Question 1 Answer:

A. The diffusion coefficient of beta-lactamase can be calculated by the Stokes-Einstein equation:

$$D = \frac{KT}{6\pi r\eta}$$

r = 1.7 nm, $\eta = 1\, mPa * s$, K is the Boltzmann's constant 1.38 * 10^{-23} J*K^{-1}, T is absolute temperature in K, and T = 298.15 K for t = 25 C. So we have,

$$D = \frac{1.38 * 10^{-23} J * K^{-1} * 298.15K}{6 * \pi * 1.7 * 10^{-9} m * 10^{-3} Pa * s} = \frac{1.38 * 10^{-23} * 298.15}{6 * \pi * 1.7 * 10^{-12}} m^2 s^{-1} = 1.28 * 10^{-10} m^2 s^{-1}$$

B. According to the Fick law of diffusion,

$$J = PA(C_A - C_B)$$

Since J_1=PA(11 mM – 1 mM), we know that the new J_2 = PA(30 mM -10 mM) = $2J_1$ = 0.002 mmol/hr.

C. Osmotic pressure can be calculated by $\pi = gC\sigma RT$. Since protein (beta-lactamase) and Na+ and Cl- are all non-permeable, so we have $\sigma = 1$ for both side. So we have,
π_{diff} = g_1C_1RT – g_2C_2RT = 2*0.5 mM*RT – 1*1 mM*RT = 0
So there will be not osmotic pressure across the wall of each vesicle, therefore there will be no net water flow.

Question 3 Answer:

A. The flow velocity of blood can be calculated using V = Q/A, where Q is blood flow rate, and A is vessel cross section area. First we can calculate Q via cardiac output:

Q = SV*HR = (125ml-50ml)/beat*(67 beats/min) = 5025 ml/min

Then we can calculate A as,

$$A = \pi r^2 = \pi \left(\frac{3.2}{2}\right)^2 = 8.04\ cm^2$$

Then the blood flow velocity is,

V = Q/A = 624.81 cm/min = 10.4 cm/s

B. The flow rate in this aorta does not change, so the new velocity at the site where the aorta narrows is:

V_{new} = Q/A_{new} = Q/(0.25A) = 4*10.4 cm/s = 41.6 cm/s

C. The aorta's flow rate does not change, only resistance changes, the new resistance is a parallel combination of two resistances. Since we have,

$$\frac{Q_A}{Q_B} = \frac{R_B}{R_A} = \frac{r_A^4}{r_B^4} = \frac{1.0^4}{0.6^4} = 7.72$$

Since $Q_A+Q_B = Q$, and Q does not change, it is easy to see that $Q_A = 0.886\ Q$, $Q_B = 0.114\ Q$. Since $r_A = 1.0$ cm and $r_B = 0.6$ cm, so we have,

$$A_A = 3.14\ cm^2,\ A_B = 1.13\ cm^2$$

Then $V_A = Q_A/A_A = 0.886 * 5025$ ml/min /3.14 $cm^2 = 23.6$ cm/s, and $V_B = Q_B/A_B = 0.114 * 5025$ ml/min/1.13 $cm^2 = 8.45$ cm/s.

MT1 Solutions

Thursday, February 23, 2017 10:22 PM

2. A. For the current of an x ion at equilibrium potential,

$$I_x = g_x(E_{mem} - E_x).$$

If we want the current contribution of Cl^- to be zero, then

$$0 = g_{Cl^-}(E_{mem} + E_o - E_{Cl^-})$$

$$\Rightarrow E_o = E_{Cl^-} - E_{mem}, \text{ where } E_o \text{ is the added potential}$$

Using the Chord Conductance equation, we have $E_{mem} = \dfrac{\sum_i g_i E_i}{\sum_i g_i}$

Let's calculate the equilibrium membrane potentials for all the ions:

$$E_i = -\frac{60\,mV}{z}\log\left(\frac{C_i}{C_o}\right)$$

$$E_{Na^+} = -\frac{60\,mV}{1}\log\left(\frac{14}{140}\right) = 60\,mV$$

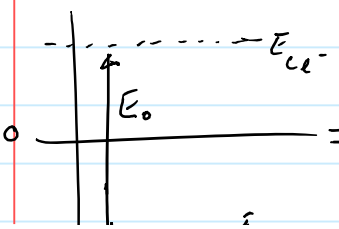
$$E_{K^+} = -\frac{60\,mV}{1}\log\left(\frac{120}{4}\right) = -88.6\,mV$$

$$E_{Cl^-} = -\frac{60\,mV}{-1}\log\left(\frac{105}{10}\right) = 61.3\,mV$$

$$E_{Ca^{2+}} = -\frac{60\,mV}{2}\log\left(\frac{0.0001}{2.5}\right) = 132\,mV$$

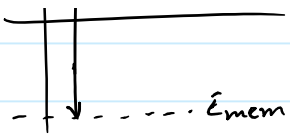
Note: We can also ignore Ca^{2+} contribution since $g_{Ca^{2+}} = 0$

$$E_{mem} = \frac{g_{Na^+}E_{Na^+} + g_{K^+}E_{K^+} + g_{Cl^-}E_{Cl^-}}{g_{Na^+} + g_{K^+} + g_{Cl^-}}$$



$$= \frac{5(60\,mV) + 100(-88.6\,mV) + 10(61.3\,mV)}{5 + 100 + 10}$$

$= \dfrac{\text{[illegible]}}{5 + 100 + 10}$



$E_{mem} = -69.1\ mV$

$\Rightarrow E_o = 61.3mV - (-69.1\ mV)$

$\boxed{E_o = 130.4mV}$

$E_{mem}' = \dfrac{5(60mV) + 100(-88.6mV) + 15(61.3mV)}{5 + 100 + 15}$

$= -63.7\ mV$

$E_o' = 61.3mV - (-63.7\ mV)$

$\boxed{E_o' = 125mV}$

B. At steady state, $\sum I_i = 0$

$\Rightarrow I_{K^+} + I_{Na^+} + \cancel{I_{Cl^-}}^{\,0} + \cancel{I_{Ca^{2+}}}^{\,0}$ (since $g_{Cl^-} = g_{Ca^{2+}} = 0$)

$\Rightarrow \boxed{\dfrac{I_{K^+}}{I_{Na^+}} = -1}$

Or Computationally:

E_{mem} w/o Cl^- $= \dfrac{g_{Na^+}E_{Na^+} + g_{K^+}E_{K^+}}{g_{Na^+} + g_K E_{K^+}}$

$\dfrac{5(60mV) + 100(-88.6\ mV)}{5 + 100}$

$\Rightarrow E_{mem} = -81.5\ mV$

$\Rightarrow \dfrac{I_{K^+}}{I_{Na^+}} = \dfrac{g_{K^+}(E_{mem} - E_{K^+})}{g_{Na^+}(E_{mem} - E_{Na^+})}$

$$= \frac{100\,(-81.5\text{mV} - (-88.6\text{ mV})}{5\,(-81.5\text{mV} - 66\text{ mV})}$$

$$\Rightarrow \frac{I_{K^+}}{I_{Na^+}} = -1$$

C. Since the conductance of Ca^{2+} is 0, meaning it cannot cross the cell membrane, then it doesn't contribute to the resting membrane potential.
⇒ Changing Ca^{2+} concentration will not change the resting membrane potential. (factor = 1)

4. A. Using Fick principle:

$$C.O. = \frac{O_2 \text{ consumption}}{[O_2]_A - [O_2]_V}$$

$$= \frac{390\text{ mL } O_2/\text{min}}{0.20\text{ mL } O_2/\text{mL blood} - 0.15\text{ mL } O_2/\text{mL blood}}$$

$$C.O. = 7800\text{ mL/min}$$

$$1 \text{ cardiac cycle} \Rightarrow \frac{1 \text{ beat}}{1/2\text{ s}}$$

$$\Rightarrow HR = \frac{1 \text{ beat}}{1/2\text{ s}} \times \frac{60\text{ s}}{1\text{ min}}$$

$$HR = 120\ \frac{\text{beats}}{\text{min}}$$

$$CO = (HR)(SV)$$

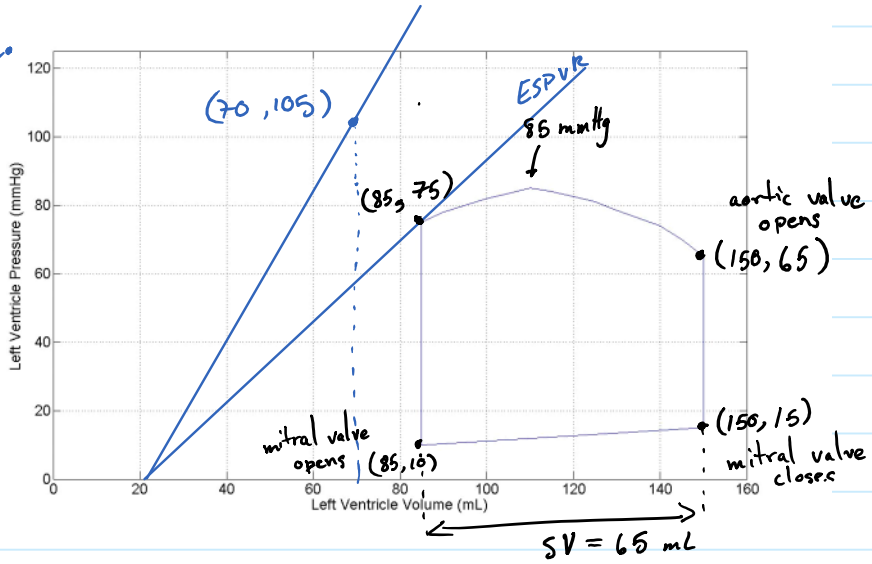
$$\Rightarrow SV = \frac{CO}{HR}$$

$$= \frac{7800\text{ mL/min}}{120\text{ beats/min}}$$

120 beats/min

SV = 65 mL/beat, or just 65 mL

B. C.



Appx SW by taking max & mins for pressure and volume

⇒ $SW = \Delta P \cdot SV$

$= (85 \text{ mmHg} - 10 \text{ mmHg})(65 \text{ mL})$

$SW = 4875 \text{ mmHg mL}$

Note: Any method that appx PV loop area is fine.

C. See chart in part B. When this patient was healthy, his/her ESPVR had a larger slope.

⇒ This patient's heart has lost contractility/ has negative inotropic effect.

PART II: MULTIPLE CHOICE (5 points each) – clearly circle or write your answer.

1. Why does a neuronal action potential only travel in one direction?

B. Recently depolarized regions of the axon are temporarily unable to conduct a new action potential.

2. A patient receives 1L of intravenous normal saline, and within seconds to minutes, her ventricular stroke volume increases. Why?

C. The extra fluid volume increases actomyosin overlap and passive stretch within the myocardium.

3. Pressure-volume loop analysis of a patient reveals that the left ventricular pressure and volume at the end of systole are 120 mmHg and 70 mL, respectively. Suppose the patient is given a drug that increases systemic vascular resistance. Which new set of end-systolic pressure/volume values would you most expect to see?

C. 140 mmHg, 90 mL. Since SVR increases, we expect MAP to increase (MAP = CO*SVR). This means that the aortic valve opens at a higher point, which is an afterload effect. Following the ESPVR line means we'll be at a higher end-systolic pressure and volume.

4. Suppose that the patient in the previous question was instead given a drug that increases inotropy (contractility). If the initial left ventricular end-systolic pressure and volume are 120 mmHg and 70 mL as before, what new pressure/volume values would you most expect to see following administration of this drug?

A. 125 mmHg, 45 mL. In this case, since contractility increases, we expect a ESPVR with a higher slope, since more pressure can be generated with less volume. It follows that we'll generate a higher end-systolic pressure at a lower volume.

5. A patient with ~20 years of poorly controlled systemic hypertension presents with shortness of breath, fatigue, and dizziness. Echocardiography reveals concentric left ventricular hypertrophy. What characteristic electrocardiography (EKG) feature is **most consistent** with the clinical presentation and echocardiography findings?

A. Increased QRS amplitude. The QRS complex is generated during ventricular contraction/depolarization. An enlarged LV (more mass/thicker walls) will generate a higher QRS amplitude.

6. In general, how do the flow rates of blood (volume/time) leaving the right and left ventricles during systole compare?

A. Since the left and right ventricles are in series, their outflows must equal at steady state (i.e., conservation of mass).

7. What characteristic tissue change is most commonly associated with acquired aortic valve disease?

B. Valve calcification. This is from the guest lecture, but in general, when a valve calcifies, it gets thicker and stiffens, making it difficult to function properly.

8. A patient is placed on a diuretic, which increases urinary output and reduces extracellular fluid volume. What hormonal change would you least expect to see as his

body responds to the drug?

D. Reduced angiotensin II. Reduction in ECF means there will be a reduction in blood volume, which decreases blood pressure. The RAAS works to retain Na+ and increase BP. All of the products of RAAS increase, so reduced angiotensin II is least expected.

9. Certain kidney diseases can cause loss of plasma protein through the urine. How would this protein loss be expected to influence Starling forces within capillaries?

A. Reduced absorption due to reduced capillary oncotic pressure.

10. Suppose a patient with normal cardiac function was administered a drug that had the side effect of increasing conduction speed through the atrioventricular node from its baseline value of 0.01 m/s to a new value of 0.05 m/s. Which of these effects would you be least likely to see?

D. Increased stroke volume. The AV node conducts longer so that the ventricles have time to fill. If this is shortened, the ventricles will contract sooner; we are likely to see a decrease in stroke volume, not an increase.