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Effects of Vasodilation and Arterial Resistance on Cardiac Output

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Abstract

Heart is one of the most important organs present in human body which pumps blood throughout the body using blood vessels. With each heartbeat, blood is sent throughout the body, carrying oxygen and nutrients to all the cells in body. The cardiac cycle is the sequence of events that occurs when the heart beats. Blood pressure is maximum during systole, when the heart is pushing and minimum during diastole, when the heart is relaxed. Vasodilation caused by relaxation of smooth muscle cells in arteries causes an increase in blood flow. When blood vessels dilate, the blood flow is increased due to a decrease in vascular resistance. Therefore, dilation of arteries and arterioles leads to an immediate decrease in arterial blood pressure and heart rate. Cardiac output is the amount of blood ejected by the left ventricle in one minute. Cardiac output (CO) is the volume of blood being pumped by the heart, by left ventricle in the time interval of one minute. The effects of vasodilation, how the blood quantity increases and decreases along with the blood flow and the arterial blood flow and resistance on cardiac output is discussed in this review Article.

Keywords: Heart; Cardiac cycle; Arteries; Blood flow; Vasodilation; Arterial Resistance; Cardiac output

Introduction

The circulatory system is composed of the heart and blood vessels, including arteries, veins, and capillaries [1,2]. Arteries and Veins play an important role in blood circulation along with heart [3]. The heart is the key organ in the circulatory system [4]. As a hollow, muscular pump, its main function is to propel blood throughout the body. It usually beats from 60 to 100 times per minute, but can go much faster when necessary. It beats about 100,000 times a day, more than 30 million times per year, and about 2.5 billion times in a 70-year lifetime. With each heartbeat, blood is sent throughout our bodies, carrying oxygen and nutrients to every cell. Each day, 2,000 gallons of blood travel many times through about 60,000 miles of blood vessels that branch and cross, linking the cells of our organs and body parts. Heart collects the deoxygenated blood from the body and pushes it to the lungs where it becomes oxygenated, and then heart pumps the oxygen rich blood to the body. Normal functioning of heart is very important to lead a healthy life [5]. Vasodilation is widening of blood vessels caused by relaxation of smooth muscle cells in the vessel walls particularly in the large arteries, smaller arterioles and large veins thus causing an increase in blood flow [6]. Arterial dilation leads to an immediate decrease in arterial blood pressure and heart rate [7]. The relationship between mean arterial pressure, cardiac output and total peripheral resistance (TPR) gets affected by Vasodilation. Cardiac output is the amount of blood that is pumped by the heart per unit time, measured in liters per minute (l/min). The amount of blood that is put out by the left ventricle of the heart in one contraction is called the stroke volume. The stroke volume multiplied by the heart rate is the "cardiac output". Numerous cardiovascular afflictions are currently known to be associated with heart including aortic root dilation, aortic regurgitation, mitral regurgitation, myocarditis, heart failure, pericarditis, pericardial effusion [8,9,10,]. Sudden deaths due to cardiac arrest, cardiac stroke, atrioventricular conduction block, and heart failure are reported worldwide [11,12,13]. Various animals like mouse were used to detect the heart disease [14]. Cardiovascular disease is one of the most frequent causes of death of women in the world [15,16,17]. Stroke is the major healthcare problem with higher mortality and morbidity rates [18]. Women are more affected with Atherosclerosis [19]. At times increase in blood pressure may leads to various kinds of health problems [20,21]. Heart failure patients are at increased risk of sudden death due to ventricular problems [22,23,24]. Diabetes Mellitus (DM) is also a main risk factor for heart failure [25,26,27]. Most of the cardiovascular emergencies are caused by coronary artery disease [28,29]. Echocardiography is the modality of choice for investigation of suspected congenital or acquired heart disease [30,31,32,] Suspected heart disorders and related heart diseases can be investigated using Echocardiogram [33,34,35].

Cardiac cycle

The sequence of events that occurs when the heart beats, is known as "cardiac cycle". The frequency of the cardiac cycle is described by the heart rate [36]. There are two phases of the cardiac cycle. The heart ventricles are relaxed and the heart fills with blood in diastole phase [37]. The ventricles contract and pump blood to the arteries in systole phase [38]. When the heart fills with blood and the blood is pumped out of the heart one cardiac cycle gets complete. The events of the cardiac cycle explains how the blood enters the heart, is pumped to the lungs, again travels back to the heart and is pumped out to the rest of the body [39]. The important thing to be observed is that the events that occur in the first and second diastole and systole phases actually happen at the same time [40].

Cardiac Cycle: 1st Diastolic Phase: During this first diastole phase, the atrioventricular valves are open and the atria and ventricles are relaxed. From the superior and inferior vena cavae the de-oxygenated blood flows in to the right atrium. The atrioventricular valves which are opened allow the blood to pass through to the ventricles [41]. The Sino Atrial (SA) node contracts and also triggers the atria to contract. The contents of the right atrium get emptied into the right ventricle. The back flow of blood into the right atrium is prevented by "Tricuspid valve".

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Cardiac Cycle: 1st Systolic Phase: During this first systole phase, the right ventricle contracts as it receives impulses from the Purkinje fibers [42]. The semi lunar valves get opened and the atrioventricular valves get closed. The de-oxygenated blood is pumped into the pulmonary artery. The back flow of blood in to the right ventricle is prevented by pulmonary valve [43]. The blood is carried by pulmonary artery to the lungs. There the blood picks up the oxygen and is returned to the left atrium of the heart by the pulmonary veins [44].

Cardiac Cycle: 2nd Diastolic Phase: In the next diastolic phase, the atrioventricular valves get opened and the semi lunar valves get closed. The left atrium gets filled by blood from the pulmonary veins, simultaneously (Blood from the vena cava is also filling the right atrium.) The Sino Atrial (SA) node contracts again triggering the atria to contract. The contents from the left atrium were into the left ventricle [45]. The back flow of oxygenated blood into the left atrium is prevented by "Mitral valve" [46].

Cardiac Cycle: 2nd Systolic Phase: During the following systolic phase, the semi lunar valves get open and atrioventricular valves get closed. The left ventricle contracts, as it receives impulses from the Purkinje fibers [47]. Oxygenated blood is pumped into the aorta. The prevention of oxygenated blood from flowing back into the left ventricle is done by the aortic valve. Aortic and mitral valves are important as they are highly important for the normal function of heart [48]. The aorta branches out and provides oxygenated blood to all parts of the body. The oxygen depleted blood is returned to the heart via the vena cavae. Left Ventricular pressure or volume overload hypertrophy (LVH) leads to LV remodeling the first step toward heart failure, causing impairment of both diastolic and systolic function [49,50]. Coronary heart disease [CHD] is a global health problem that affects all ethnic groups involving various risk factors [51,52].

Vasodilation

Vasodilation is increase in the internal diameter of blood vessels or widening of blood vessels that is caused by relaxation of smooth muscle cells within the walls of the vessels particularly in the large arteries, smaller arterioles and large veins thus causing an increase in blood flow [53]. When blood vessels dilate, the blood flow is increased due to a decrease in vascular resistance [54]. Therefore, dilation of arteries and arterioles leads to an immediate decrease in arterial blood pressure and heart rate (hence, chemical arterial dilators are used to treat heart failure, systemic and pulmonary hypertension, and angina) [55]. At times leads to respiratory problems [56]. The response may be intrinsic (due to local processes in the surrounding tissue) or extrinsic (due to hormones or the nervous system). The frequencies and heart rate were recorded while surgeries [57]. "Vasoconstriction" is the narrowing of blood vessels resulting from contraction of the muscular wall of the vessels, particularly the large arteries, small arterioles and veins [58]. The process is the opposite of vasodilation. "Vasodilators" are the factors that results in vasodilation. Some of the examples of Vasodilators include carbondioxide (CO_2) and Nitric oxide (NO) [59,60]. The primary function of Vasodilation is to increase the flow of blood in the body, especially to the tissues where it is required or needed most. This is in response to a need of oxygen, but can occur when the tissue is not receiving enough glucose or lipids or other nutrients [61]. In order to increase the flow of blood localized tissues utilize multiple ways including release of vasodilators, primarily adenosine, into the local interstitial fluid which diffuses to capillary beds provoking local Vasodilation [62].

Vasodilation and Arterial Resistance

The relationship between mean arterial pressure, cardiac output and total peripheral resistance (TPR) gets affected by Vasodilation. Vasodilation occurs in the time phase of cardiac systole while vasoconstriction follows in the opposite time phase of cardiac diastole [63]. Cardiac output (blood flow measured in volume per unit time) is computed by multiplying the heart rate (in beats per minute) and the stroke volume (the volume of blood ejected during ventricular systole) [64]. TPR depends on certain factors, like the length of the vessel, the viscosity of blood (determined by hematocrit) and the diameter of the blood vessel. Vasodilation works to decrease TPR and blood pressure through relaxation of smooth muscle cells in the tunica media layer of large arteries and smaller arterioles [65,66]. A rise in the mean arterial pressure is seen when either of these physiological components (cardiac output or TPR) gets increased [67]. Vasodilation occurs in superficial blood vessels of warm-blooded animals when their ambient environment is hot; this diverts the flow of heated blood to the skin of the animal [68], where heat can be more easily released into the atmosphere [69]. Vasoconstriction is opposite physiological process. These processes are naturally modulated by local paracrine agents from endothelial cells like nitricoxide [70], bradykinin, potassium ions and adenosine etc, as well as an organism's autonomic nervous system and adrenal glands, both of which secrete catecholamine's such as epinephrine and norepinephrine respectively [71].

Vascular Resistance

The resistance to flow that must be overcome to push blood through the circulatory system is known as "Vascular resistance". Systemic vascular resistance (SVR) is the resistance offered by the peripheral circulation [72], while the resistance offered by the vasculature of the lungs is known as the pulmonary vascular resistance (PVR) [73]. The systemic vascular resistance may also be referred as the "Total Peripheral Resistance" (TPR). Vasodilation (increase in diameter) decreases SVR, where as Vasoconstriction (i.e., decrease in blood vessel diameter) increases SVR [74]. The Units for measuring vascular resistance are dyn.s.cm^{-5}, pascal seconds per cubic metre (Pa.s/m^3) or, deriving it by pressure (measured in mmHg) and cardiac output (measured in l/min), it can be given in mmHg.min/l. This is numerically equivalent to hybrid reference units (HRU), also known as Wood units, frequently used by pediatric cardiologists. To convert from Wood units to MPa.s/m^3 you must multiply by 8, or to dyn.s.cm^{-5} you must multiply by 80.

Calculation of Resistance can be done by using these following formulae:

Calculating resistance is that flow is equal to driving pressure divided by resistance. The systemic vascular resistance can therefore be calculated in units of dyn.s.cm^{-5} as

$$\frac{(\text{mean arterial pressure} - \text{mean right atrial pressure})}{\text{Cardiac output}}$$

Where Mean Arterial pressure is 2/3 of diastolic blood pressure plus 1/3 of systolic blood pressure.

The basic tenet of calculating resistance is that flow is equal to driving pressure divided by resistance.

The systemic vascular resistance can therefore be calculated in units of dyn.s.cm^{-5} as

$$\frac{(\text{mean arterial pressure} - \text{mean right atrial pressure})}{\text{Cardiac output}}$$

The pulmonary vascular resistance can therefore be calculated in units of dyn.s.cm^{-5} as

$$\frac{\text{mean pulmonary arterial pressure} - \text{mean pulmonary artery wedge pressure}}{\text{Cardiac output}}$$

Where the pressures are measured in units of millimetres of mercury (mmHg) and the cardiac output is measured in units of litres per minute (L/min).

Cardiac Output

Cardiac output (CO) is the quantity of blood or volume of blood that is pumped by the heart per minute. Cardiac output is a function of heart rate and stroke volume [75]. It is the product of stroke volume (SV; the volume of blood ejected from the heart in a single beat) and heart rate (HR; expressed as beats per minute or BPM) [76]. Ivabradine (IVB) is a novel, specific, heart rate (HR), lowering agent which is very useful [77,78]. Increasing either heart rate or stroke volume increases cardiac output. Most of the strokes are caused by atrial fibrillation [79].

Cardiac Output in mL/min = heart rate (beats/min) X stroke volume (mL/beat)

An average person has a resting heart rate of 70 beats / minute and a resting stroke volume of 70 mL/beat. The cardiac output for this person at rest is:

Cardiac Output = 70 (beats/min) X 70 (mL/beat) = 4900 mL/minute

The total volume of blood in the circulatory system of an average person is about 5 liters (5000 mL).With strenuous activity, an adult's cardiac output can increase up to 7 fold (35 liters/minute) to satisfy the body's demand for oxygen and nutrients. Treatment for multiple congenital cardiac defects usually refers to open-heart surgery or a combination of medical treatment and open heart surgery [80,81,82]. The timing and outcomes of cardiovascular diseases are linked with surrounding power fields also [83].

Control of Heart Rate: With the activity of both sympathetic and parasympathetic nerve fibers, Sino Atrial node of the heart gets enervated [84]. The parasympathetic fibers release acetylcholine, under rest conditions which slows the pacemaker potential of the Sino Atrial node, thus reducing the heart rate [85]. The sympathetic nerve fibers release norepinephrine, under physical or emotional conditions which speeds up the pacemaker potential of the Sino Atrial node, increasing the heart rate [86]. Epinephrine is released from adrenal medulla by the activity of Sympathetic nervous system [87]. Epinephrine enters the blood stream, and is delivered to the heart where it binds with Sino Atrial node receptors. Binding of epinephrine leads to further increase in heart rate.

Control of Stroke Volume: The heart does not fill to its maximum capacity, under rest conditions. If the heart were to fill more per beat then it could pump out more blood per beat, thus increasing stroke volume. During systole, the heart ventricles empty only about 50% of their volume. The heart could pump out more blood per beat if the heart were to contract more strongly [88]; in other words, a stronger contraction would lead to a larger stroke volume. During the exercise time or exercise periods, the stroke volume increases because of these mechanisms; the heart contracts more strongly and the heart fills up with more blood [89].

The Stroke volume is increased by 2 mechanisms:

- Increase in end-diastolic volume and
- Increase in sympathetic system activity

End-diastolic volume: volume of blood in the ventricles at the end of diastole, called "end-diastolic volume". A larger end-diastolic volume will stretch the heart [90]. Stretching of the heart muscles optimizes the length and strength relationship of the cardiac muscle fibers, which results in stronger contractility and greater stroke volume [91].

Increase in sympathetic system activity increases the Stroke Volume: Release of norepinephrine by sympathetic nerve fibers causes an increase in the strength of myocardial contraction, thus increasing the stroke volume [92]. Epinephrine, like norepinephrine will stimulate an increase in the strength of myocardial contraction and thus increase stroke volume.

Conclusion

Heart is a major organ and plays a key role in circulatory system of body. The main function of heart is to pump the blood to all parts of the body through various blood vessels. The force exerted by the blood against the vessel wall is referred to as "Blood pressure". Every blood vessel in the circulatory system has its own blood pressure, which changes continually. Arterial blood pressure rises and falls in a pattern corresponding to the phases of the cycles of the heart, the cardiac cycle. Flow through a blood vessel is determined by two factors: the force that pushes the blood through the vessel, and the resistance of the vessel to the blood flow. Usually the rate of blood flow is measured in milliliters or liters per minute (ml/min or l/min). Vasodilation causes increase in blood flow due to decrease in vascular resistance. The resistance produced mainly in the arterioles is known as the systemic vascular resistance (SVR) or the peripheral vascular resistance (PVR). Cardiac output is a function of heart rate and stroke volume. If the pressure in a vessel increases then the blood flow will increase. However, if the resistance in a vessel increases then the blood flow will decrease. Vasodilation, flow of blood and arterial pressure can be examined and resistance to arterial pressure on cardiac output can be studied, determined and controlled.

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