UNIVERSITY OF MORATUWA, SRI LANKA
Faculty of Engineering
Department of Electronic and Telecommunication Engineering
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BM2012 - Anatomy and Physiology for Engineers

Assignment 1

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Introduction

The heart is a fist-sized organ which is responsible for pumping blood thought out the body. It is considered as the primary organ of the circulatory system.
The heart consists of four main chambers made up of muscle and powered by electrical pulses which are controlled by the brain and the nervous system.

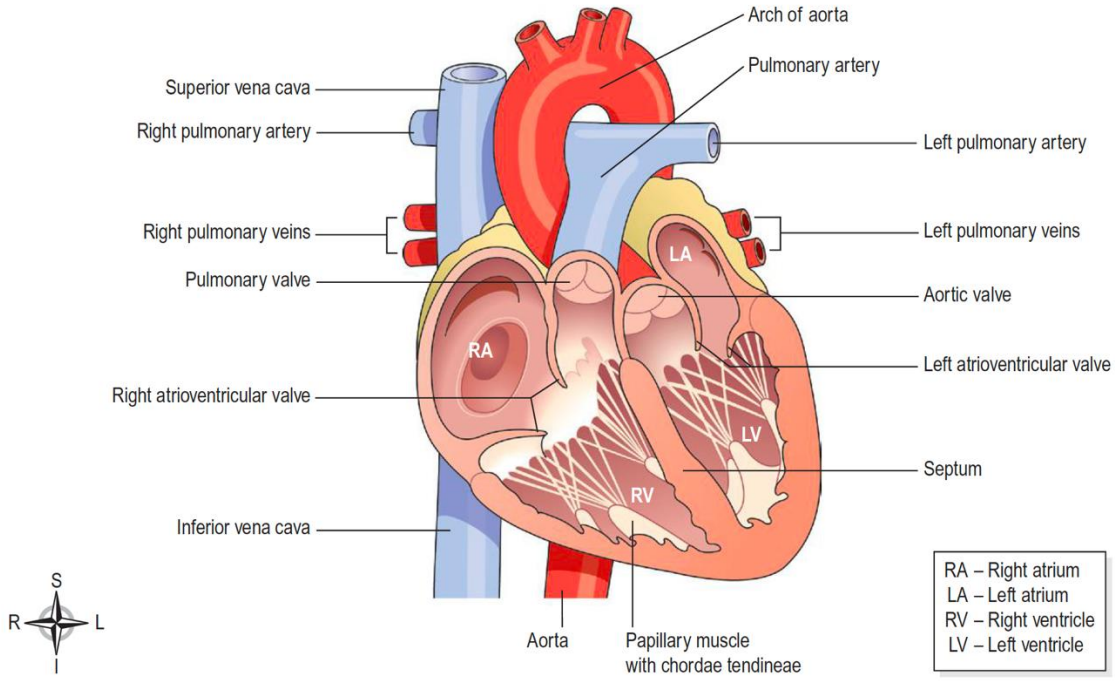

Figure 1 – Interior of the heart

The report discusses some of the main concepts associated with the heart which are the factors affecting heart rate, cardiac cycle and the factors affecting the cardiac output.

1. Heart Rate

The number of times the heart beats per minute is referred to as the heart rate and is a measure of the heart's activity.

The main factors affecting the heart rate are,

- Autonomic activities
- Circulating hormones
- Activity and exercise
- Body position
- Temperature
- Baroreceptor reflex

- Emotional states
- Age
- Gender

Autonomic nervous activities

The intrinsic rate at which the heart beats is a balance between sympathetic and parasympathetic activity and this is the most important factor in determining heart rate. The sympathetic nervous system has a wide variety of cardiovascular effects, including heart-rate acceleration, increased cardiac contractility, reduced venous capacitance. Conversely, the parasympathetic nervous system affects the cardiovascular system by slowing the heart rate through vagus nerve.

Circulating hormones

The hormones adrenaline (epinephrine) and noradrenaline (norepinephrine), secreted by the adrenal medulla, have the same effect as sympathetic stimulation, increasing the heart rate. Other hormones, including thyroxine, increase heart rate.

Hypoxia and elevated carbon dioxide levels stimulate heart rate. Electrolyte imbalances may affect it. For example, hyperkalaemia reduces cardiac function and leads to bradycardia (slow heart rate). Some drugs, such as β-receptor antagonists used in hypertension, can also cause bradycardia.

Activity and exercise

When the body is engaged in any activity or an exercise the relevant muscle needs more blood supply than when it is in the resting stage. This is achieved by increasing the heart rate and hence increasing the cardiac output. By local vasodilation the blood flow to the relevant muscle will be enhanced.

Body Position

The heart rate is usually faster when the body is in an upright position, than when laying down.

Temperature

The heart rate increases with the increase in body temperature.

Baroreceptor reflex

Baroreceptors are the nerve endings sensitive to touch which are located within the wall of aortic and carotid sinuses. They are the body's principle moment-to-moment regulatory mechanism for controlling the blood pressure. Baroreceptor control of blood pressure is known as baroreceptor reflex. Due to some baroreceptor reflex, the heart rate may change in order to regulate the blood pressure.

Emotional states

The heart rate changes based on the emotional state as well. For example, during excitement, fear or anxiety, the heart rate is increased.

Age

In new-borns and infants, the heart rate is more rapid than in older children and adults.

Gender

The heart rate is usually faster in women than in men.

2. Cardiac Cycle

The cardiac cycle includes the stages the heart goes through during each heartbeat, during which the heart contacts (systole) and the relaxes (diastole).

The stages of the cardiac cycle include,

- Atrial systole – contraction of the atria
- Ventricular systole – contraction of the ventricles
- Complete cardiac diastole – relaxation of the atria and ventricles

The following figure demonstrates the cardiac cycle taking 74 beats per minute as the heart rate which will allow each cycle a span of 0.8s.

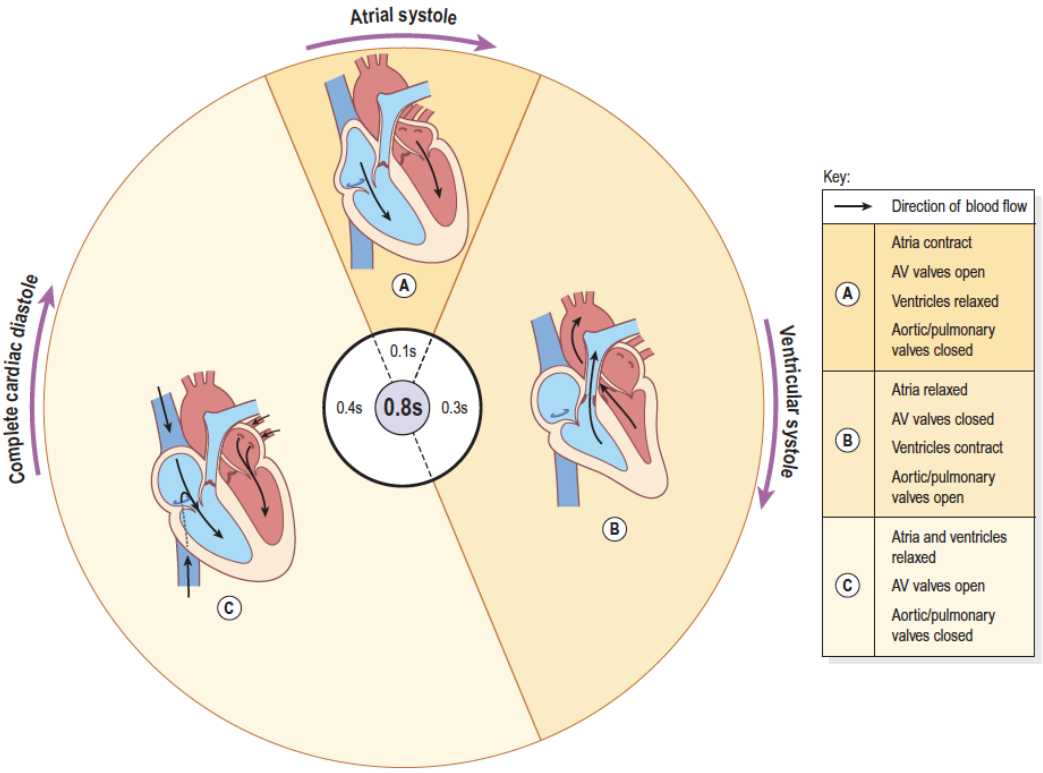

Figure 2 – Cardiac Cycle

The superior vena cava and the inferior vena cava transports deoxygenated blood from the body to the right atrium. At the same the four pulmonary veins bring oxygenated blood form the lungs into the left atrium.

Then the atrioventricular valves are opened and the blood in the atria flow passively into the ventricles. The Sinoatrial node (SA node) triggers a wave of contractions that spreads over the myocardium of both left and right atria will empty the blood in the atria and complete ventricular filling (Atrial systole stage of 0.1s).

The Atrioventricular node (AV node) is slowed down when the electric impulse reaches it. This will delay the atrioventricular transmission. This delay means that the mechanical result of atrial stimulation, atrial contraction, lags behind the electrical activity by a fraction of a second. This allows the atria to finish emptying into the ventricles before the ventricles begin to contract.

Then the AV node will trigger an electric impulse which will spread to the ventricular muscle. This results in a wave of contractions allowing walls of the ventricles to pump blood into the pulmonary artery and the aorta (Ventricular systole of 0.3 s). the high pressure generated during this stage will close the atrioventricular valves preventing the blood to flow into the atria.

After contraction of the ventricles there is complete cardiac diastole (a period of 0.4 s), when atria and ventricles are relaxed. During this time the myocardium recovers ready for the next heartbeat, and the atria refill ready for the next cycle.

3. Cardiac Output

Cardiac output is the amount of blood ejected from each ventricle per minute and is expressed in litres per minute (L/min). The cardiac output can be calculated as follows,

Cardiac output = Stroke volume x Heart rate

Where Stroke volume is the volume of blood expelled by each ventricle in each contraction.

The cardiac output is affected by the stroke volume as well as the heart rate. Hence it will be affected by the factors affecting stroke volume and the factors affecting heart rate. The latter was discussed previously. The factors affecting the stroke volume are as follows,

- Venous return
- Ventricular end-diastolic volume (VEDV) / preload
- Atrial blood pressure
- Blood volume

Venous return

This is the amount of blood returning to the heart (after circulating in the body) through the superior and inferior venae cavae and is the major determinant of the cardiac output.
Usually the force of contraction of the left ventricle ejecting blood into the aorta is not sufficient to push blood through the arterial and venous circulation and back to the heart. Therefore, the following factors are aiding in achieving the venous return.

- Position of the body (Due to gravity) – assists venous return from head and neck when standing or sitting and provides less resistance to venous return from the lower parts of the body when lying flat.

- Muscular contractions – backflow of blood in veins of the limbs (when standing) is prevented by valves. The contraction of skeletal muscles surrounding the deep veins compresses them, pushing the blood towards the heart resulting in a higher venous return.

- Respiratory pump – during inspiration, a negative pressure is created within the thorax by the expansion of the chest, assisting in blood flow towards the heart. In addition, when the diaphragm descends, the increased intra-abdominal pressure pushes blood towards the heart. Both these methods will result in a higher venous return.

By the increase in venous return, the stroke volume will be increased and hence increasing the cardiac output.

Ventricular end-diastolic volume (VEDV)

VEDV (also known as preload) is the volume of blood in the ventricles immediately before their contraction. This depends on the venous return. Increase in preload leads to stronger myocardial contractions due to the larger volume and hence more blood will be expelled increasing the stroke volume and thereby resulting in a higher cardiac output.
Within the physiological limits of the heart, it pumps out all the blood that it receives. If the limit is exceeded by any mean (more blood arrives in the atria than the ventricles can pump out), cardiac output decreases and the heart begins to fail.

Atrial blood pressure

This creates resistance against blood being pumped into the great arteries from the ventricles and hence affects the stroke volume. This resistance (also known as afterload) is determined by the elasticity of the large arteries and the peripheral resistance of arterioles.
Increase of this resistance increases the work that has been to done by the ventricles, since it increases the pressure against which they have to pump blood.
This may result in a decrease in stroke volume if the systemic blood pressure becomes significantly higher than normal.

Blood Volume

This is normally kept constant by the kidneys. If the blood volume decreases (by sudden haemorrhage), the stroke volume will fall causing a lower cardiac output. However, the body's compensatory mechanisms will attempt to return the blood level back to normal, unless the blood loss is too severe.

References

- Waugh, A. and Grant, A., 2014. *Ross & Wilson Anatomy & Physiology in Health and Illness*. 12th ed. Churchill Livingstone Elsevier