

Space Biomedical Engineering, Spring 2006  
SOLUTIONS to Assignment #5 Cardiovascular Homework

1. Compare the heart rate and blood pressure at time = +100 sec to the baseline values at time = -10 sec. What is the physiologic basis for the observed change in arterial BP?

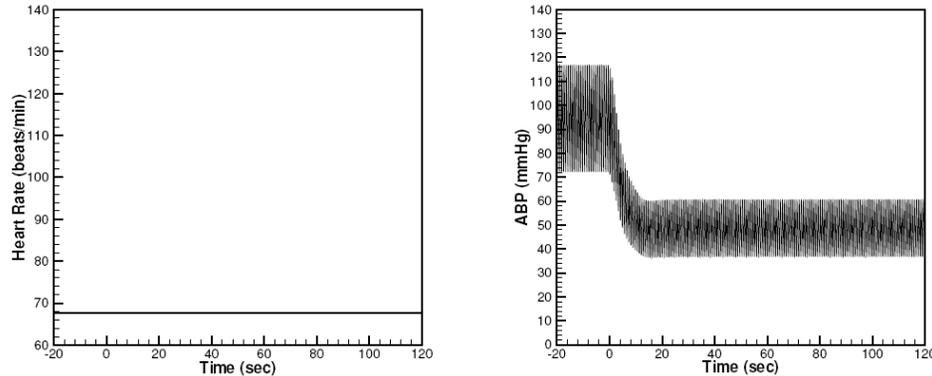


Figure 1: HR and BP in stand test—no CV controls

Description: Before and after the stand test, the heart rate remains fixed at 68 beats/min. The systemic arterial pressure (ABP) is 117/72 mmHg (average = 94.5 mmHg) while supine, but it decreases sharply upon standing and has dropped to 60/37 mmHg (average = 48.5 mmHg) by the time 100 seconds have passed.

Analysis: When a human moves from a supine to standing position, there is a major translocation of the body's blood; it shifts towards the feet and pools in the veins of the leg. This shift into the leg veins is effectively an increase in the venous zero pressure filling volume (ZPFV). Since veins are elastic, increasing the ZPFV causes an increase of blood volume in the veins before any stretch in the wall of the veins (and any pressure) appears. Assuming that the volume of blood in the body doesn't change, there is less blood in the other parts of the cardiovascular system, including in the systemic arteries. Therefore, the pressure drops in the systemic arteries. With the "operational control systems" switched off, the baroreflex cannot do its job of signaling the heart to increase its cardiac output. Therefore, the heart rate does not increase upon standing in this "no-control" case.

2. The curves in Figure 3 show the patient's status at time = -10 sec. Using the axes provided, sketch CO/VR curves and a LV P-V loop to represent the hemodynamics at time = +100 sec. (Hint: First estimate the new CO assuming constant peripheral resistance.)

The post-standing LV P-V loop is determined by the post-standing systolic pressure (60 mmHg), the post-standing diastolic pressure (38 mmHg), and the post-standing stroke

volume (SV is proportional to pulse pressure, which is systolic pressure minus diastolic pressure → since the post-standing pulse pressure is about half the pre-standing pulse pressure, the post-standing SV is about half of the pre-standing SV. SV is the distance between the two vertical lines in the cardiac cycle, so the pre-standing SV is about 75 cc. Thus, the post-standing SV is about 38 cc.) In this solution set's Figure 2, the points in the cardiac cycle are labeled 1, 2, 3, and 4. Cycle point #3 is determined by the intersection of the 60 mmHg systolic pressure and the already-drawn systole curve. Then, point #2 must be the point that is one stroke volume (38 cc) to the right of point #3 and at a pressure of 38 mmHg. Point #1 must lie on the diastole curve but at the same volume of point #2. Finally, point #4 lies just above the diastole curve but at the same volume of point #3.

The post-standing venous return curve is determined by estimating the post-standing cardiac output, CO. We know that cardiac output is equal to stroke volume times heart rate ( $CO = HR \cdot SV$ ), and that stroke volume is proportional to pulse pressure. In the previous paragraph, we discussed that the pulse pressure, and thus the stroke volume, decrease by a factor of two upon standing. Consequently, the cardiac output also decreases by a factor of two (in other words, it is cut in half). To draw the new venous return curve, we place the equilibrium point on 0 mmHg right atrial pressure but at half the cardiac output. Then, we draw the rest of the venous return curve with the same slope as the pre-standing venous return curve.

To intersect the venous return curve but also maintain an equilibrium point at 0 mmHg, the post-standing cardiac output curve must move to the right and have a decrease in slope.

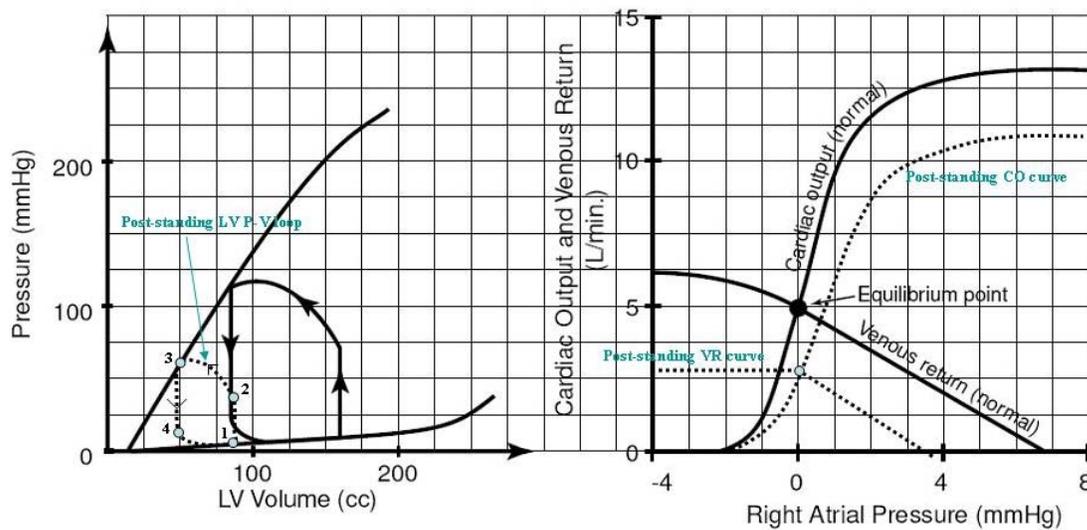


Figure 2: Cardiac output-venous return curves; LV pressure volume loops  
WITHOUT control, for  $t = -10$  sec (solid) and  $t = +100$  sec (dashed)

**3. What sensors and effectors are responsible for the behavior of heart rate and BP in Figure 4? What is the primary effector mechanism responsible for the initial increase in HR?**

The sensors responsible for the behavior of heart rate and blood pressure during the stand test are the atrial stretch receptors, which sense for blood volume, and the baroreceptors, which sense for pressure. The relevant effectors are heart rate, contractility, venous tone, and peripheral resistance. The primary effector mechanism that initiates the increase in HR is the sympathetic stimulus, brought on by the baroreceptor reflex.

**4. Why is the mean BP at 100 seconds slightly higher than the control level? (You may use the approximation  $P_m = P_d + 1/3 (P_s - P_d)$ .)**

Mean BP is equal to cardiac output times total peripheral resistance ( $P_m = CO * TPR$ ). With sympathetic stimulation working (i.e., control system “on”), the peripheral resistance increases substantially while the cardiac output decreases just slightly. The net effect is an increase in the product of CO and TPR, or an increase in  $P_m$ . Another way to look at this is that increased peripheral resistance causes an increase in diastolic pressure without much of a change to systolic pressure. Thus, the mean pressure increases upon standing.

**5. Using the axes of Figure 3 sketch the new situation at  $t = 100$  sec. Demonstrate the expected impact of the control mechanisms by appropriately modifying the curves. Hint: Estimate the new CO by assuming that stroke volume is proportional to pulse pressure (systolic–diastolic ABP). Fill in the work sheet below as a guide to drawing the curves.**

Parameter	$t = -10$ sec	$t = 100$ sec
<b>HR</b>	(from Assign. Fig. 4) 68 bpm	78 bpm
<b>CO</b>	(from Assign. Fig. 3) 5000 cc/min	$60 \text{ cc} * 78 \text{ bpm} = 4680 \text{ cc/min}$
<b>SV</b>	$SV = CO/HR = 5000 \text{ cc}/68 = 74 \text{ cc}$	$36 * 74/44 = 60 \text{ cc}$
<b>Pulse Press.</b>	$P_s - P_d = 116 - 72 = 44 \text{ mmHg}$	$P_s - P_d = 118 - 82 = 36 \text{ mmHg}$
<b>Mean ABP</b>	(from Assign. Fig. 4) 95 mmHg	(from Assign. Fig. 4) 102 mmHg

(Follow procedure of Problem 2 to create the LV P-V curve.)

[CORRECTION:] It is important to note that the data given to us in Problem 5 indicates that the stroke volume decreases, and this precludes the possibility of increased contractility, which is a control mechanism that would typically happen upon sympathetic stimulation. (Increased contractility causes stroke volume to increase rather than to decrease.) According to Problem 5’s assumption that stroke volume is proportional to pulse pressure, stroke volume decreases upon standing. Consequently, the venous return curve must also *decrease*, as shown in Figure 3.

However, the data for Problem 5 does indicate the normal sympathetic response of an increase in heart rate, so the cardiac output curve does move up and to the left. Figure 3 shows the post-standing curves that correspond to the data in Problem 5.

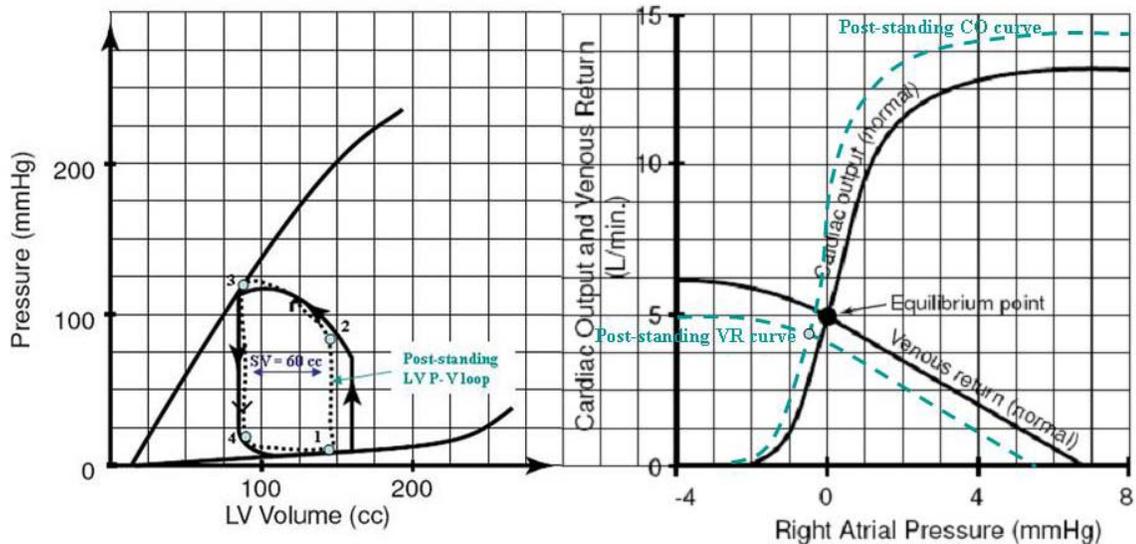


Figure 3: Cardiac output-venous return curves; LV pressure volume loops

WITH increased heart rate but NO increased contractility,  
for  $t = -10$  sec (solid) and  $t = +100$  sec

In a normally-functioning CV system, sympathetic stimulation normally causes increased contractility, constriction of veins, decreased venous capacitance, and increased mean system pressure – hence, if the increased contractility had occurred along with the increased heart rate, the venous return curve would have moved up and to the right [as in the original figure in the uncorrected solution set], and the stroke volume would have increased rather than decreased.

**6. What shifts of blood volume would you expect in the early hours of weightlessness? Explain your rationale.**

In the absence of gravity, first the blood that is normally attracted towards the feet will shift and be redistributed in the upper parts of the body. This is part of the total body fluid shift that occurs when weightlessness begins. Next, the volume sensors that are used to measure pressure at the atria will detect the increase in upper-body blood volume and thus measure the increase in upper-body pressure that has occurred. To reduce the pressure in the upper body, the cardiovascular control system takes action to reduce the volume of blood in the body by eliminating plasma through urine. Consequently, after a few hours of weightlessness, there will be a significant reduction in total blood volume.

**7. Why is the post-flight stand test result different from the pre-flight result?**

As described above, the microgravity environment eventually causes a loss of total blood volume (typically about 500 mL are lost). Furthermore, the heart's cardiac output decreases because the removal of the hydrostatic pressure gradient requires much less work from the heart. When the astronaut returns to gravity and conducts a stand test, the heart must compensate for the decreased blood volume and decreased cardiac output by producing a much larger increase in heart rate upon standing.

While total blood volume has the largest effect on the heart rate increase, we can mention at least three additional spaceflight-induced causes of the difference between the post-flight and pre-flight responses to standing. They include:

- 1) reduction in arterial resistance gain (decreased ability to increase peripheral resistance)
- 2) reduction in heart rate gain (because of reduced vagal component of baroreflex)
- 3) reduction in venous tone (decreased ability to constrict veins).

These three reductions are all a result of decreased peripheral sympathetic neurotransmission (in other words, decreased sympathetic gain). The sympathetic gain decreases because the lack of hydrostatic pressure gradient means that the baroreflex is not used in microgravity.

**8. Can you think of any countermeasures to reduce the problem of orthostatic hypotension? (Consider mechanical, pharmacological, or other methods.)**

- Fluid loading immediately before return to gravity
- Pharmaceuticals, that cause increase in total peripheral resistance or stroke volume
- Pharmaceuticals that increase heart rate
- Mechanical counterpressure around legs to augment venous return
- Aerobic exercise in space to maintain oxygen uptake rate