

LECTURE 7

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CARDIOVASCULAR PATHOPHYSIOLOGY

Background Physiology

Read Chapter 22 in your textbook as a review of background cardiovascular anatomy. You may have to consult an introductory physiology text for a review of the factors affecting blood pressure and the method of its measurement. Review also the cardiac cycle and the direction of blood flow through the heart and the great vessels entering and leaving it. Refresh your knowledge of basic heart anatomy including chambers, valves, and great vessels connecting the heart to both pulmonary and systemic circulation.

For review of the importance of hydrostatic blood pressure, go back and reexamine Lecture 1 on Capillary Hemodynamics. Recall the importance of hydrostatic pressure in forming interstitial fluid by forcing plasma filtration across capillary walls into interstitial spaces. Now, this hydrostatic pressure needs to be more closely examined. In fact, hydrostatic pressure is termed **Mean Arterial Pressure (MAP)** or, clinically speaking, simply **blood pressure** that is commonly measured with a sphygmomanometer using the brachial artery in the upper arm. MAP and brachial artery pressures are technically not the same. Mean arterial pressure is difficult to determine so brachial artery pressure is used for convenience. But keep in mind that it measures only the pressure in that one artery and other pressures in the circulatory system become progressively less and less as arteries branch into smaller diameter vessels and continue back to the heart across capillaries and veins. However, since vessels of the circulatory system are interconnected, pressure changes in one are often good indicators of pressure changes in others. There are important exceptions that will be dealt with later on.

In any blood vessel of the circulatory system, hydrostatic pressure is directly related to volume. More volume leads to higher pressures and vice versa. Thus, understanding the factors that affect volume in a given vessel leads to an understanding of the pressures in that vessel. Specifically, volume in a given vessel depends on the amount of blood entering the vessel versus the amount running out into smaller branches. In this regard, a simple garden hose is a useful analogy. A spigot controls the amount entering the hose while a nozzle at the other end controls the amount leaving it. Varying either or both will change the volume in the hose and the pressure as well. Finally, pressure differences within the circulatory system affect both capillary filtration and flow of blood from one point to another. The greater the pressure difference between two

points in the cardiovascular system, the greater the flow of blood between these two points. Small diameter vessels have pressure limits and will burst (arteries) or become distended (varicosities in veins), so homeostatic hydrostatic pressures have limits. Normal **systolic** pressure (during ventricular ejection/systole) is about 120 mm Hg while normal **diastolic** pressure (during ventricular relaxation/diastole) is about 80 mm Hg.

The relationships among the factors that affect the volume of blood in a given vessel and, therefore, its pressure are summarized by the following equation:

$$\text{PRESSURE} = \text{CARDIAC OUTPUT (C.O.)} \times \text{PERIPHERAL RESISTANCE (P.R.)}$$

In the hose analogy, cardiac output would be the *spigot* adding blood to a vessel and peripheral resistance would be the *nozzle* controlling the amount escaping. Since the factors of the equation are multiplied, increases in either C.O. or P.R. will result in increases in blood volume and, therefore, blood pressure. Conversely, decreases will have the opposite effect.

REVIEW QUESTIONS:

1. How would pulmonary artery hypertension affect lung interstitial fluid formation? Pulmonary vein congestion?
2. How would systemic hypertension affect renal filtration and urine output? Systemic hypotension?

Factors Affecting Cardiac Output and Blood Pressure

Looking at cardiac output in more detail, it is determined by the following equation:

$$\text{CARDIAC OUTPUT (C.O.)} = \text{STROKE VOLUME (S.V.)} \times \text{RATE (in beats/minute)}$$

CARDIAC OUTPUT is simply the amount of blood pumped by the heart (or a ventricle) expressed in milliliters per minute. The effect of HEART RATE on cardiac output is easily understood. Since the heart ejects blood with each beat, the more beats per minute the more blood ejected in that time interval. If output increases or decreases, going back to the first equation shows that pressure will change accordingly. Recall that heart rate is controlled by the **vasomotor center** of the medulla in response to changes in systemic blood pressure. **Baroreceptors** in the aortic arch and carotid sinus detect increased blood pressure and act reflexively via the vasomotor center to slow heart rate via parasympathetic nerves to the S-A Node or “pacemaker”. Absence of baroreceptor activity has the opposite effect and increases heart rate and the resultant blood pressure.

STROKE VOLUME effects are somewhat more involved. Simply defined, stroke volume is the volume of blood ejected by the heart (or a ventricle) with each beat or stroke. Stroke volume is related to the clinical term “ejection fraction” since both

describe the same volume but express it differently. Ejection fraction is simply the percentage of “full volume” ejected with each beat ($S.V./\text{full volume} \times 100 = \text{Ejection Fraction}$). Since the ventricles never empty completely with each beat, stroke volume is described by the following equation:

$$\text{STROKE VOLUME (S.V.)} = \text{END DIASTOLIC VOL. (EDV)} - \text{END SYSTOLIC VOL. (ESV)}$$

Consequently, factors affecting the two end volumes in the equation above will impact stroke volume, which will affect cardiac output and blood pressure.

END DIASTOLIC VOLUME is simply the “full” volume of blood in the heart and the EDV is often referred to as “**preload**”. Since the heart fills from venous return to it, anything that affects venous return of blood will change EDV and preload. For example, hemorrhagic blood loss drops volume and reduces venous return. As a result the heart does not fill completely and EDV goes down. The drop in EDV reduces stroke volume producing a weak or “thready” pulse. The lower stroke volume drops cardiac output and blood pressure reducing baroreceptor stimulation and allowing heart rate to increase.

REVIEW QUESTIONS:

1. What pathophysiological state would show the signs of rapid, weak pulse?
2. How might the body compensate for the resultant low pressure without using the heart?
3. How might a systemic histamine release produce the above signs? What is this condition called?

Read in your textbook (pp. 464-467) about the types of myocardial infarctions.

END SYSTOLIC VOLUME, the volume remaining at the end of systole (contraction), is a bit more involved. ESV refers to the amount of blood remaining in the heart (ventricle) at the end of a beat. Notice that ESV indicates that the heart does not empty completely with each beat. There will always be a small amount left in the ventricle(s) at the end of each beat. Subtracting this “leftover” volume from the full volume (EDV) gives the amount ejected (SV). Anything that affects the ventricles’ capacity to empty upon contraction affects ESV. For example, weakened cardiac muscle would reduce the strength of contraction and increase the ESV. A **myocardial infarction** (MI) would have that effect. If the heart fails to empty completely, stroke volume falls as less blood enters blood vessels and cardiac output drops causing a drop in blood pressure. Chronic hypertension is another example. Systemic or pulmonary hypertension results in an increased pressure in the arteries into which either ventricle must empty. For example, as systemic pressures go up, the left ventricle must contract more strongly in order to eject a stroke volume. A point can be reached beyond which the ventricle fails to generate enough force to empty completely against the high arterial pressure (“**aferload**”). The blood that was not ejected remains in the ventricle and

increases the ESV. Notice that increases in ESV result in corresponding decreases in stroke volume. Often, the muscle (**myocardium**) of the ventricle will enlarge (hypertrophy) when faced with this level of hypertensive stress. Ventricular enlargements appear on an EKG as either right or left **axis deviations** depending upon the involved ventricle.

REVIEW QUESTIONS:

1. How might PAH (pulmonary artery hypertension) appear on an EKG?
2. How could left ventricular MI's lead to pulmonary edema and renal failure?
3. Why might elastic stockings and/or leg massages be indicated for a client with long term bed confinement?

Factors Affecting Peripheral Resistance and Blood Pressure

By definition, peripheral resistance refers to the resistance to the flow of blood in the circulatory system. This resistance directly correlates with friction generated as blood contacts vessel walls and rubs against itself as it flows through vessels of the circulatory system. A number of factors affect this friction: (1) **vessel length**; (2) **vessel diameter**; (3) and the **viscosity** of blood itself.

Vessel length effects are apparent, yet the ways it may change may not be. The human circulatory system has about 25,000 miles of vessels constituting a large vessel wall surface against which blood must rub as it flows through the system. The branching nature of vessels produces large numbers of small diameter vessels, which exposes proportionately more blood to the wall surface. Arterioles, small diameter arteries leading to capillaries, are the most numerous of the vessel types and have the greatest wall surface and resistance effect. So, adding more arterioles increases peripheral resistance dropping the pressure in the arterioles but raising the pressure in larger, upstream trunk arteries that supply them as blood backs up (congests) in these larger arteries. Adding adipose tissue with weight gains requires growing more small arteries to supply the new tissue and can result in hypertension in obese people.

Changes in vessel diameter, **vasoconstriction** or **vasodilation**, are the normal ways the body diverts blood flow from one area to another. Constricting distributing arteries and arterioles reduces pressure and flow in the affected vessels but diverts blood to some other body part increasing pressure and flow in these areas. Consequently, a distinction must be made between **upstream** and **downstream pressures** relative to the constriction or dilation. For example, smokers are often hypertensive with poor peripheral circulation. Nicotine is a peripheral vasoconstrictor which reduces blood flow and pressure at the periphery of circulation (downstream) but congests blood in major trunk arteries causing the pressure to rise there (upstream). Since pressure is measured only in the upstream brachial artery, hypertension is easily observed in the clinical

environment. Although, clinical hypertension is apparent, the cold hands and feet with pale skin reflect poor peripheral blood flow (perfusion).

Since blood flows through vessels in layers (laminar flow), it must rub against itself as it moves. The more viscous blood becomes, the greater the friction and resistance as the layers rub against each other. A major determiner of blood viscosity is hematocrit. Increased red cell numbers result in more viscous blood, greater resistance, and potential hypertension. CAREFUL!! Don't confuse "blood thinners" that slow clotting time with factors that affect the flow properties of moving blood. The term can be misleading.

Read in your textbook (pp 455-464) about arteriosclerosis and atherosclerosis.

Be aware that "arteriosclerosis" is a general term referring to the normal hardening of arteries that occurs with aging. However, the process can be abnormally accelerated. "Atherosclerosis" is one of the ways of accelerating the hardening process. In atherosclerosis, lipid materials (particularly those rich in cholesterol) penetrate artery walls and cause an inflammatory response within the wall. Contrary to popular belief, atheromatous plaque develops within the artery wall and does not just adhere to its inner surface. As the process progresses, the artery wall thickens and weakens and may become calcified in extreme cases. The thickening of the wall narrows the vessel lumen and mimics vasoconstriction by reducing blood flow downstream and raising upstream pressures. Also, the affected wall surface is disrupted and roughened attracting platelets, which can rupture and initiate clot formation. A clot so formed is an "intravascular" **thrombus** and can break loose becoming an **embolus**. By forming in an artery, the embolus moves downstream until vessel diameter gets too small for it to pass and it lodges there. The resulting blocked blood flow (**infarction**) is a major cause of MI's and strokes if it occurs in coronary circulation or the brain.

REVIEW QUESTIONS:

1. Explain why "shock" victims show pale, cold skin.
2. How are the signs and symptoms of hemorrhagic and anaphylactic shock similar? Different?
3. Why is it physiologically unwise to eat a large lunch before class?
4. What is phlebitis and where would clots resulting from the condition lodge?
5. What are the dangers of and the treatments for hypertension?

DISCUSSION QUESTIONS: (Post answers to the “Patho Discussion Group”)

1. What are the possible consequences of atheromatous plaque in the anterior descending interventricular artery? The carotid arteries?
2. What changes in EDV and ESV might be expected with a right ventricular aneurysm?