SymBioSys Exercise 1 – Arterial Hemodynamics
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Blood pressure and cardiac output are intimately linked. This exercise focuses on the relationship between cardiac output and blood pressure in the systemic and pulmonary circulations. Like any fluid flow, blood flow must obey the laws of physics, dissipating energy to overcome the effects of viscous resistance in the vessels throughout the circulation. In analogy with Ohm's law, the vascular resistance is defined as $\Delta P/Q$, where Q is the cardiac output, and where ΔP is the difference between the upstream pressure and the downstream pressure. The vascular resistance is set by the caliber of the flow-controlling vessels in the microcirculation.

Note that a lot of explanatory material on basic physiology is available in the SimBioSys help system.

Exercises

If you haven't done so yet, minimize the clinical tools to the left, and load exercise *E01.EXR*. This will set up the desktop, and make some changes to the simulations to let you study the arterial circulation without any unnecessary complications. For example, in this lesson the control systems (autonomic feedback) for the circulatory system have been disabled. This means that if blood pressure falls, you won't see any reflex increase in heart rate: we have done this to let you study the heart and arterial circulations by themselves. Later exercises will explore the integrated system. In addition, the time course of drug effects has been sped up by a factor of 60. This way, when you start or stop a drug, you won't need to wait 20 minutes to see what will happen. Once loaded, please go through the three exercises listed below.

1. Systemic Vascular Resistance

In the systemic circulation, the lion's share of the vascular resistance occurs in medium-sized arterioles, generally less than 100mm in diameter. For the systemic circulation, the upstream pressure is the mean arterial pressure, while the downstream pressure is found in the great veins. Since no valves separate the right atrium from the great veins, right atrial pressure equals the venous pressure, and we generally use the right atrial pressure to describe the downstream pressure for systemic blood flow. Arterial pressure is determined by cardiac output, systemic vascular resistance, and right atrial pressure. In Practice, right atrial pressure is generally very small compared to arterial pressure, so it can be neglected in the equation. Hence, arterial pressure is very nearly equal to $Q \cdot SVR$.

a. It is often said that mean arterial pressure is estimated as the diastolic pressure plus one third of the pulse pressure ($P_{systolic} - P_{diastolic}$). Record these variables and test this hypothesis.

b. Record mean arterial pressure, right atrial pressure, cardiac output, heart rate, and *SVR*. Calculate *SVR* using the recorded pressure and flow data and compare it with the displayed value.

c. Start a phenylephrine infusion at 4 mcg/kg/min. (Phenylephrine is an α-1 agonist). The subject may take a minute or two to stabilize; when stable, record new values of mean arterial pressure, right atrial pressure, cardiac output, heart rate, and *SVR*. What effect did the phenylephrine have on systemic vascular resistance? What effect did it have on cardiac output and arterial pressure?

d. Stop the phenylephrine and after stabilization, infuse nitroprusside at a rate of 0.8 mcg/kg/min. Again allow the subject to stabilize. Record new values of mean arterial pressure, right atrial pressure heart rate, and cardiac output. What effect did the nitroprusside have on systemic vascular resistance? How did it affect cardiac output and arterial pressure?

2. Pulmonary Vascular Resistance

The pulmonary vascular bed has the same total flow as the systemic circulation. The pulmonary vascular resistance is given by a formula analogous to the systemic vascular resistance. The upstream pressure in the pulmonary circuit is the mean pulmonary artery pressure and the downstream pressure is the mean left atrial pressure. Between the artery and left atrium, resistance is distributed more evenly in the pulmonary circulation than in the systemic circulation. However, the same principles hold true, and much of the pulmonary vascular resistance is located in arteriolar-like vessels upstream from the pulmonary alveolar capillaries.

Although the basic principles are similar between the arterial and pulmonary circulations, the magnitudes of the pressures are quite different. As a result, the downstream pressure (left atrial) is a much larger fraction of the upstream pressure (the pulmonary artery pressure) than on the systemic side. Accordingly, an increase in left atrial pressure resulting from left heart failure is a leading cause of pulmonary hypertension. The pulmonary circulation ordinarily has a very low resistance, so that the right heart has relatively little work to do. Significant pulmonary hypertension (as occurs with emphysema from smoking or chronic heart failure) can provide a major problem for the relatively flimsy right ventricle.

If the Pulmonary viewer is minimized , restore it now.

a. Record mean pulmonary arterial pressure, mean left atrial pressure, cardiac output, and *PVR*. Calculate *PVR* using the recorded pressure and flow data and compare it with the displayed value. Which elements in this calculation lead to the difference in magnitude between *PVR* and *SVR*?

b. Assuming the same cardiac output, what pressures would exist in the pulmonary circulation if its resistance were the same as the systemic circulation?

c. Can you identify any functional reasons why pressures and resistances should be so different in the systemic and pulmonary circulations?

3. Shape of the Pulse Waveform

As the ventricle ejects blood during systole, the vessel walls can expand and store some of the pressure energy. In diastole, this energy is recovered to produce blood flow out into the capillaries even when the ventricle is no longer ejecting. This ability of the arteries to distend, storing blood and energy, is one of several physical determinants of the compliance of the arterial system. Other physical determinants, including the transmission and reflection of pressure pulse waves in the arteries, also contribute but are too complex to describe in detail here. The net effect of arterial compliance is that the pressure fluctuations are reduced (a consequence of energy storage during systole) and a near-continuous capillary blood flow (a consequence of energy recovery in diastole).

a. Record systemic arterial systolic, diastolic, mean and pulse pressures, and stroke volume.

b. To decrease cardiac output, increase extracardiac pressure to 2 Torr and allow 60 sec for stabilization. Record new values of systolic and diastolic pressures, stroke volume and pulse pressure.

c. Now, increase extracardiac pressure to 4 Torr (mm Hg) and repeat stabilization and measurements.

d. Reset extracardiac pressure and wait a minute for stabilization.

How did the decrease in stroke volume affect systolic, diastolic, mean, and pulse pressures? Examine your three sets of data, by plotting pulse pressure against stroke volume. Can you think of a way to estimate changes in cardiac output from changes in vital signs?

As reviewed earlier, the product $Q \cdot SVR$ determines the mean value of systemic arterial pressure. As a first approximation, the pulse pressure (i.e., systolic minus diastolic arterial pressure) is determined by stroke volume (amount of blood ejected into the arterial circulation with each heartbeat) and arterial compliance. For a fixed compliance, pulse pressure increases with increments in stroke volume. For a fixed stroke volume, pulse pressure increases as compliance decreases. Therefore, changes in pulse pressure may be used as a practical index of stroke volume variations, provided that arterial compliance remains constant during the observation. In chronic pathological conditions (e.g., atherosclerosis, hypertension), arterial compliance is reduced significantly, so that a larger pulse pressure is developed for a given stroke volume. However, since these

pathologic changes are chronic, acute changes in pulse pressure usually signal changes in stroke volume even in these patients.

e. Expand the waveform viewer, to see the femoral and arterial pressure waveforms in detail.

Note that the aortic waveform shows a deep dicrotic notch, while the femoral artery pressure has only an inflection point reflecting valve closure. Note that there is a time delay between the aortic and femoral artery pressure waveforms. Finally, notice that the systolic pressure is higher in the femoral artery, and that the diastolic pressure is lower. What do you think accounts for the time delay between the two pressure measurements? Why does the pressure peak increase as one moves peripherally? Can you figure out why the aortic pressure rises after the aortic valve closes (i.e. why is there a dicrotic notch)?

Appendix A – Terminology

Mean Arterial Pressure (mm Hg): P_{art}
Mean Pulmonary Artery Pressure (mm Hg): P_{pa}
Mean Right Atrial Pressure (mm Hg): P_{ra}
Mean Left Atrial Pressure (mm Hg): P_{la}
Systemic Vascular Resistance (mm Hg/(l/min) or (ml/min)): *SVR*
Pulmonary Vascular Resistance (mm Hg/(l/min) or (ml/min)): *PVR*
Total flow or cardiac output (l/min or ml/min): *Q*
Heart Rate (/min): *HR*
Stroke Volume (liters or ml): *SV*
Systolic and Diastolic Blood Pressures (respectively, mm Hg): $P_{systolic}$, $P_{diastolic}$

Appendix B – Required Formulae

Stroke Volume from Cardiac Output and Heart Rate: $SV = Q/HR$
Systemic Vascular Resistance: $SVR = (P_{art}-P_{ra})/Q$
Pulmonary Vascular Resistance: $PVR = (P_{pa}-P_{la})/Q$