contraction.* Describe the pressure and volume changes in the left ventricle at the onset of diastole *Prompt : What is meant by isovolumetric relaxation*	Graph with appropriate axis, curves and approximate pressures **(a to b)**Start of systole, **mitral valve closes**. **Isovolumetric contraction** until LVP>Aortic P (80mmHg) then **Aortic valve opens**. ESV 50ml **(c to d)** Momentum of ejected blood is overcome by arterial pressure, then the **Aortic valve closes**. **Isovolumetric relaxation** as the ventricular pressure drops rapidly until below atrial pressure. Then **AV valve opens** to start ventricular filling. EDV 130ml, Stroke volume 70-90ml	Correct graph & bold to pass with reasonable understanding of the loop  **FIGURE 10–2** Normal pressure–volume loop of the left ventricle.

Cardiac Cycle 2010-2

Question 1: **Mechanical events/phases of cardiac cycle.**	List in order, the mechanical phases of the cardiac cycle Please draw the pressure changes in the ventricle that occur during the cardiac cycle	• Atrial systole. • Isovolumetric ventricular contraction. • Ventricular ejection. • Isovolumetric ventricular relaxation. • Ventricular filling.  	Three out of five phases to pass this subsection. Basic diagram and correct pressure

Cardiac Cycle 2009-2

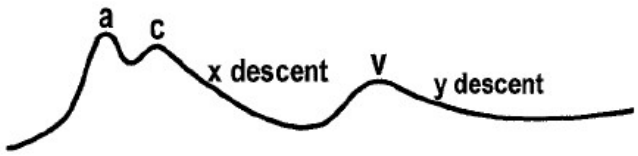
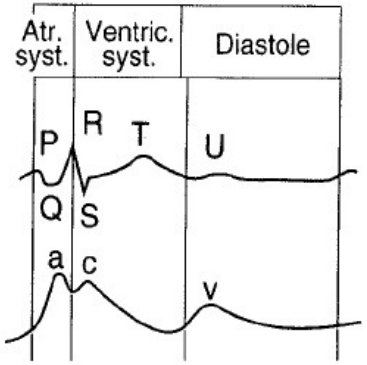
Question 1:	**On this sheet of paper, please draw an ECG trace and, below this, identify the 5 phases of the cardiac (contractile) cycle** **On this sheet of paper, please draw an ECG trace and, below this, demonstrate the left ventricular volume trace. Please give approximate volume values on the y-axis.**	1. Atrial systole 2. Isovolumetric ventricular contraction 3. Ventricular ejection 4. Isovolumetric ventricular relaxation 5. Ventricular filling  	4/5 1. The end-diastolic ventricular volume is approx. 130ml 2. The end-systolic ventricular volume is approx. 50ml [thus about 80ml is ejected by each ventricle per contraction, at rest and the ejection fraction (the percent of the EDV that is ejected with each contraction) is about 65%.

Cardiac Cycle 2009-1

Question 1: Isovolumetric contraction and relaxation Ganong 565-8	In the heart: i) Describe the pressure and volume changes in the ventricles at the onset of systole. Prompt: describe the events that occur around the isovolumetric relaxation phase of systole. ii) Describe the pressure and volume changes in the ventricles at the onset of diastole Prompt: describe the events that occur around the isovolumetric relaxation phase of diastole iii) Additional / offer as an option if candidate struggles with the question: Can you draw the pressure-volume loop of the left ventricle?	i) At the start of **systole mitral and tricuspid valves close**. Ventricular muscle initially shortens very little and pressure rises sharply. AV valves bulge into atria causing a small but sharp rise in atrial/venous pressure. **Isovolumetric contraction** lasts about 0.05 secs until left pressure exceeds aorta (80mmHg), right pressure exceeds pulmonary artery (10mmHg) and so **aortic and pulmonary valves open.** ii) When the momentum of ejected blood is overcome by arterial pressure the **aortic and pulmonary valves close** (setting up transient vibrations). Ventricular pressures drop rapidly until they fall below atrial pressures – **isovolumetric relaxation**. Then **AV valves open** to start ventricular filling. iii) Pressure-volume loop.P566	Core knowledge in bold
Question 2:	i) In the lung, what is surfactant and how	a) Surfactant is a phospholipid. Dipalmitoyl	

Cardiac Cycle 2007-2

QUESTION: 2. Cardiac cycle

Question	Required response [Key items marked with*]	To Pass
Please draw and label a diagram of the jugular venous pressure wave.		Shape with 2 peaks a and v waves
Explain the origins of the fluctuations in this wave.	• The 'a' wave is due to atrial systole as some blood regurgitates into the great veins when the atria contract and venous inflow. • The 'c' wave is the transmitted rise in atrial pressure produced by the bulging of the tricuspid valve into the atria during isovolumetric ventricular contraction. • The 'x' descent is due to increased atrial volume consequent upon the tricuspid valve ring being pulled distally during ventricular emptying. • The 'v' wave mirrors the rise in atrial pressure before the tricuspid valve opens during diastole. • The 'y' descent is due to emptying of the atrium after the tricuspid valve opens during diastole.	a and v wave
How does the ECG relate to the jugular venous pressure wave?		Line up with phases - a/c wave with QRS - v wave with / after T wave

Cardiac Cycle 2005-1

Cardiac cycle	Describe the phases of the cardiac cycle.	Atrial systole; Isovolumetric ventricular contraction; Ventricular contraction; Isovolumetric ventricular relaxation; Ventricular filling.	
	Relate the aortic pressure to the phases of the cardiac cycle	Require that they can either draw the curve or describe the change in pressure in the aorta, including the points at which the aortic valve opens and closes.	

Cardiac Cycle 2003-2

TOPIC: The Cardiac Cycle _____ **NUMBER:** _____ 4b

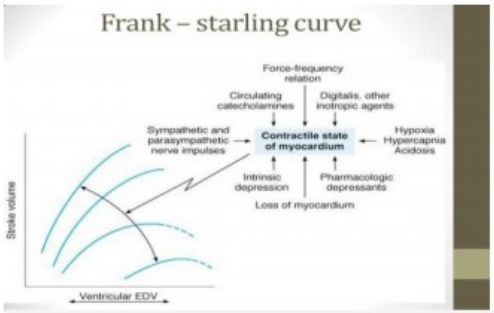
OPENING QUESTION	Describe the mechanical events that occur during the cardiac cycle	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Diastole-slow filling, decreasing in rate	Start during diastole	7/9 to pass
	2 Atrial systole		
	3 Closure of mitral and tricuspid valves		
	4 Isovolumetric Ventricular contraction		
	5 Opening of pulmonary and aortic valves		
	6 Ventricular ejection		
	7 Protodiastole		
	8 Isovolumetric relaxation		
	9 Opening of AV valves and commencement of diastole		
SECOND QUESTION	When do the heart sounds occur?		
POINTS REQUIRED	1		Name valves to pass
	2		
	3		
	4		
	5		
	6		
	7		

Cardiac Function 2006-2

TOPIC: Cardiac Function_____ NUMBER: 2 _____

OPENING QUESTION	What are the determinants of myocardial oxygen cnsumption?	PROMPTS	COMMENTS
POINTS REQUIRED	1 Heart rate	1	2/3 = pass
	2 Wall tension	2	
	3 Myocardial contractility	3	
SECOND QUESTION (if needed)	What are the changes in cardiac function with exercise and how these mediated?		
POINTS REQUIRED	1 Rate and stroke volume	1	2/3 = pass
	2 Adrenaline and sympathetic discharge	2	
	3 Venous return	3	
THIRD QUESTION (if needed)	What are the physical laws involved?		
POINTS REQUIRED	1 Starling	1	½ = pass
	2 La Place P =2T/R	2	

Cardiac Output 2017-2-C

Stem: Moving on to Physiology.			
Question 2 **Subject: Physiology** Cardiac Output LOA: 1	What two factors determine cardiac output?	**CO = HR x SV** **SV** is related to preload and afterload of the heart and the intrinsic contractility of the myocardial cells. **HR** – sympathetic versus parasympathetic stimulation.	**Bold**
	Can you draw a graph to show the Frank Starling law as it relates to cardiac muscle?	 The dashed lines indicate portions of the ventricular function curves where maximum contractility has been exceeded;	Correctly draws and labels curve and able to discuss reason for dotted lines.
	What factors shift the Frank Starling curve?	Circulating catecholamines Inotropes (caffeine, theophylline, digitalis) Sympathetic input *All shift the curve up and to the left.* Acidosis/Hypercarbia/Hypoxia Vagal/parasympathetic stimulation Pharmacological depressants (quinidine, procainamide & barbiturates) Intrinsic depression (with heart failure) *All shift the curve down and to the right.* (The causes of this depression are not fully understood but may reflect down-regulation of β-adrenergic receptors and associated signaling pathways and impaired calcium liberation from the sarcoplasmic reticulum).	**Two positive** and **two negative** factors.

Cardiac Output 2013-2-C

STEM: Following administration of anti-venom for a snakebite, a 60 yr old man is noted to be hypotensive.
We will begin with Physiology....

TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
Question 1 **PHYSIOLOGY** **Cardiac Output** LOA: **1** (Ganong 24th ed p545-552)	What is cardiac output?	**Output of the heart per unit time.** **HR x SV**	**Bold**
	What factors determine cardiac output?	SV is related to the **preload (degree of stretch prior to contraction) and afterload** (resistance to flow) of the heart and the **intrinsic contractility** of the myocardial cells. HR- Sympathetic vs parasympathetic stimulation.	
	What methods can be used to measure cardiac output?	**Direct Fick method or indicator (or thermal) dilution** Can also measure by **Doppler U/sound** techniques Fick principle; amount of substance taken up by organ per unit time = (A-V conc difference) x blood flow. In the heart can use O_2. LV output =O_2 consumption ml/min/[A_{O2}]-[V_{O2}] (both in ml/**L**) Indicator dilution; substance injected IV and serial sampling in arterial blood performed, log plotted and extrapolated to find circulation time (indicator must not be lost from circulation)	*2 to pass*
	What causes of decreased cardiac output could be causing this man's hypotension?	1) variation in heart rate due to induction of **arrhythmias or heart block** (too fast or too slow) 2) **Reduced preload** (venodilatation with reduced venous return due to anaphylaxis) 3) Increased afterload (not too likely in this case **4) Reduced contractility** (i.e. ischaemia, venoms, drugs)	

Cardiac Output 2011-1

Question 1	1.1 How are cardiac stroke volume and cardiac output related?	CO = SV X HR	Need to know equation to pass
	1.2 What is cardiac preload?	Degree of stretch of cardiac muscle compared to resting length Equivalent to end diastolic volume	Definition to pass
	1.3 What factors affect preload? PROMPT - What are the causes of reduced end diastolic volume (preload)?	**Blood volume** **Change in driving pressure** (pericardial (tamponade), intrathoracic (tension pneumothorax, IPPV)) **Venous return** Sympathetic tone Muscle pump Loss of atrial contraction Venous compression (eg uterus in pregnancy) Reduced cardiac compliance Diastolic dysfunction / infiltrative diseases	2/3 bold to pass

Cardiac Output 2010-2

Question 1: **Cardiac output and its measurement.**	What are the parameters that define cardiac output? What are the factors that influence stroke volume? How can cardiac output be measured?	Cardiac output (CO) = heart rate (HR) X stroke volume (SV) • Preload • Afterload • Myocardial contractility The **Fick principle** states that the amount of a substance taken up by an organ (or by the whole body) per unit of time is equal to the arterial level of the substance minus the venous level (A-V difference) times the blood flow. The principle can be used to determine cardiac output by measuring the amount of O_2 consumed by the body in a given period and dividing this value by the A-V difference across the lungs. $$\text{Output of left ventricle} = \frac{O_2 \text{ consumption (mL/min)}}{[A_{O_2}] - [V_{O_2}]}$$ Whole body O_2 consumption is calculated by collecting expired gas in a spirometer and determining its O_2 content, which is then subtracted from the calculated O_2 content of inspired gas. The arterial O_2 content can be measured in an arterial sample and the mixed venous blood O_2 content is obtained from a pulmonary artery catheter. In the **indicator dilution method**, a known amount of a substance is injected into a vein and the concentration of the indicator in serial samples of arterial blood is determined. The output of the heart is equal to the amount of indicator injected divided by its average concentration in arterial blood after a single circulation through the heart. The cardiac output for that period is calculated and then converted to output per minute. $$\text{Flow} = \frac{\text{amount of indicator injected}}{\text{instantaneous concentration of indicator in arterial blood}}$$ The indicator must, of course, be a substance that stays in the bloodstream during the test and has no harmful or haemodynamic effects. A popular indicator dilution technique is thermodilution, in which the indicator used is cold saline.	Full equation to pass this subsection. 3 to pass this subsection. Basic explanation of either principle.

Cardiac Output 2008-1

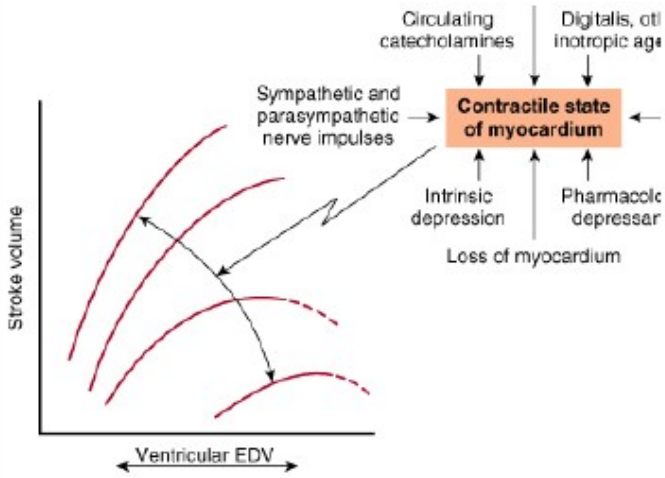
2.3 Factors controlling cardiac output & O_2 consumption Ganong pp 571-576	What factors control cardiac output?	**Cardiac Output = Heart Rate x Stroke Volume** **Heart rate** controlled by cardiac innervation – symp. / parasymp. **Stroke Volume:** • **Afterload** • **Preload** - Starling Curve (Fibre length-tension) **(2 out of 5):** Pericardial pressure Ventricular compliance Atrial filling Blood volume Intrathoracic pressure • **Contractile state (3 out of 7):** Cardiac innervations Hypoxia; hypercapnia; acidosis Drugs +ve & -ve inotropes Circulating catecholamines Loss of myocardium Intrinsic depression (Heart failure) Force-frequency relationship	
	What are the major factors which determine myocardial oxygen consumption?	**(2 out of 3)** Intramyocardial tension Contractile state of myocardium Heart rate (= Ventricular work/beat = SV x MAP)	/2

Cardiac Output 2003-2

TOPIC: Factors controlling cardiac output _____ **NUMBER:** _____ 4a

OPENING QUESTION	What are the parameters that define cardiac output?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 HR x Stroke Vol	1	must pass
SECOND QUESTION	What factors influence stroke volume?		
POINTS REQUIRED	1 afterload	1	3 to pass
	2 preload	2	
	3 contractility	3	
THIRD QUESTION (if needed)	What are the factors that influence contractility?		
POINTS REQUIRED	1 Hypoxia	1	4 of 6 to pass
	2 Drugs +ve / -ve inotropes	2	
	3 pH	3	
	4 sympathetic tone	4	
	5 hypercapnoea	5	
	6 myocardial damage	6	

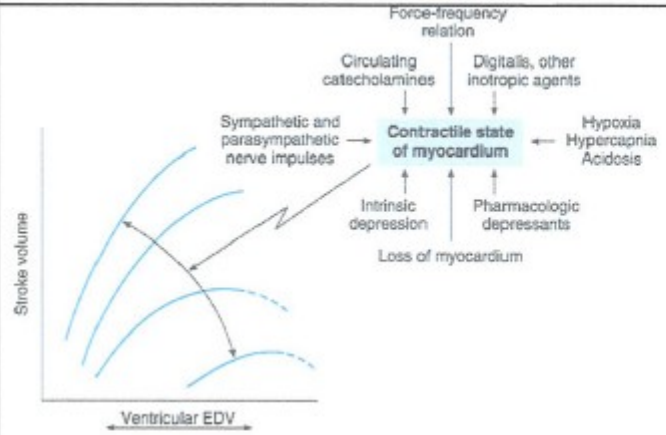
Contractility 2011-1

Question 1	1.1 What factors may reduce myocardial contractility?	**Metabolic abnormalities** • Hypoxia • Severe acidosis • Hypercarbia **Reduced sympathetic tone** Increased parasympathetic tone **Blockade of circulating catecholamines** **Myocardial disease** (muscular dystrophies) or loss **Pharmacological depressants** (antiarrhythmics, Ca channel blockers) Intrinsic depression in heart failure. Hypothermia Abnormal myofibrils (actin, myosin or troponin) Reduced intracellular calcium Reduced levels of c AMP (reduced catecholamines or beta blockade)	3 of 5 of bold to pass
	1.2 How do changes in myocardial contractility alter the relationship between end diastolic volume and stroke volume?	Increasing contractility moves the curve upwards and to the left. Decreasing contractility moves the curve downwards and to the right.  	At least one of the two appropriate directions of movement of the curve to pass. Must get axes correct.

Contractility 2010-2

Question 1:
 	
Myocardial contractility
	Please draw the starling curve
	
	What factors influence myocardial contractility?
		• Positively Inotropic:
		  o Sympathetic stimulation via nerves or circulating catecholamines;
		  o Post-extrasystolic potentiation;
		  o Increased heart rate (small effect);
		  o Drugs such as xanthines, glucagon, cardiac glycosides, adrenergic agents;
		  o Increased myocardial mass (chronic).
		• Negatively Inotropic:
		  o Parasympathetic stimulation (small)
		  o Hypercapnoea, hypoxia, acidosis;
		  o Drugs such as calcium channel blockers, beta-blockers, quinidine, barbiturates;
		  o Cardiac failure (intrinsic myocardial depression;
		  o Cardiomyopathy or infarction.
			Five of the factors listed with at least two each positively or negatively inotropic to pass this subsection.

Contractility 2009-2

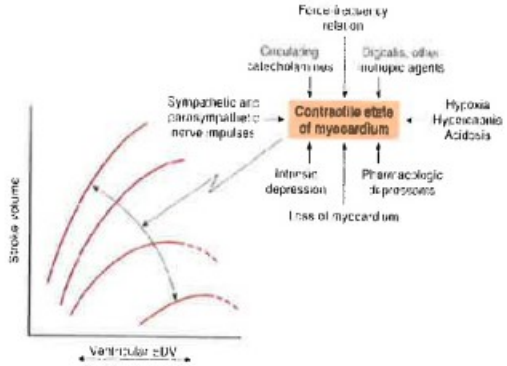
Question 1: a) b)	**Draw Frank Starling curve.** **List the factors that alter contractility**	 Source: Ganong WF: *Review of Medical Physiology*, 22nd Edition: http://www.accessmedicine.com 	1. Sympathetic nerves moves up & left, 2. Parasympathetic nerves move down and right; 3. Force frequency relationships, postextrasystolic potentiation (Ca2+mediated 4. Catechols (via beta 1 and cAMP), digitalis (via Na/K ATPase block) and inotropes increase 5. Hypoxia, hypercarbia, acidosis, quinidine, procainamide, barbs etc depress MC 6. Intrinsic depression with CHF, AMI **Must label FS curve, and 2up /2 down**

Contractility 2006-1

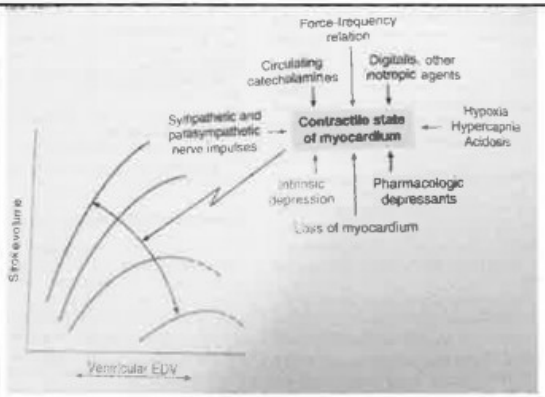
TOPIC: Factors affecting myocardial contractility **NUMBER:** 1

OPENING QUESTION	How does stroke volume relate to end diastolic volume in the heart?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Draw diagram of SV vs EDV Energy of contraction is proportional to the initial length of the muscle fibre.	Prompt: can you describe the Frank Starling law?	Need to describe the relationship to pass.
SECOND QUESTION (if needed)	What factors affect the myocardial contractility?		
POINTS REQUIRED	Neural – symp / parasym, catecholamines, drugs eg digoxin, negative ionotropes, hypoxia, acidosis, temperature, ischemia, muscle mass		Need to mention symp / parasympathetic & 2 more to pass.

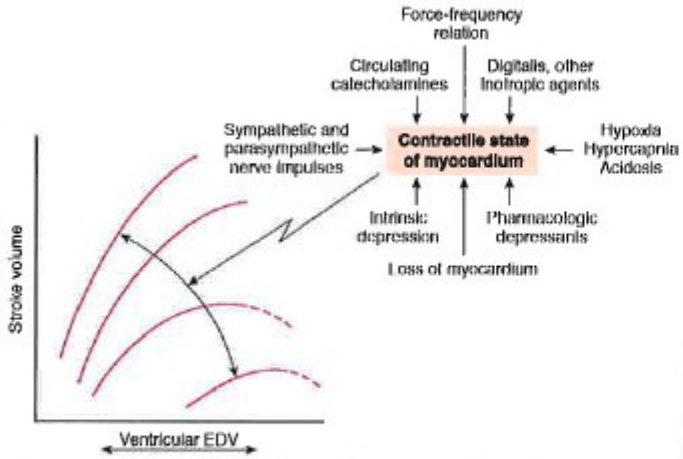
Frank Starling Curve 2016-2-B

Stem: A adult male presents with fever and dyspnea. Starting with Physiology			
TOPIC	QUESTIONS	KNOWLEDGE	NOTES
Question 1 Cardiac contractility (Frank Starling curve) **Subject:** Physiology: LOA: 1	1. Please draw or describe the Frank Starling law as it relates to cardiac muscle.	 Curve of SV against ventricular EDV (energy of contraction is proportional to initial length of cardiac muscle fibre (represented as EDV)	**Pass criteria: Must be able to draw or describe the curve and correctly label/discuss the axes and the dotted lines** Prompt - describe
	2. What factors influence the Frank Starling curve?	Circulating catecholamines, inotropes: (eg digoxin), sympathetic input - (positive, shifting curve up and to left) Acidosis, hypercarbia, hypoxia, vagal /psymp stim, pharm depressants (barbits), intrinsic depression –negative, shifting curve down and to right	**For a pass:** **2 positive factors with correct influence** **2 negative factors with correct influence** Does anything move the curve LEFT/RIGHT?

Frank Starling Curve 2015-2-A

Moving on to Physiology			
Question 2 Frank- Starling Curve **Subject:** Phys LOA: 1	1. Please draw the Frank Starling curve as it relates to human cardiac muscle Prompt: What effect does EDV have on SV?		Q2.1 – to pass must be able to draw the FS curve including the hump and correctly label axes (SV or Pressure on y axis)
	2. What factors influence the Frank-Starling curve?	2. Circulating catecholamines; inotropes, hypoxia, hypercarbia, acidosis, pharmacol depressants; loss of myocardium; intrinsic depressing; symp NS & PSym, fluid status	Q2.2 – 4 factors with correct influence

Frank Starling Curve 2012-2

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOA: 1	Draw or describe the Frank-Starling law as it applies to human cardiac muscle? What factors influence the FS curve?	Curve of SV against Ventricular EDV  Circulating catecholamines; inotropes (inc dig); hypoxia, hypercarbia, acidosis – (negative); pharmacological depressants; loss of myocardium (-ve); intrinsic depression; sympathetic and parasympathetic input	Draw or describe a curve and + explain 2 +ve, 2 -ve

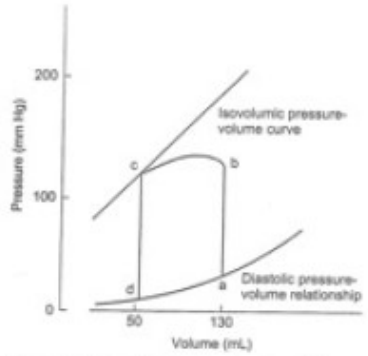
Myocardial O2 Demand 2011-2

Question 1 LOA: 1	1.What factors influence myocardial oxygen consumption?	1. intramyocardial **tension** which is dependant Pressure –after load, systolic pressure, contractility Radius – preload Wall thickness 2. **contractile state of the heart** ionotropy 3. **HR** ALSO -(cardiac work α SV x MAP) Pressure load increases O_2 consumption more than volume load	Need bold to pass
	2.How does decreasing a patient's heart rate improve symptoms of angina?	1. **decreasing HR** decreases O2 demands 2. at a slower heart rate there is more time for coronary **circulation which occurs in diastole**	Prompt: What happens if a patient has tachycardia?

Myocardial O2 Demand 2009-1

Question 1: Factors determining myocardial O2 demand Ganong pp 575-76	i) What factors determine myocardial oxygen demand?	i) **1) Heart Rate** **2)Intramyocardial Tension** **3)Contractile state of the myocardium** OR 1) **Stroke Volume** **2) MAP**	Core knowledge in bold. 2 out of 3
	ii) What effect does increase in preload and afterload have on myocardial O2 demand? Prompt: How does it work?	**i) Both increase** Ventricular work per beat correlates to O2 consumption Work = SV x MAP Stroke work LV is 7x that of RV Theoretically, volume changes and pressure changes should affect myocardial O2 consumption equally. HOWEVER, pressure work produces a greater increase in O2 consumption than does volume work. Reason not well understood Net result ; **Changes in afterload have greater effect than changes in preload.** Tension in the wall of a hollow viscus is proportional to the radius of the viscus. Myocardial fibres are stretched with increased stroke volume in a dilated heart. Increased radius of dilated heart increases wall tension which explains the increased oxygen consumption	Core knowledge in bold. Both increase Changes in afterload have greater affect than changes in preload

Pressure Volume Loop 2014-2-A

Stem: Now we will move on to Physiology			
Question 3 Pressure Volume Loop (pp 540-550) **Subject:** Phys LOA: 1	1. What is the stroke volume in a normal adult at rest? 2. Please draw and label the pressure volume loop of the left ventricle. Prompt: Describe the changes in pressure and volume that occur during systole and diastole.  **FIGURE 30–2 Normal pressure–volume loop of the left ventricle.** During diastole, the ventricle fills and pressure increases from d to a. Pressure then rises sharply from a to b during isovolumetric contraction and from b to c during ventricular ejection. At c, the aortic valves close and pressure falls during isovolumetric relaxation from c back to d. (Reproduced with permission from McPhee SJ, Lingappa VR, Ganong WF (editors): *Pathophysiology of Disease*, 6th ed. McGraw-Hill, 2010.)	**Stroke vol – 70-90ml** A. Start of systole: mitral (and Tric) valves close **Isovolumetric contraction** til LVP > Aortic P (80mmHg) Aortic (and Pulmonary) valves open. B. Ventricular ejection (rapid at first) peak pressure 120mmHg End systole: momentum of ejected blood overcome by aortic pressure. C Aortic valve closes. ESV – 50ml C-D. **Isovolumetric relaxation** LVP drops below atrial pressure – mitral valve opens – ventricle begins to fill (rapidly at first) EDV – 130ml	Bold to pass Correct graph needed to pass. Need to demonstrate reasonable understanding of the loop.

Pressure Volume Loop 2012-2

TOPIC	QUESTIONS	KNOWLEDGE (**essential in bold**)	NOTES
Question 1 LOA: 1	Please draw a pressure-volume loop for the left ventricle.	▪ a → b isovolumetric contraction ▪ b → c ventricular systole ▪ c → d isovolumetric relaxation ▪ d → a ventricular filling	**The candidate must be able to label the axes and draw a reasonable pressure-volume loop to pass this question.**
	Please relate the phases of the cardiac cycle to this pressure-volume loop.	▪ 75% along the line'd' to 'a' and closer to 'a' atrial systole (phase 1) occurs. ▪ The mitral valve closes at 'a' and the pressure rises sharply from 'a' to 'b' during isovolumetric ventricular contraction (phase 2) ▪ The aortic valve opens at 'b' and the pressure rises to a plateau and volume falls from 'b' to 'c' during ventricular ejection (phase 3) ▪ The aortic valve closes at 'c' and pressure falls from 'c' to 'd' during isovolumetric ventricular relaxation (phase 4) ▪ At 'd' the mitral valve opens and diastole commences (phase 5) from 'd' towards 'a'.	**The candidate must be able to relate three of the five phases of the cardiac cycle to the pressure-volume loop.**