

**MASSACHUSETTS INSTITUTE OF TECHNOLOGY**

Departments of Electrical Engineering, Mechanical Engineering, and the Harvard-MIT Division  
of Health Sciences and Technology

6.022J/2.792J/BEH.371J/HST542J: Quantitative Physiology: Organ Transport Systems

**PROBLEM SET 4**

**SOLUTIONS**

March 9, 2004

## Problem 1

You are assigned to the Holter laboratory, and the Holter technician brings several rhythm strips to you, asking for advice. For each strip:

- Identify the arrhythmia as completely as possible.
- Draw the appropriate ladder diagram under each strip.

A. *Sinus arrhythmia. Normal finding. No symptoms expected.*

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B. *This is ventricular bigeminy, with three forms of VPBs seen. The VPBs are late-cycle, and do not interrupt the P-wave sequence. Since the cardiac rhythm is almost normal, it is quite probable that this arrhythmia would be asymptomatic. The patient might experience palpitations, however. It is considered clinically significant due to the possibility of developing more serious ventricular ectopic rhythm.*

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- C. *This is, on first inspection, sinus rhythm with APBs, one of which is conducted aberrantly with a classical RBBB pattern. Closer inspection of the P-wave morphology, however, suggests varying P-wave shapes and hence a wandering atrial pacemaker as the underlying rhythm rather than straight NSR. The patient might be aware of some palpitations due to the ectopic beats. (The beat following the pause will be more forceful due to increased filling time.)*

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- D. *This is supraventricular tachycardia at rates of about 230 bpm. The ladder diagram shows the mechanism to be re-entrant process in the AV junction. (P-waves are not easy to identify without ambiguity.) At such a rapid heart rate, the patient would experience symptoms due to inadequate C.O. (insufficient diastolic filling time). There would be lightheadedness, possibly syncope, possibly CHF, S.O.B., pulmonary edema, chest pain, etc.*

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E. *This is an agonal rhythm. There is complete heart block, with ventricular escape rhythm of only 15/min. This HR is probably inadequate to maintain BP and perfusion of vital organs. Patient would probably be unconscious, in shock, and would die unless rate could be increased by chronotropic drugs (atropine, isuprel) or pacing.*

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F. *This is low nodal rhythm. The HR is about 50 bpm, and should maintain adequate C.O. and BP, and would probably cause no symptoms.*

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G. *This is second degree heart block of Mobitz I (Wenkebach) type. The average heart rate is adequate, and the patient may experience only palpitations following the dropped beats. It is also possible that no symptoms would occur.*

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H. *This is atrial tachycardia (atrial rate = 200) with 4:1 heart block (ventricular rate = 50). The origin of the rhythm is not the SA node, but probably is a re-entrant mechanism within the AV junction. The heart rate is adequate for normal C.O. and BP, and the patient would probably be asymptomatic.*

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- I. *This is ventricular fibrillation. Ladder diagram is not really possible. There is no synchronous cardiac contraction, and no cardiac output. The patient would lose consciousness in 10-15 seconds, and die within minutes unless rhythm corrected.*

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- J. *The underlying rhythm is uncertain since P-waves are not clearly seen. It could be atrial fibrillation since the HR is slightly irregular, or a mid-nodal rhythm. There is one VPB, and then a run of ventricular tachycardia at a rate of 210 bpm. The ventricular tachycardia would probably cause a drop in C.O. and BP, with symptoms of palpitations, lightheadedness/dizziness, and possibly syncope. If it continues for a long time, deterioration to ventricular fibrillation is possible.*

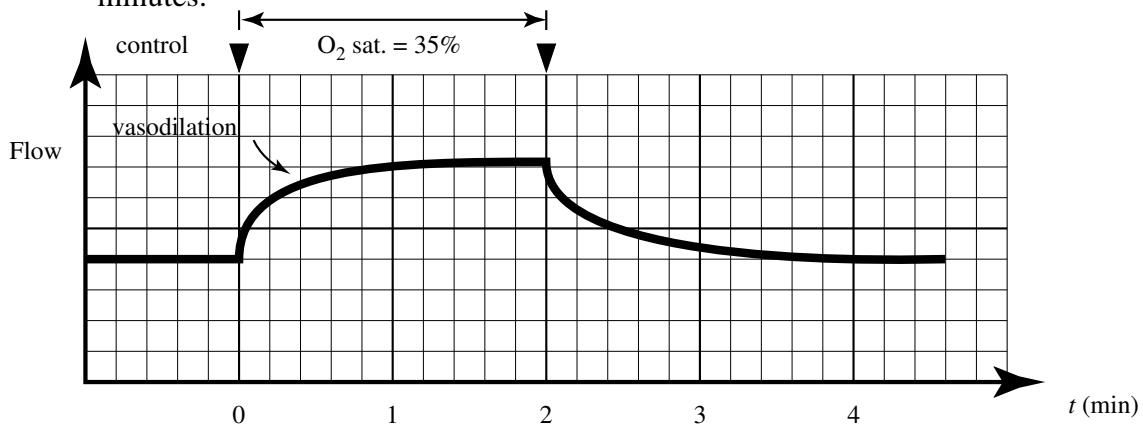
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## Problem 2

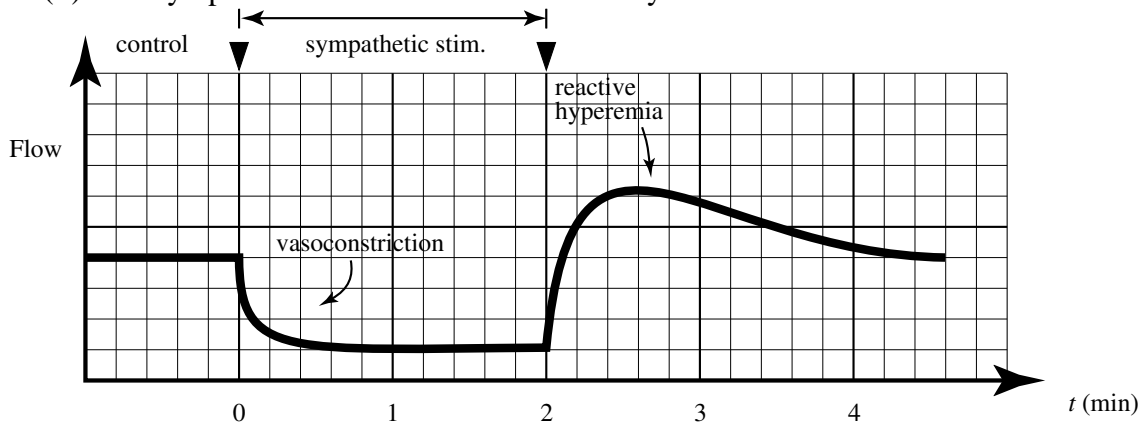
The following hypothetical experiments are carried out on an anesthetized rabbit with intact cardiovascular reflexes.

A. A hind limb is hemodynamically isolated and perfused with a constant pressure of 100 mmHg. The flow into the femoral artery is continuously measured. The venous outflow is returned to the perfusion reservoir. The nerves supplying the vasculature of the limb are cut. For each of the following hypothetical experimental procedures, please sketch the expected changes in blood flow into the limb (use the attached graphs), and *briefly explain the rationale for the changes you predict*.

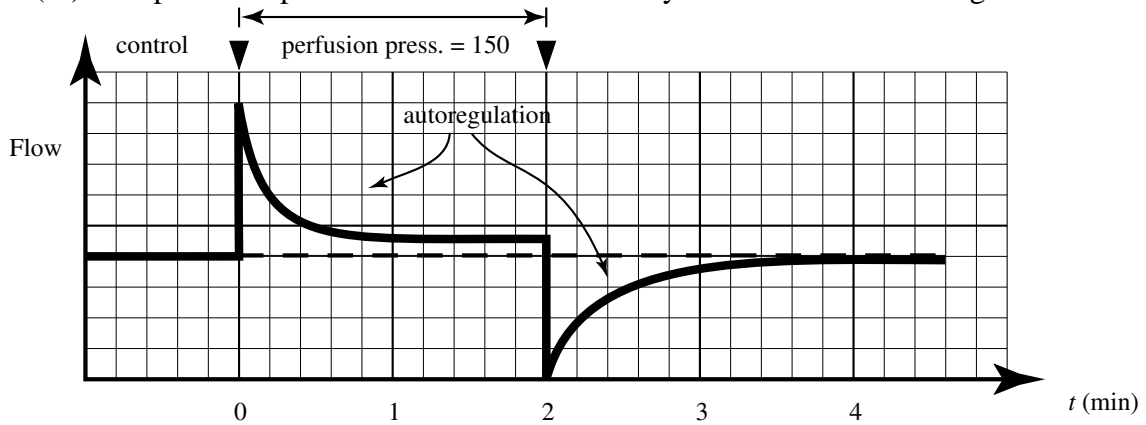
(i) The oxygen saturation of the perfusing blood is decreased from 100% to 35% for two minutes.



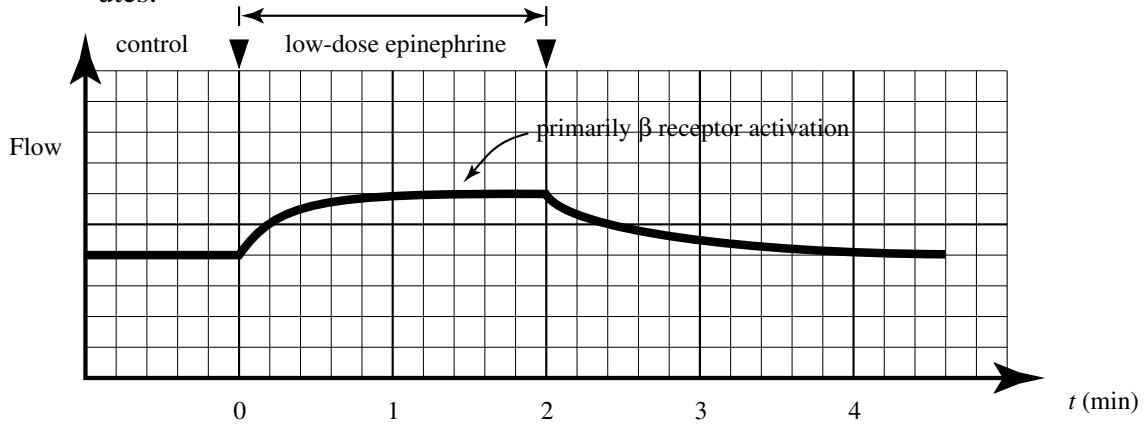
(ii) The sympathetic nerve fibers are electrically stimulated for two minutes.



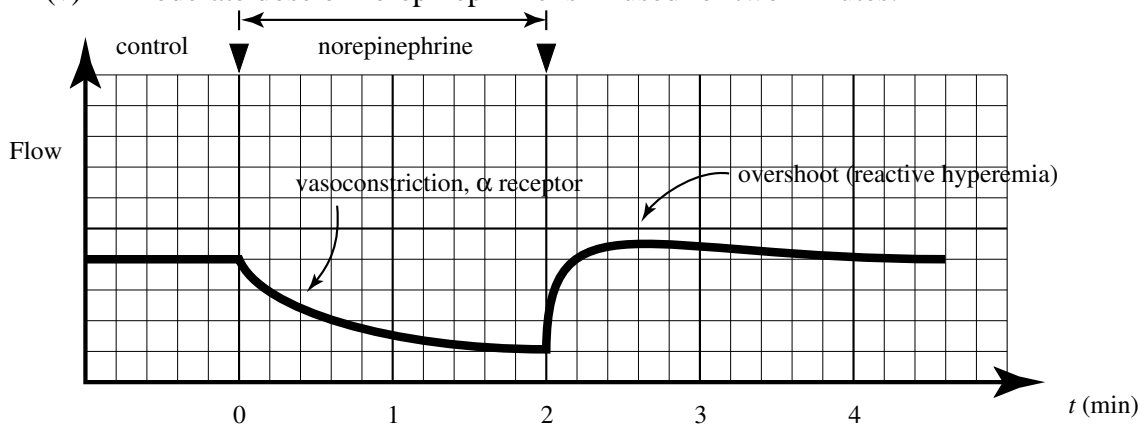
(iii) The perfusion pressure to the limb is suddenly increased to 150 mmHg for two minutes.



(iv) A low dose of epinephrine (0.3 micrograms/kg.) is infused into the limb for two minutes.



(v) A moderate dose of norepinephrine is infused for two minutes.

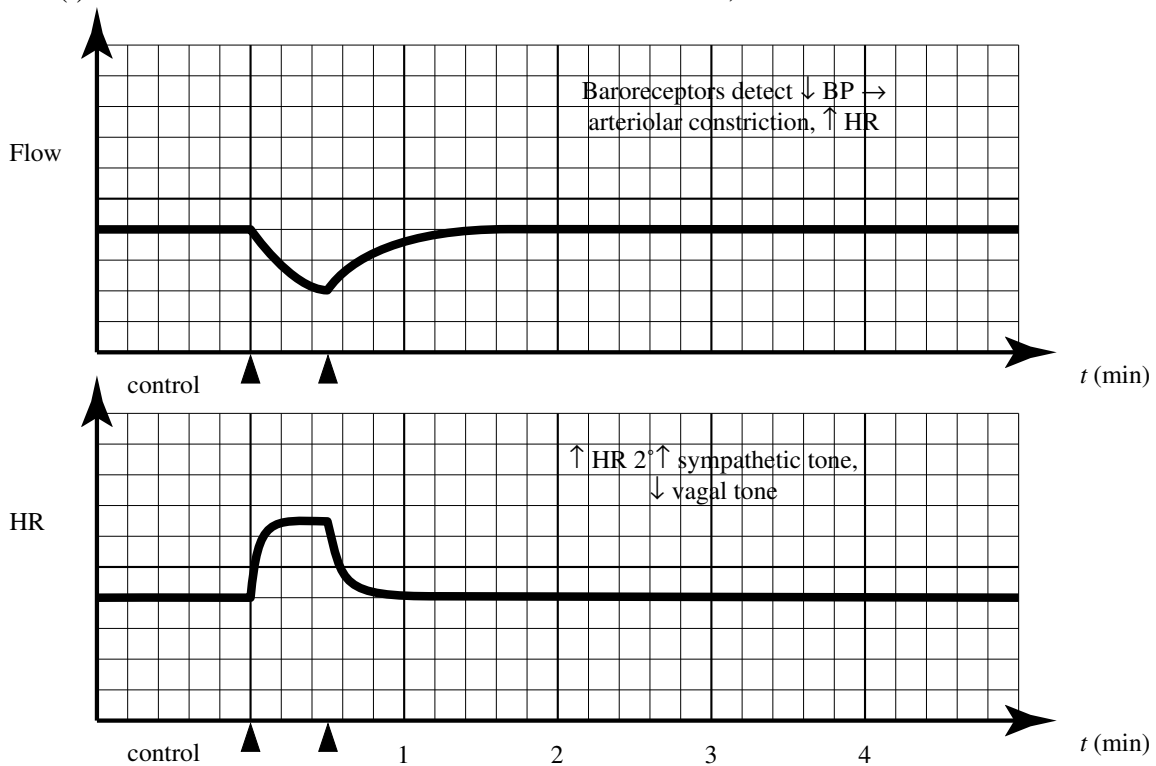




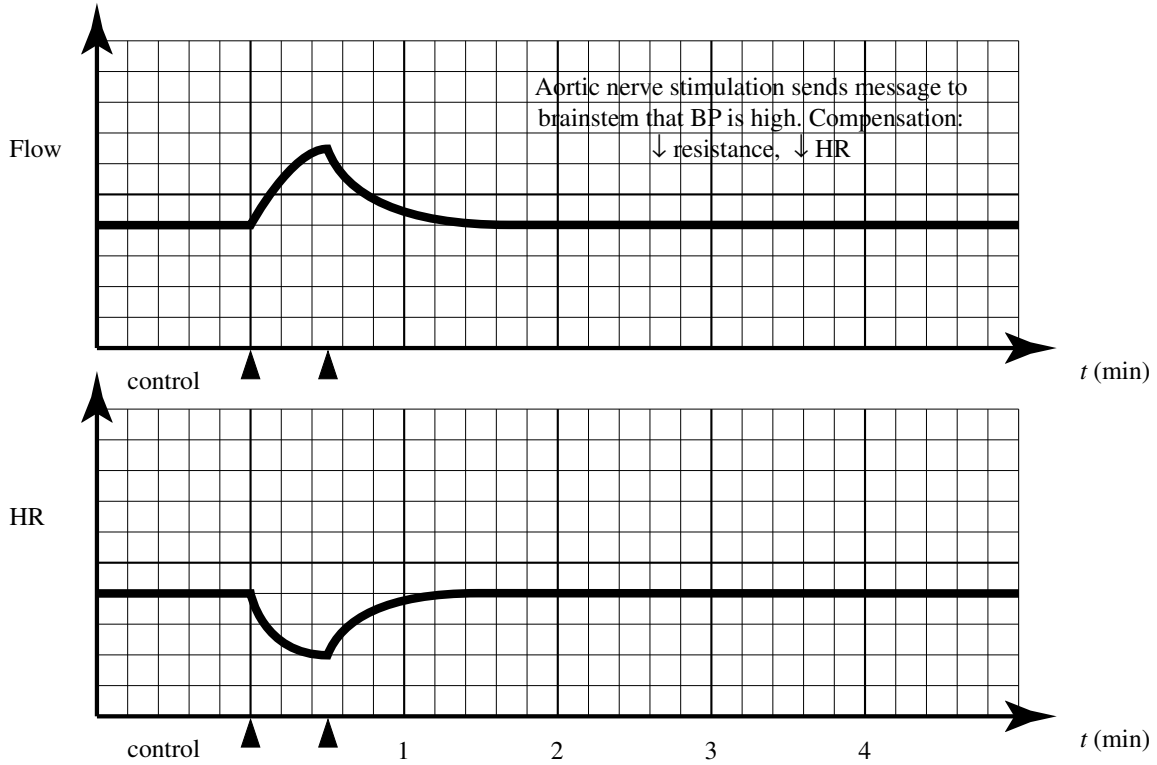
B. The same experimental preparation is used as in part A, except that the innervation to the hind limb is left intact. Thus, the leg is perfused from a separate blood supply at a constant pressure of 100 mmHg, and the blood flow into the limb is continuously monitored. In addition, the rabbit's ECG is monitored, and a continuous recording of heart rate is produced. The rabbit's neck is dissected, permitting access to the carotid arteries, the aortic nerve (afferent nerve from aortic baroreceptors), and the vagus nerve.

Sketch the responses you expect in the limb blood flow and the heart rate (using the attached graphs) for each of the following experimental procedures, and briefly explain your rationale:

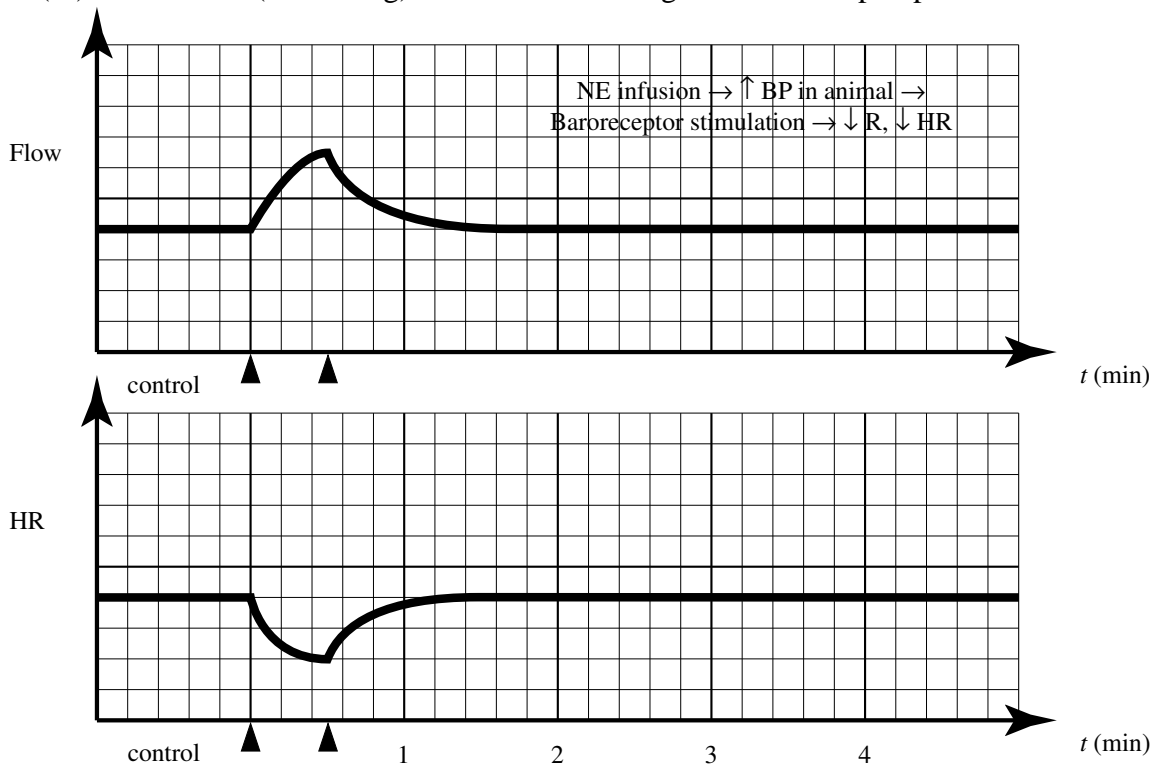
(i) Both carotid arteries are occluded for 30 seconds, then released.



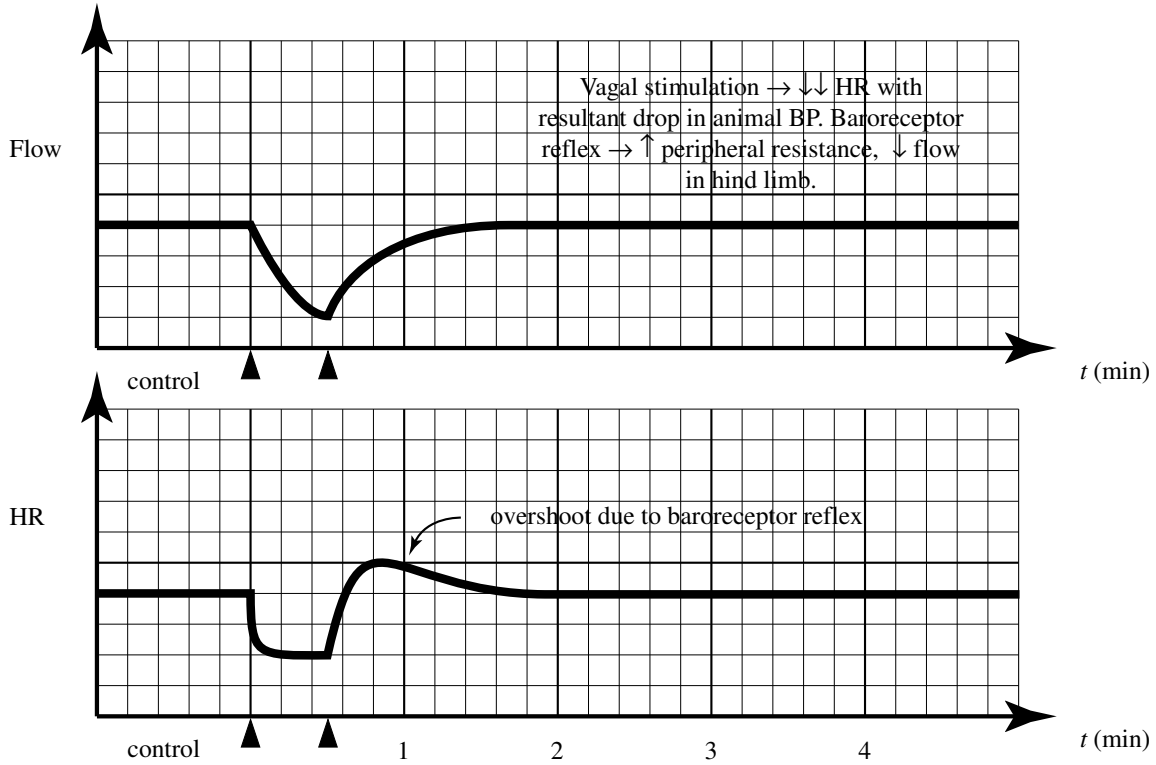
(ii) The aortic nerve is electrically stimulated for 30 seconds.



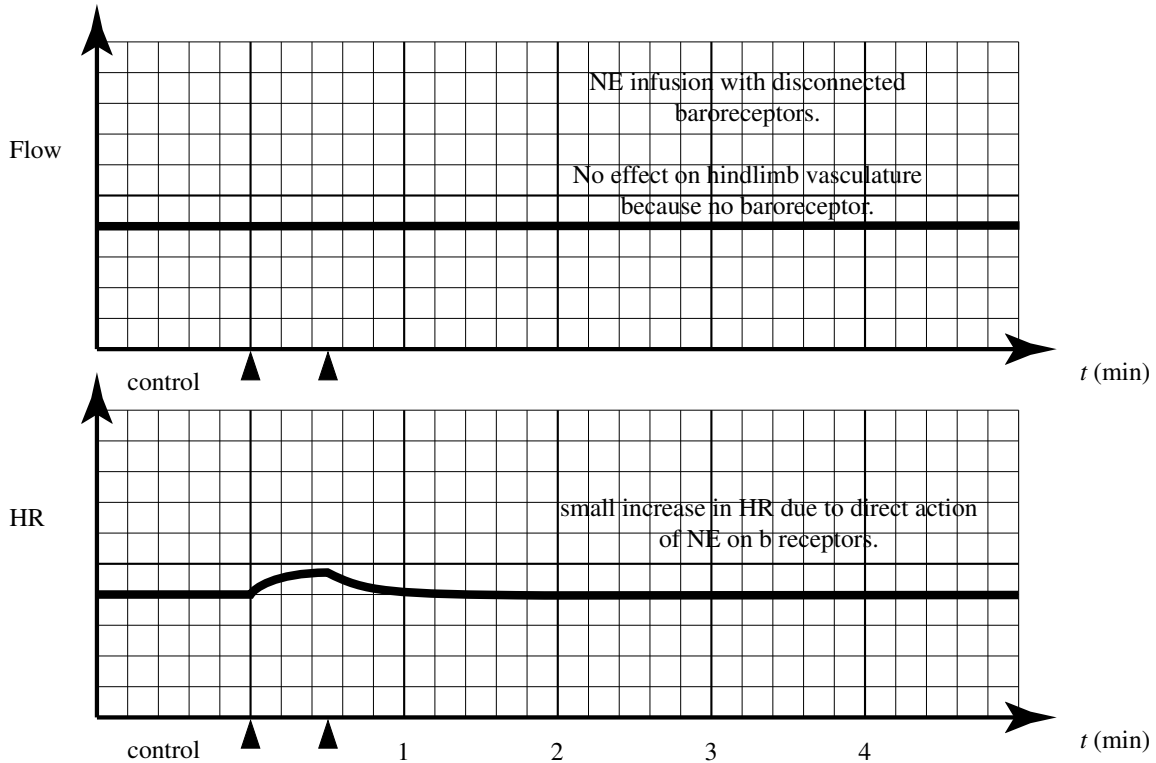
(iii) The animal (not the leg) is infused with a high dose of norepinephrine for 30 seconds.



(iv) The animal's vagus nerve is stimulated electrically for 30 seconds.



(v) Part iii above is repeated after cutting the aortic nerve and clamping both carotid arteries.



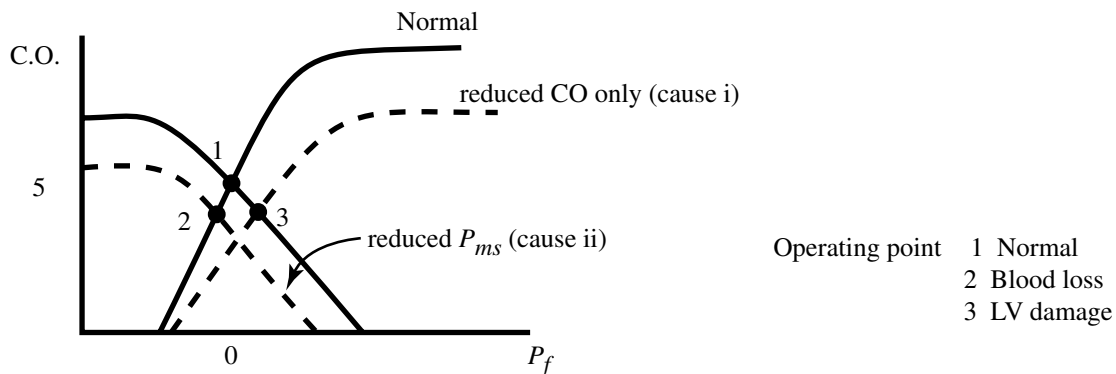
### Problem 3

A patient entered the hospital in shock (very low blood pressure) with chest pain. He was found to be pale, with cool, moist skin, dilated pupils, a pulse rate of 150 bpm (weak and thready), a blood pressure of 60/30, in a semicomatose state, with very little urine output. Two possible causes for the low blood pressure were considered:

- inadequate cardiac output because of damage to the left ventricle.
- severe internal blood loss—possibly due to a ruptured aortic aneurysm.

A. What single pressure measurement (preferably one that can be estimated by non-invasive inspection) would differentiate between the two diagnoses? Explain why, using CO/VR curves.

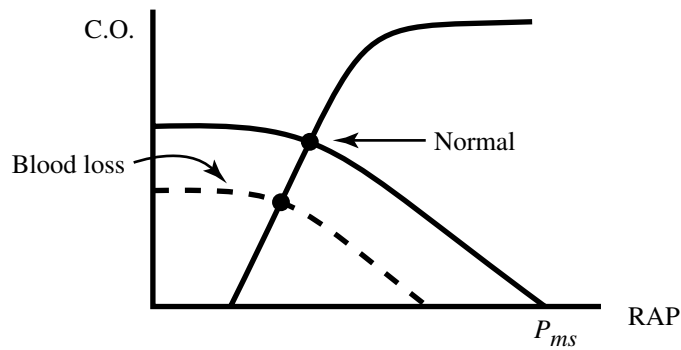
*Left atrial filling pressure is probably the most accurate choice, since it would differentiate clearly the condition of the LV. It is difficult to measure and is invasive, however. A second choice would be the right atrial pressure, and this measurement may be estimated by the non-invasive assessment of the jugular venous pressure. Blood loss → decreased filling pressure; heart failure → increased filling pressure.*



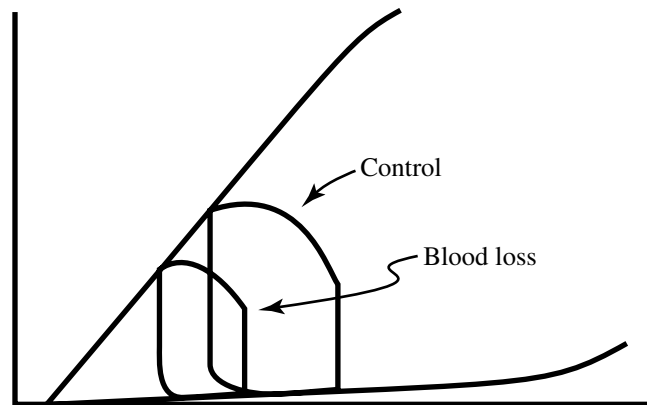
Further examination confirmed that the problem was one of *blood loss*, and that the heart was normal.

- B. Using cardiac output/venous return curves, show the situation for this hypothetical patient with no compensatory mechanisms. (Indicate a “normal,” and then the situation with the blood loss.)

*Patient is in shock, presumably hypovolemic shock due possibly to blood and fluid loss from internal bleeding. This results in movement of VR curve to L with drop in C.O. and therefore BP.*

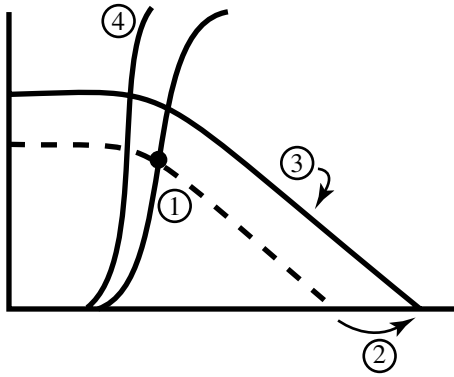


- C. Repeat part B but demonstrate the corresponding changes in the LV P-V domain.



D. Next, discuss as many compensatory mechanisms as you can (both short-term and long-term), and indicate how each will change the PV loops and VR/CO curves.

*The short-term compensation is via the baro-reflex. Effectors: (1)  $\uparrow$  HR, (2)  $\uparrow$  contractility, (3)  $\uparrow$  peripheral resistance, (4)  $\downarrow$  ZPFV.*



- (1) Hemorrhage — original operating point.
- (2) Increase  $P_{ms}$  by mechanism (4).
- (3) Slight decrease in slope via mechanism (3).
- (4) Increase C.O. slope via mechanisms (1), (2).

*The long-term mechanisms refer to renal mechanisms to retain salt and water (renin-angiotensin-aldosterone, anti-diuretic hormone, etc.).*

E. Explain each of the physical findings in terms of the appropriate physiology:

Low BP of 60/30

Paleness

Cool moist skin

Dilated pupils

HR — 150

Semicomatose state

Low urine output

*The clinical findings, therefore, are continued low BP (assuming incomplete compensation),  $\uparrow$  HR, pale, cool skin because of peripheral vasoconstriction, moist skin and dilated pupils because of  $\uparrow$  sympathetic stimulation. The low blood pressure causes low blood flow to the brain causing the semicomatose state. The low urine output is a result of renal compensatory mechanisms for hypovolemia.*

#### Problem 4

Two men are involved in an argument in a bar. One has had a good deal to drink, the other none.

Each sustains a gunshot wound to the leg and rapidly loses the same large amount of blood before the bystanders can stop the bleeding with direct pressure. Assume no other significant injury occurs.

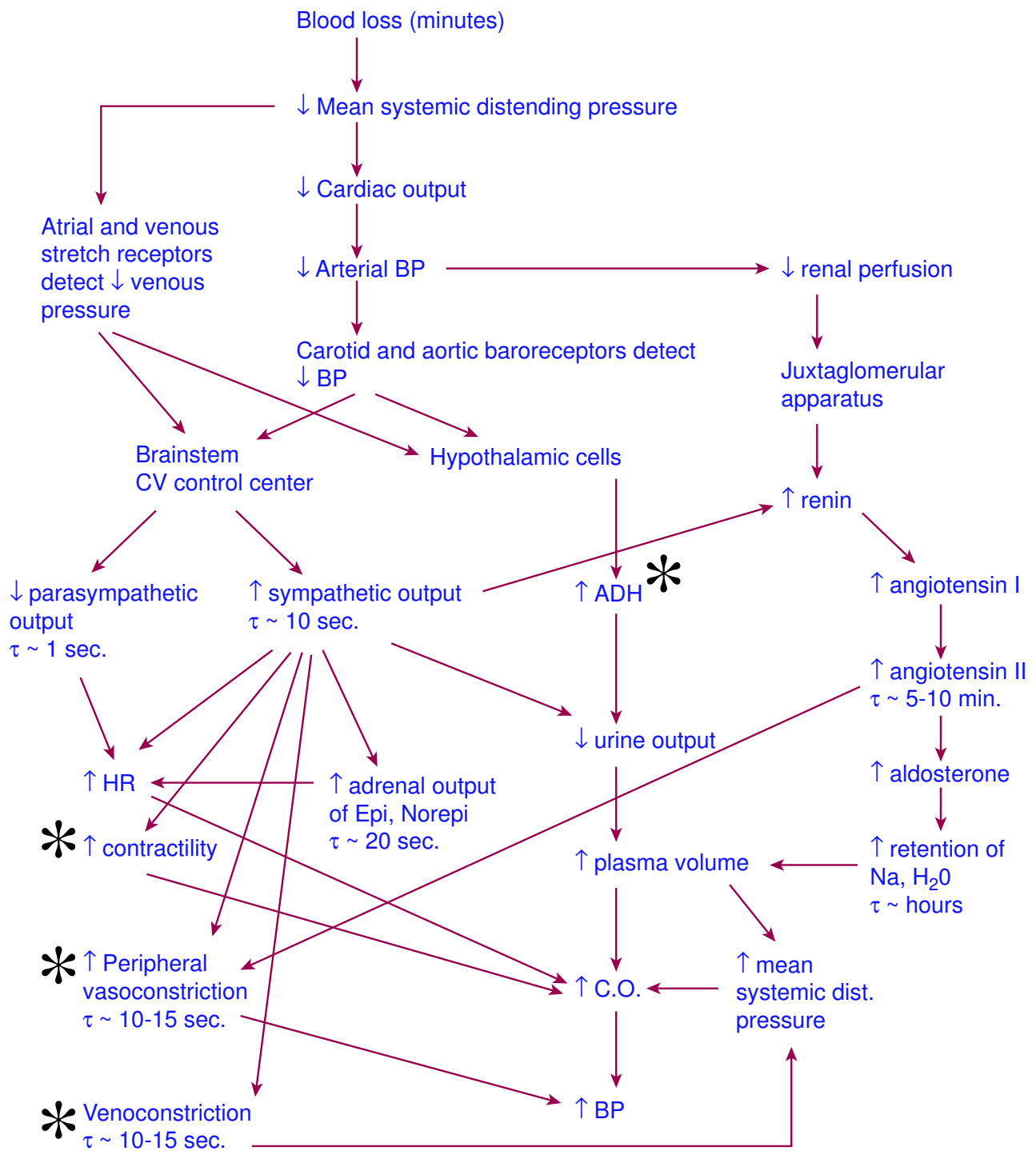
- A. List in an outline form what reflexes and adjustments would be expected to take place restore the circulation to normal without therapy. Please indicate the approximate time constant of each, and make use of function curves to illustrate the recovery. (Especially cardiac output/venous return, as well as ventricular pressure/volume curves).

*See flowchart and plots on next two pages.*

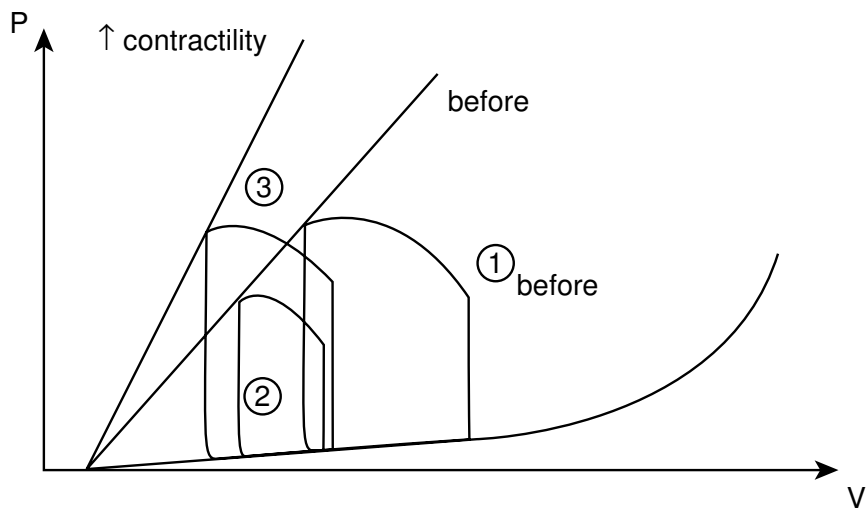
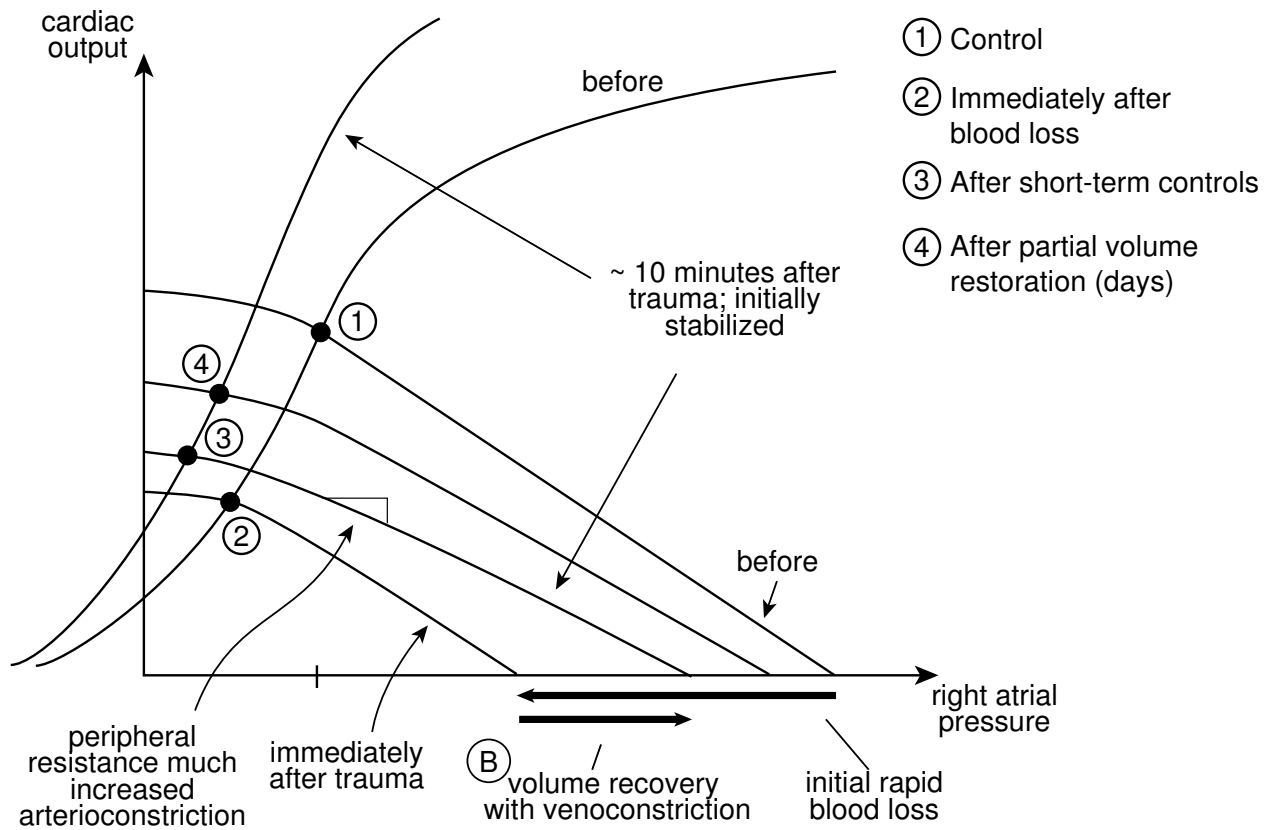
- B. Alcohol is a peripheral (both arterial and venous) dilator and indirectly a diuretic (increases urine output). Please show where this might interfere with or alter the recovery.

*The effect of alcohol is to inhibit the functions marked on the flowchart with an asterisk (\*).*

- *alcohol prevents ADH secretion, reducing renal compensation, inhibiting the consequent increase in blood pressure and volume.*
- *alcohol is a vasodilator, conflicting with the vasoconstriction compensation. This doesn't allow the peripheral resistance to increase as much to maintain blood pressure. Alcohol also prevents the venous zero pressure filling volume from decreasing to increase active blood volume and PMS.*
- *alcohol decreases cardiac contractility, thus decreasing to some extent the heart's compensation.*







- ②: immediately after trauma
- ③: ~10 minutes after trauma, initially stabilized.  
 HR ↑ and CO ↓, so SV ↓. BP ~ same as ①