BMED 3100 Systems Physiology Test 3	
Bill 5100 Systems Thysiology Test 5	
	Last Name, First Initial
October 31, 2013	
Be sure to read all instructions and questions carefully. Be brief in your answers. Write clearly. Backs of pages will not be graded.	
Honor Pledge All students are required, when requested, to attach the turned in for a grade in any course at Georgia Institute of	
On my honor, I pledge that I have neither given nor recepreparation of this assignment.	eived inappropriate aid in the
KEY Signature	
Name (Printed clearly)	

## BMED 3100 Systems Physiology Test 3 Circle the best answer (2 pts ea)

1. When an arteriole dilates, resistance to blood flow

**INCREASES** DECREASES, or STAYS THE SAME.

2. Cardiac myocyte contraction requires Ca<sup>2+</sup> to enter the cytosol from the

**ISF** SR

3. Increased parasympathetic stimulation to the heart causes left ventricular contractility to:

**BOTH ISF & SR** 

**INCREASE DECREASE** STAY THE SAME

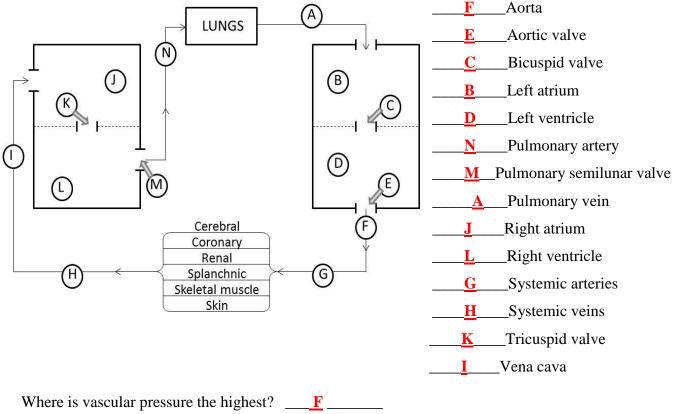
4. Increased **parasympathetic stimulation** to the bronchicles causes airway diameter to:

DECREASE **INCREASE** STAY THE SAME or

5. During a lab experiment you open the chest cavity of an anesthetized frog and observe the beating heart. You apply a solution of a  $\beta_1$ -adrenergic agonist to the heart. Predict the effect on heart rate:

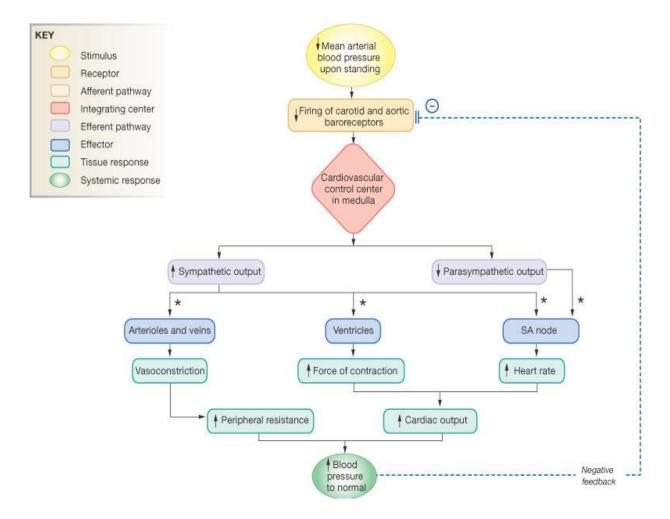
> **INCREASE** DECREASE STAY THE SAME or

6. Match the letter below to the correct anatomic feature on the right or description on the bottom. Write letter on the line. (2 pts ea)



Where is the SA node located?

7. Draw a concept map that describes the baroreceptor reflex when a normal person suddenly goes from a supine to a standing position and gets dizzy for a few seconds and then recovers. (18 pts)



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8. (18 pts) Pulmonary hypertension is due to increased resistance of the pulmonary artery and ultimately results in right ventricular failure if left untreated, as well as edema in the lower extremities. Consider a person who develops pulmonary hypertension and has the following measurements available:

Parameter	Value 1 year pre-diagnosis	Value at diagnosis of Pulmonary Hypertension
Systolic pressure (aorta)	124 mmHg	125 mmHg
Diastolic pressure (aorta)	82 mmHg	80 mmHg
R-R interval	800 msec	800 msec
Left ventricular end-diastolic volume	140 mL	145 mL
Left ventricular end-systolic volume	70 mL	72 mL
Mean pulmonary artery pressure	15 mmHg	30 mmHg
Right atrial pressure	2 mmHg	10 mmHg
Left atrial pressure	5 mmHg	5 mmHg
Capillary pressure venous end (P <sub>c</sub> ,	15 mmHg	26 mmHg
ven)		
Capillary pressure arterial end (P <sub>c</sub> , art)	35 mmHg	45 mmHg
Fluid pressure in ISF (P <sub>i</sub> )	2 mmHg	2 mmHg
Oncotic pressure in capillary $(\pi_c)$	25 mmHg	25 mmHg
Oncotic pressure in ISF $(\pi_i)$	2 mmHg	4 mmHg

a) Calculate the <u>Pulmonary vascular resistance</u> BEFORE and AFTER the diagnosis of Pulmonary Hypertension.

Units (1 pt); Correct values for necessary calculated components (hear rate, SV, Q) (1 pt ea)

HR = 1 beat/800 msec = 1.25 beats per second = 75 bpm

 $SV_{pre} = EDV-ESV = 70 \text{ mL}$ 

CO<sub>pre</sub>=HR\*SV = 75 bpm \* 70 mL = 5250 mL/min = 5.25 L/min

 $SV_{diag} = EDV-ESV = 75 \text{ mL}$ 

CO<sub>diag</sub>=HR\*SV = 75 bpm \* 75 mL = 5625 mL/min = 5.625 L/min

before:  $R = \Delta P/Q = (mean pulmonary artery pressure-left atrial pressure)/CO$ 

= (15 mmHg - 5 mmHg)/(5250 mL/min) = 0.0019 mmHg/mL/min

after: = (30 mmHg - 5 mmHg)/(5625 mL/min) = 0.0044 mmHg/mL/min

b) Calculate the <u>Systemic vascular resistance</u> BEFORE and AFTER the diagnosis of Pulmonary Hypertension.

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Necessary values: MAP_{pre} = 82 + 1/3(124-82) = 96 \text{ mmHg}

MAP_{diag} = 80 + 1/3(125-80) = 95 \text{ mmHg}
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before:  $R = \Delta P/Q = (MAP-right atrial pressure)/CO$ 

= (96 mmHg - 2 mmHg)/(5250 mL/min) = 0.0179 mmHg/mL/min

after: = (95 mmHg - 10 mmHg)/(5625 mL/min) = 0.0151 mmHg/mL/min

c) Edema results due to the "backup" of blood on the venous side of the systemic circulation. This is partially due to the increased pressure in the  $P_c$  on the venous end of the capillaries. Using the values above and Starling's Law, explain why edema is occurring. (Starling's eqn, 3 pts; reasoning, 3 pts)

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net pressure = (P_c - P_i) - (\pi_c - \pi_i)
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before:  $\Delta P = (35-2)-(25-2) = 10$  mmHg (filtration); after: (45-2)-(25-4) = 22 mmHg (more filtration); If the mean capillary pressure was used, then  $\Delta P_{pre} = 0$  mmHg (no fluid movement) and  $\Delta P_{diag} = 12.5$  mmHg (increased filtration)

Reasoning: The system is now favoring filtration over a balance of filtration / absorption of fluid causing an increase in interstitial fluid, which caused Edema

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- 9. Consider an individual who has one collapsed lung. Rather than a normal 98% hemoglobin-oxygen saturation, her pulse oximetry estimated her O<sub>2</sub> saturation at 85%. (16 pts)
- a) How will this affect her ability to transport oxygen to her cells? (4 pts) Why? (4 pts)

There will be less  $O_2$  transported to her cells, since the  $O_2$ -hemoglobin saturation is lower than normal (hypoxemia), shifting the curve right and down. The fewer Hb sites bound, the less oxygen delivered.

b) If her hemoglobin concentration is 15 g Hb/100 mL of blood, and the partial pressure of oxygen in the aveoli is 100 mmHg, calculate the total oxygen content that she can deliver to her cells. Oxygen solubility in blood is 0.003 mL  $O_2/100$  mL blood/mmHg and 1 g of hemoglobin can bind 1.34 mL  $O_2$ .

 $O_2$  content =  $(O_2$ -binding capacity \* % saturation) + dissolved  $O_2$  (2 pts for correct equation)

 $O_2$ -binding capacity = 15 gHb/100 mL \* 1.34 mL  $O_2$ /g Hb = 20.1 mL  $O_2$ /100 mL blood

Dissolved  $O_2 = 100 \text{ mmHg} * 0.003 \text{ mL } O_2/100 \text{ mL blood/mmHg} = 0.3 \text{ mL } O_2/100 \text{ mL blood}$ 

 $O_2$  content =  $(20.1 \text{ mL } O_2/100 \text{ mL} * 0.85) + 0.3 \text{ mL } O_2/100 \text{ mL} = 17.4 \text{ mL } O_2/100 \text{ mL}$ 

- 10. What is the mechanism of action of tissue plasminogen activator? Why can't a thrombolytic drug be used for a hemorrhagic stroke?
- (3 pts) Tissue plasminogen activator (TPA) converts plasminogen to plasmin that can then break down the fibrin in blood clots (fibrinolysis).
- (3 pts) It can not be used for hemorrhagic stroke because it will cause for further bleeding in the patient