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Physiology, Cardiovascular

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Introduction

The cardiovascular system provides blood supply throughout the body. Responding to various stimuli can control the velocity and amount of blood carried through the vessels. The cardiovascular system comprises the heart, arteries, veins, and capillaries. The heart and vessels work intricately to provide adequate blood flow to all body parts. The regulation of the cardiovascular system occurs via a myriad of stimuli, including changing blood volume, hormones, electrolytes, osmolarity, medications, adrenal glands, kidneys, and much more. The parasympathetic and sympathetic nervous systems also play a key role in regulating the cardiovascular system.[1][2][3]

Organ Systems Involved

The heart is the organ that pumps blood through the vessels. It pumps blood directly into arteries, specifically the aorta or the pulmonary artery. Blood vessels are critical because they control the amount of blood flow to specific parts of the body. Blood vessels include arteries, capillaries, and veins. Arteries carry blood away from the heart and can divide into large and small arteries. Large arteries receive the highest blood flow pressure and are thicker and more elastic to accommodate the high pressures. Smaller arteries, such as arterioles, have more smooth muscle, which contracts or relaxes to regulate blood flow to specific body portions. Arterioles face a smaller blood pressure, meaning they don't need to be as elastic. Arterioles account for most of the resistance in the pulmonary circulation because they are more rigid than larger arteries. Furthermore, the capillaries branch off of arterioles and are a single-cell layer. This thin layer exchanges nutrients, gases, and waste with tissues and organs. Also, the veins transport blood back to the heart. They contain valves to prevent the backflow of blood.

Function

- The cardiovascular system consists of 2 main loops: systemic circulation and pulmonary circulation. Its purpose is to provide adequate blood circulation through the body. Pulmonary circulation allows for the oxygenation of the blood, and systemic circulation allows oxygenated blood and nutrients to reach the rest of the body.

Mechanism

To understand the physiology of the heart, it is important to understand the cardiac output, stroke volume, preload, Frank-Starling law, afterload, and ejection fraction.

Cardiac Output

The cardiac output (CO) is the amount of blood ejected from the left ventricle; normally, it equals the venous return. The calculation is CO = stroke volume (SV) x heart rate (HR). CO also equals the rate of oxygen consumption divided by the difference in arterial and venous oxygen content.

SV

The SV is the amount of blood pumped out of the heart after 1 contraction. It is the difference between end-diastolic (EDV) and end-systolic (ESV) volume. It increases with increased contractility, increased preload, and decreased afterload. Also, the left ventricle's contractility increases with catecholamines by increasing intracellular calcium ions and lowering extracellular sodium.

Preload

The preload is the pressure on the ventricular muscle by the ventricular EDV.

Frank-Starling Law

Frank-Starling law describes the relationship between EDV and SV. This law states that the heart attempts to equalize CO with venous return. As venous return increases, a larger EDV in the left ventricle leads to further stretching of the ventricle, leading to a larger contraction force and a larger SV. A larger SV leads to a larger CO, thus equalizing CO with venous return.

Afterload

Afterload is the pressure the left ventricular must exceed to push blood forward. Mean arterial pressure best estimates this. Also, afterload can be estimated by the minimum pressure needed to open the aortic valve, equivalent to the diastolic pressure. Thus, diastolic blood pressure is one of the better ways to index afterload.

Ejection Fraction

The ejection fraction (EF) equals SV/EDV. EF of the left ventricle is an index for contractility. A normal EF is greater than 55%. A low EF indicates heart failure.[4][5][6][7]

Cardiac Cycle

The cardiac cycle describes the path of the blood through the heart. It runs in the following order:

- Atrial contraction closure of the mitral valve
- Isovolumetric phase
- Opening of the aortic valve
- Ejection phase (rapid and reduced ejection), emptying of the left ventricle
- Closure of the aortic valve
- Isovolumetric relaxation
- The opening of the mitral valve
- The filling phase (rapid and reduced filling) of the left ventricle

Vasculature plays a significant role in regulating blood flow throughout the body. In general, blood pressure decreases from arteries to veins, and this is because of the pressure overcoming the resistance of the vessels. The greater the change in resistance at any point in the vasculature, the greater the pressure loss. Arterioles have the most increase in resistance and cause the largest decrease in blood pressure. The constriction of arterioles increases resistance, which causes a decrease in blood flow to downstream capillaries and a larger decrease in blood pressure. Dilation of arterioles causes a decrease in resistance, increasing blood flow to downstream capillaries and a smaller decrease in blood pressure.

Diastolic Blood Pressure

Diastolic blood pressure (DP) is the lowest pressure in an artery at the beginning of the cardiac cycle while the ventricles are relaxing and filling. DP is directly proportional to total peripheral resistance (TPR). Also, the energy stored in the compliant aorta during systole is now released by the recoil of the aortic wall during diastole, thus increasing diastolic pressure.

Systolic Blood Pressure

Systolic blood pressure (SP) is the peak pressure in an artery at the end of the cardiac cycle while the ventricles contract. It is directly related to stroke volume; as stroke volume increases, SP also increases. SP is also affected by aortic compliance. Because the aorta is elastic, it stretches and stores the energy caused by ventricular contraction, decreasing the SP.

Pulse Pressure

Pulse pressure is the difference between SP and DP. Pulse pressure is proportional to SV and inversely proportional to arterial compliance. Thus, the stiffer the artery, the larger the pulse pressure.

Mean Arterial Pressure

Mean arterial pressure (MAP) is the average pressure in the arteries throughout the cardiac cycle. The MAP is always closer to DP. MAP is calculated by MAP= DP + 1/3 (pulse pressure). Also, MAP = CO x TPR. This value is significant because whenever CO decreases, the TPR increases to maintain the MAP, which is relevant in many pathophysiology problems.

Velocity

Systemic veins have a lower decrease in pressure because they have low resistance. The venous system is very compliant and contains up to 70% of the circulating blood. A small change in venous pressure can mobilize the blood stored in the venous system. The velocity of blood in the vasculature has an inverse relationship with the cross-sectional area: volumetric flow rate (Q) = flow velocity (v) x cross-sectional area (A).

As the cross-sectional area increases, velocity decreases. Arteries and veins have smaller cross-sectional areas and the highest velocities, whereas capillaries have the most cross-sectional and lowest velocities. The vasculature also gives resistance; resistance is R= (8*viscosity*length)/(πr^4). Viscosity depends on hematocrit and increases in multiple myeloma or polycythemia. As tube length increases, the resistance increases. As the tube radius increases, the resistance decreases. The fact that the radius is to the power of 4 means that slight changes in the radius profoundly affect resistance. The total resistance of vessels in a series is R1 + R2 + R3, and so on, and the total resistance of arteries in parallel is 1/TR = 1/R1+1/R2+1/R and so on, where TR is the total resistance.

Poiseuille Equation

The Poiseuille equation measures the flow of blood through a vessel. It is measured by the change in pressure divided by resistance: Flow = (P1 - P2)/R, where P is pressure, and R is resistance. Increasing resistance in a vessel, such as the constriction of an arteriole, causes a decrease in blood flow across the arteriole. At the same time, there is a larger decrease in pressure across this point because the pressure is lost by overcoming the resistance. Increasing the resistance at any point increases upstream pressure but decreases downstream pressure. The Poiseuille equation applies to the systemic circulation such that F is the cardiac output (CO), P1 is the mean arterial pressure (MAP), P2 is the right atrial pressure (RAP), and R is the total peripheral resistance (TPR). Because RAP is close to 0 and very small compared to MAP, the equation approximates F=P1/R or CO=MAP/TPR, where MAP=CO*TPR means cardiac output and total peripheral resistance control MAP. Its application is important because in trauma situations with hemorrhage, there is also a decrease in cardiac output, but at times, the blood pressure is near normal. This is because the TPR at the level of the arterioles has increased. As applied to the pulmonary vasculature, this equation determines the cause of pulmonary hypertension. As related to the pulmonary vasculature, F represents CO, P1 represents pulmonary artery pressure (PAP), P2 represents left atrial pressure (LAP), and R is pulmonary vascular resistance

(PR); CO=(PAP-LAP)/PR. A Swan-Ganz catheter helps to measure both PAP and LAP, allowing for the measurement of PR and, thus, the etiology of pulmonary hypertension.

Baroreceptors and Chemoreceptors

The nervous system regulates the cardiovascular system with the help of baroreceptors and chemoreceptors. Both receptors are located in the carotid and aortic arch. Also, both have afferent signals through the vagus nerve from the aortic arch and afferent signals through the glossopharyngeal nerve from the carotids.

Baroreceptors are more specifically located in the carotid sinus and aortic arch. They respond quickly to changes in blood pressure. A decrease in blood pressure or blood volume causes hypotension, which leads to a decrease in arterial pressure. This decrease in arterial pressure decreases the baroreceptors' stretch and decreases afferent baroreceptor signaling. This decrease in afferent signaling from the baroreceptor causes an increase in efferent sympathetic activity and a reduction in parasympathetic activity, which leads to vasoconstriction, increased heart rate, increased contractility, and an increase in BP. The vasoconstriction increases TPR in the equation MAP=CO*TPR to increase pressure (MAP). An increase in blood pressure or blood volume causes hypertension, increasing the baroreceptors' stretch.

Chemoreceptors come in 2 types: peripheral and central. Peripheral chemoreceptors are specifically located in the carotid body and aortic arch. They respond to oxygen levels, carbon dioxide levels, and the pH of the blood. They become stimulated when oxygen decreases, carbon dioxide increases and the pH decreases. Central chemoreceptors are located in the medulla oblongata and measure the cerebral spinal fluid's pH and carbon dioxide changes.

Autoregulation

Autoregulation is how an organ or tissue maintains blood flow despite a change in perfusion pressure. When blood flow decreases to an organ, arterioles dilate to reduce resistance.

- **Myogenic theory:** Myogenic regulation is intrinsic to the vascular smooth muscle. When there is an increase in perfusion, the vascular smooth muscle is stretched. This causes it to constrict the artery. If there is a decrease in perfusion to the arteriole, then there is decreased stretching of the smooth muscle. This leads to the smooth muscles' relaxation and arteriole dilation.
- **Metabolic theory:** Blood flow is closely related to metabolic activity. When there is an increase in metabolism to muscle or any tissue, there is an increase in blood flow to that location. Metabolic activity creates substances that are vasoactive and stimulate vasodilation. The increase or decrease in metabolism leads to increased or decreased metabolic byproducts that cause vasodilation. Increased adenosine, carbon dioxide, potassium, hydrogen ions, lactic acid levels, decreased oxygen levels, and increased oxygen demand all lead to vasodilation. Adenosine is from AMP, which derives from the hydrolysis of ATP and increases during hypoxia or increased oxygen consumption. Potassium is increased extracellularly during metabolic activity (muscle contraction) and directly affects the relaxing of smooth muscles. Carbon dioxide is produced as a byproduct of the oxidative pathway and increases with metabolic activity. Carbon dioxide diffuses to the smooth vascular muscle and triggers an intracellular relaxing pathway.
- **Heart:** Metabolites that cause coronary vasodilation include adenosine, NO, carbon dioxide, and low oxygen.
- **Brain:** The primary metabolite controlling cerebral blood flow is carbon dioxide. An increase in arterial carbon dioxide causes vasodilation of cerebral vasculature. A decrease in arterial carbon dioxide causes vasoconstriction of the cerebral vasculature. Hydrogen ions do not cross the blood-brain barrier and thus are not a factor in regulating cerebral blood flow. A decrease in oxygen pressure in arteries causes vasodilation of the cerebral arteries; however, an increase in oxygen pressure in arteries does not cause vasoconstriction.
- **Kidneys:** Autoregulation of the kidneys is myogenic and with tubuloglomerular feedback. In severe cases of

hypotension, kidney arterioles constrict, and renal function is lost.

- **Lungs:** Hypoxia of the lungs causes vasoconstriction, creating a shunt away from poorly ventilated areas of the lung and redirecting perfusion to ventilated portions of the lung.
- **Skeletal muscle:** Adenosine, potassium, hydrogen ion, lactate, and carbon dioxide all increase during exercise and cause vasodilation. When resting, the skeletal muscle is controlled extrinsically by sympathetic activity, not metabolites.
- **Skin:** Skin regulation occurs through sympathetic stimulation. The purpose of regulating blood flow in the skin is to regulate body temperature. In a warm environment, skin vasculature dilates due to decreased sympathetic stimulation. In cold environments, skin vasculature constricts due to increased sympathetic activity. During fever, body temperature regulation is at a higher setpoint.

The starling equation can explain the capillary fluid exchange. This equation describes oncotic and hydrostatic pressure forces on fluid movement across the capillary membrane. Edema can result from increased capillary pressure (heart failure), decreased plasma proteins (liver failure), increased interstitial fluid due to lymphatic blockage, or increased capillary permeability due to infections or burns.

Related Testing

Swan-Ganz catheter is a thin tube inserted peripherally and passed to the right side of the heart and into the pulmonary artery. This catheterization measures the pressures in the pulmonary vasculature and the left atrium. Pulmonary capillary wedge pressure (PCWP) estimates the pressure in the left atrium given by the Swanz-Ganz catheter. PCWP is significant because it helps to differentiate pathologies. In cardiac shock, PCWP increases, whereas in hemorrhagic shock, it decreases.

Pathophysiology

Chronic hypertension is a common pathological process related to the cardiovascular system. This condition is significant because, with hypertension, there is an increase in afterload. A long-term increase in afterload leads to concentric hypertrophy of the heart and eventual left-sided diastolic heart failure. Also, an S4 heart sound is audible at the apex of the heart. Another type of heart disease is alcoholic cardiomyopathy, which occurs in alcoholics and causes dilated cardiomyopathy, which means the ventricles become dilated, leading to systolic failure. It can be reversible if the patient stops drinking alcohol.

Heart failure or cardiac tamponade can cause cardiogenic shock. In cardiogenic shock, there is an increase in PCWP because there is a backup of blood; the heart cannot pump blood forward because it cannot overcome the afterload. Subsequently, there is a decrease in CO. In response to low CO, systemic vascular resistance (SVR) increases.

In hemorrhagic shock, blood is lost, thus reducing total volume. Because of this, pressure decreases, resulting in a decrease in PCWP. Also, cardiac output increases because more blood is needed in the periphery. While CO increases, SVR increases to maintain MAP.

Clinical Significance

Blood pressure is an essential clinical value because it describes the status of the vasculature in acute and chronic states. If a patient has elevated blood pressure in the clinic on more than 2 occasions, the clinician can diagnose the patient with essential hypertension. Blood pressure can also be significant in acute settings, such as in the emergency room after a patient is brought in by an ambulance due to a motor vehicle accident. At this point, it is important to assess the patient's BP because if it is low, it might indicate the patient is bleeding somewhere, and the clinician must determine the location of the bleeding as soon as possible.[8]

S1 and S2 heart sounds are normal heart sounds heard on auscultation of the heart. S1 is the sound made due to the

closure of the mitral and tricuspid valves. This is followed by systole. Then, the S2 sounds are heard, which are the closure of the aortic and pulmonary valves. Diastole follows this. Recognizing these normal heart sounds on auscultation is important because abnormal heart sounds such as S3, S4, and murmurs can be signs of a pathological condition.

Review Questions

- Access free multiple choice questions on this topic.
- Comment on this article.

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