MIDTERM 1 SOL'N

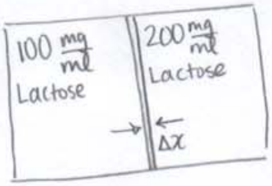
$[Pa] = \left[\frac{kg}{m \cdot s^2}\right]$

1

A) $D = \dfrac{kT}{6\pi R \eta} = \dfrac{\left(1.38\times10^{-23}\ \frac{m^2 kg}{s^2 K}\right)(310\,K)}{6\pi\,(0.42\times10^{-9} m)(0.65\times10^{-3}\,Pa\,s)}$

$\boxed{D = 8.31\times10^{-10}\ m^2/s}$

B)



$\Delta x = 0.5\,mm \qquad K = 0.9$

$P = \dfrac{KD}{\Delta x} = \dfrac{(0.9)(8.31\times10^{-10}\ m^2/s)}{(0.5\times10^{-3}\,m)}$

$\dfrac{J}{A} = P\,\Delta C = (1\times10^{-6}\,m/s)(292\ mM)$

$= 4.37\times10^{-4}\ \dfrac{mmol}{s\,m^2}\times\dfrac{1\,m^2}{(100\,cm)^2}$

$\boxed{\dfrac{J}{A} = 4.37\times10^{-5}\ \dfrac{mmol}{s\,cm^2}}$

$C_1 = 100\ \frac{mg}{mL}\times\frac{1\,mL}{1\times10^{-3}L}\times\frac{1\times10^{-3}g}{1\,mg} = 100\ \frac{g}{L}$

$100\,g/L \times \frac{1\,mol}{342\,g} = 0.292\ M$

$= 292\ mM$

$C_2 = 200\ \frac{g}{L}\times\frac{1\,mol}{342\,g} = 584\ mM$

$[mM] = \frac{mmol}{m^3}$

C) $\dfrac{J}{A} = \dfrac{K D \Delta C}{\Delta x} = \dfrac{(0.9)(8.31\times10^{-6}\ cm^2/s)}{0.05\ cm}\left(200-100\ \frac{mg}{mL}\right)\times\frac{1}{3}\times\frac{1}{342\ \frac{mg}{mol}}\times\frac{1\,mL}{1\,cm^3}$

$= 1.458\times10^{-5}\ \dfrac{mmol}{cm^2 s}$

b/c now the radius of $(Gal-Glc)_3$ is three times larger

D) $\pi = g[C]\sigma RT \qquad \sigma = 0.75 \qquad [C]_1 = 0.292\ M$

$g = 1^{*} \qquad [C]_2 = 2\times[C_1] = 0.584\ M$

* g=1 because lactose does <u>not</u> dissociate in water.

$\pi_{21} = (1)(0.584\,M - 0.292\,M)(0.75)\left(0.0821\ \frac{L\,atm}{mol\,K}\right)(310\,K)$

$= 5.58\ atm$

This should be applied to the 200 mg/mL solution.

2 A) At T= 37°C, we can use: $E = \frac{-60mV}{Z} \log\left[\frac{C_i}{C_o}\right]$

$E_{Na^+} = \frac{-60mV}{1} \log\left[\frac{14}{4}\right] = -32.64mV$

$E_{K^+} = \frac{-60mV}{1} \log\left[\frac{120}{140}\right] = 4.02\ mV$

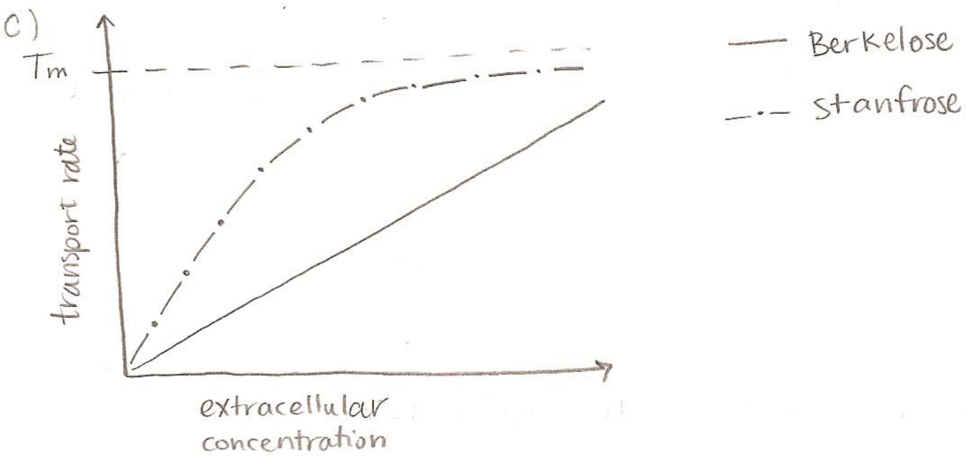
$E_{Cl^-} = \frac{-60mV}{-1} \log\left[\frac{10}{100}\right] = -60\ mV$

$E_{Ca^{++}} = \frac{-60mV}{2} \log\left[\frac{0.0001}{2.5}\right] = 131.9\ mV$

B) $E_m = \frac{g_{Na}E_{Na} + g_K E_K + g_{Cl} E_{Cl} + g_{Ca} E_{Ca}}{g_{total}} = \frac{8(-32.64) + 4(4.02) + 2(-60) + 1(131.9)}{8+4+2+1}$

$\boxed{E_m = -15.5\ mV}$

C)



Since Berkelose is diffusing simply, we expect a linear relationship b/w its extracellular concentration and transport rate. Stanfrose is diffusing by facilitated transport, so the relationship is not neccesarily linear, and will max out at the transport maximum.

Midterm Problems

MIDTERM 1 SOL'N

$\boxed{3}$ $R_{brain} : R_{kidneys} : R_{heart} : R_{muscle} : R_{GI} : R_{skin}$

1 : 2 : 3 : 4 : 6 : 8

A) +5 $0.02 : 0.04 : 0.06 : 0.08 : 0.12 : 0.16 \leftarrow \left[\frac{mmHg \cdot min}{mL}\right]$

B) Aorta $\longrightarrow$ Vena Cavae

$Q = 3.6\ L/min$ $\quad R_{tot} = 0.00842$

$\frac{1}{R_{tot}} = \frac{1}{0.02} + \frac{1}{0.04} + \frac{1}{0.06} + \ldots$

$R_{tot} = \frac{1}{118.75} = 0.00842\ \frac{mmHg\ min}{mL}$ +3

$\Delta P = QR = \frac{3.6\cancel{L}}{\cancel{min}} \cdot \frac{0.00842\ mmHg\ \cancel{min}}{\cancel{mL}} \cdot \frac{1000\cancel{mL}}{1\cancel{L}}$

$= 30.3\ mmHg$ +2

C) $Q =$ $\quad R_{GI} = 0.12\ \frac{mmHg \cdot min}{mL}$

$\boxed{Q = \frac{\Delta P}{R}} = \frac{30.3\ \cancel{mmHg}}{\frac{0.12\ \cancel{mmHg} \cdot min}{mL}} = 252.5\ mL/min$ to GI tract

+2 +3

D) $3\ cP = 3\ mPa \cdot s = \eta$

$r = 0.9\ cm = .009\ m$ $\quad 3.6\ L/min = 3600\ cm^3/min$

$\boxed{\frac{\Delta P}{l} = \frac{8\eta}{\pi r^4} Q} = \frac{8\ (0.003\ Pa \cdot s)}{\pi\ (0.9\ cm)^4}\ 3.6\ L/\cancel{min}\left(\frac{1000\ cm^3}{1\ L}\right)\left(\frac{1\ \cancel{min}}{60\ \cancel{sec}}\right) = 0.698\ Pa/cm$

+2

$\frac{1\ m}{100\ mm}$ $\quad$ +3 $= 70\ Pa/m = 0.070\ \frac{mPa}{m}$

$= 700\ \frac{mPa}{cm}$

$= \frac{0.005235\ mmHg}{cm}$

E) β-blocker to arterioles in GI tract will prevent smooth muscle relaxation that dialate the arterioles, allowing less blood to enter the GI tract b/c of increased resistance.

(5)

+5

UNITS +5

+30

$1\ Pa = \frac{N}{m^2} = \frac{kg\ m}{m^2 s^2} = \frac{kg}{m\ s^2}$

$1\ N = \frac{kg \cdot m}{s^2}$

$\frac{8\ (0.003\ Pa \cdot s)}{\pi\ (0.9\ cm)^4} \cdot \frac{3600\ cm^3}{\cancel{min}} \cdot \frac{1\ \cancel{min}}{60\ s} = 0.70\ \frac{Pa}{cm}$

4

$P_c = ?$

$\pi_c = 25\,mmHg$

$\pi_i = 6\,mmHg$

$P_i = 1.0\,mmHg$

$K_f = 0.8 \frac{ml}{min\ mmHg}$ +3 $J = {}^{+}16 \frac{mL}{min}$

A)

$16 \frac{mL}{min} = 0.8 \frac{mL}{min\ mmHg}\left[\left(\underset{P_c}{\overset{40\,mmHg}{X}} - \underset{P_i}{1.0\,mmHg}\right) - \left(\underset{\pi_c}{25\,mmHg} - \underset{\pi_i}{6\,mmHg}\right)\right]$ +5

$\boxed{P_c = \frac{J}{K_f} + (\pi_c - \pi_i) + P_i}$

$\boxed{P_c = 40\,mmHg}$ +5

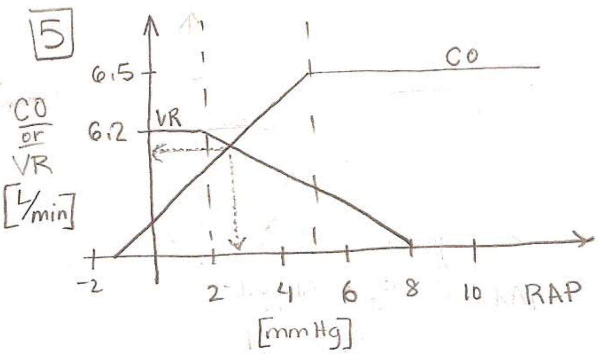
+25

B) i. Reduced production of protein by the liver would decrease oncotic pressure in the capillary (π_c) and increase filtration. +4

ii. Ingesting Sudafed which increases blood pressure, would increase hydrostatic pressure in the capillary (P_c) and increase filtration. +4

iii. During allergic reaction, the surrounding tissue releases histamine, causing π_i to increase, thus filtration would increase. +4

5



CO = VR @:

$CO = 1.1\,RAP + 2 \quad RAP \leq 5$

$VR = -1.0\,RAP + 8 \quad RAP \geq 2$

$\boxed{1.1x + 2 = -1x + 8}$ +5

$2.1x = 6$

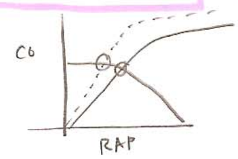
$x = RAP = 2.85$

units +2

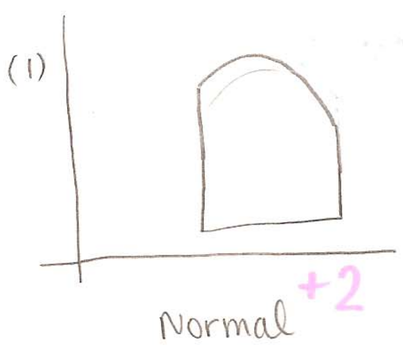
$\boxed{RAP = 2.85\,mmHg \quad \frac{VR}{CO} = 5.135\ L/min}$ +1 +2

Stroke Vol $= \frac{CO}{HR} = 5.135\ L/min \div 72\ b/min = \boxed{0.0713\ L/beat}$ +3 $= \frac{71.3\,mL}{beat}$ +2

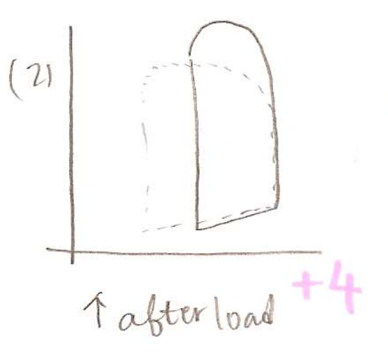
A positive inotropic agent would increase CO. +5



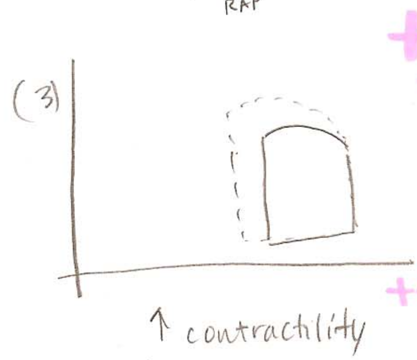
+30



Normal +2



↑ afterload +4



↑ contractility +4

MIDTERM 1 SOL'N

6 Want $x'(t) + a\,x(t) = b$

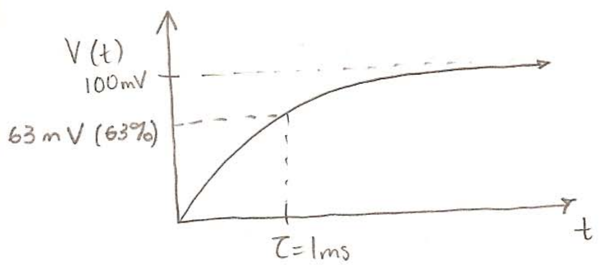
A) $v'(t) + \frac{g}{c} v(t) = \frac{g}{c} E \Rightarrow v(t) = x_0 e^{-at} + \frac{b}{a}(1 - e^{-at})$

$g/c = 1/\tau \qquad \Rightarrow v(t) = V_0 e^{-g/c\, t} + \frac{gE/c}{g/c}(1 - e^{-g/c\, t})$

$V_0 = 0$ at $t = 0$

$\Rightarrow \boxed{V(t) = E(1 - e^{-t/\tau})}$

B) $V(t) = 100\,mV\,(1 - e^{-t/1ms})$



C) If ion channels were reduced by half, $g \rightarrow \frac{1}{2}g$, thus if $\tau = c/g$, then $\tau' = 2c/g$. $\tau' = 2\tau$.

τ will double.

7 A. Acetylcholine (ACh); In myasthenia gravis, antibodies are produced against ACh. Result is muscle weakness b/c ACh receptors are blocked + depolarization of the motor end plate will not occur (i.e. no action potentials).

B. Passive tension is mainly executed by titin, a spring like protein (see p.34). It refers to the tension in the muscle when there is no active contraction.

C. The total cross sectional area of the total capillary bed is much larger than the arteries.

→

D. An L-type Ca^{2+} channel blocker would stop Ca^{2+} flow into the cell during an atrial / ventricular action potential. The cell will repolarize quicker, and there would be little or no plateau phase.

E. $P = \frac{2HT}{R}$, $\Delta P = QR$. $\uparrow R \Rightarrow \uparrow P|_{rt\ ventricle} \Rightarrow \uparrow H$.

F. Holding breath increases pressure at the baroreceptors, which will set off a chain of rx's to reduce heart rate (pg. 159).

G. P_{O_2}. Chemoreceptors exist in carotid bodies and aortic bodies which can sense P_{O_2}. If $P_{O_2} \downarrow$, you get vasoconstriction which increases resistance (see pg. 162).