Class: Four
Subject: Mechanics of Artificial Organs 2
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Lecture # 5

The Design of Heart Valves / Part 1

1. Introduction

Our cardiovascular system transports important substances, such as oxygen and nutrients, between tissues and organs. It also helps transport and eliminates waste products. Our heart, blood vessels, and blood form a sophisticated network that transports materials around our body. These materials are carried by the blood through the blood vessels and are kept in motion by the pumping action of the heart. The blood vessels of the cardiovascular system are divided into two main pathways. The blood vessels in the pulmonary circuit carry blood from the heart to the lungs and back to the heart. The systemic circuit consists of the pathways between the heart and all other areas of the body.

2. The Heart

The heart is a hollow muscular structure that contracts in a rhythmic pattern to pump approximately 5–6 l of blood per minute. The human heart has a pair of pumps that are divided into four chambers. The two top chambers are called atria, and the two lower chambers are called ventricles (right and left) (Figure 1 a, b).

The two types of chambers in the heart perform different functions: The atria collect the blood that enters the heart and push it to the ventricles, while the ventricles push blood out of the heart and into the arteries to go to the rest of the body.

The two atria are separated by an interatrial septum, while the interventricular septum divides the two ventricles. The atrium and ventricle of each side of the heart communicate with each other via an atrioventricular orifice. This orifice can be opened or closed off by an atrioventricular valve, also known as an A–V valve. The left A–V valve is known as the bicuspid (or mitral) valve, while the right A–V valve is termed the tricuspid valve.

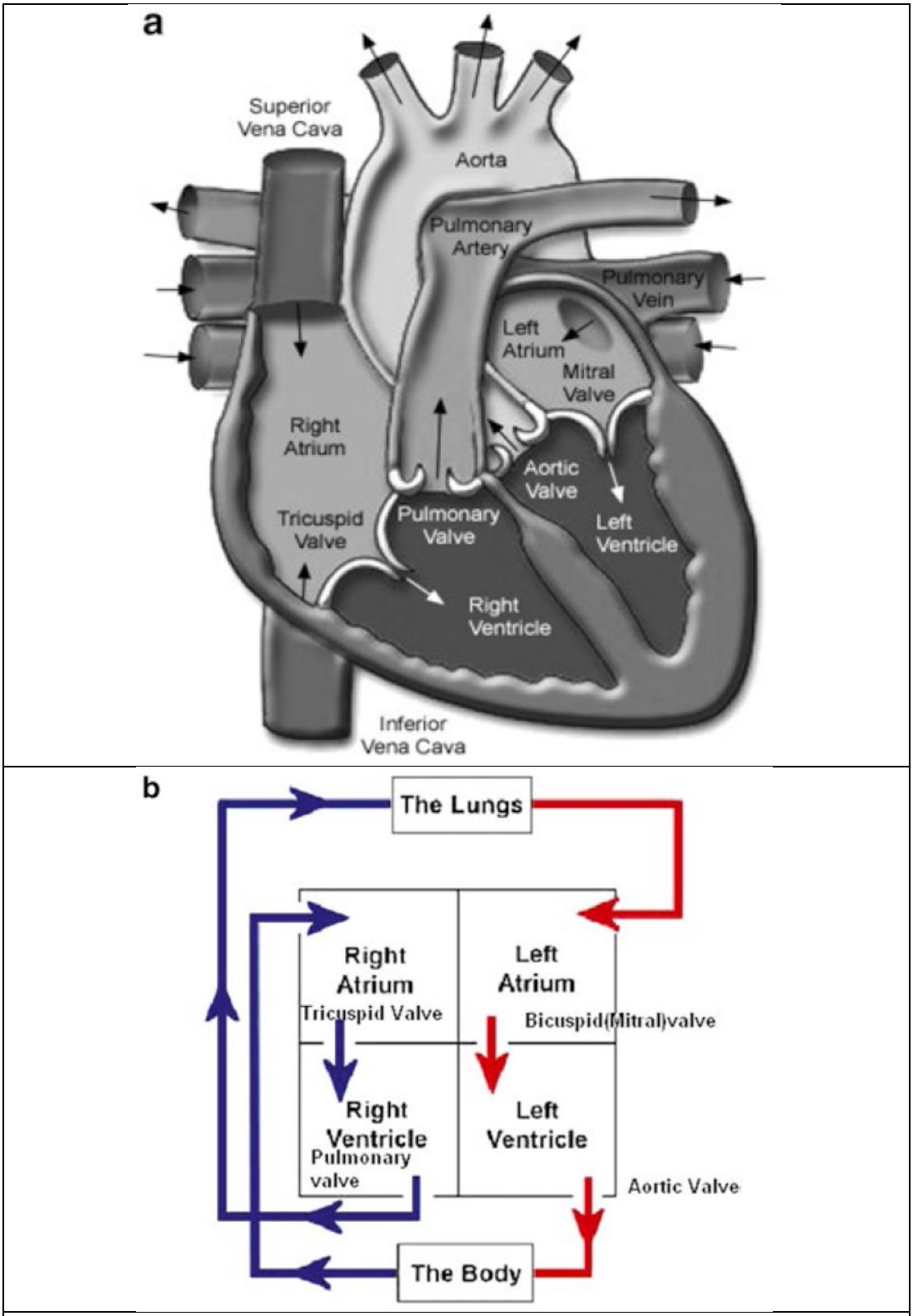

Figure 1: (**a**) The cutaway view of a human heart showing the pair of pumps with four chambers (acknowledgment: Texas Heart Institute); (**b**) schematic of the heart and blood flow in the entire human body

In order to move blood through heart, our heart chambers undergo alternating periods of relaxation (diastole) and contraction (systole), allowing the chambers to fill up with and pump blood, respectively (Figure 2).

The right atrium of the heart receives deoxygenated blood from two major veins: the superior vena cava and the inferior vena cava, as well as a smaller coronary sinus that drains blood from the heart wall. When this chamber contracts, blood moves out of the right atrium and goes into the right ventricle through the tricuspid valve.

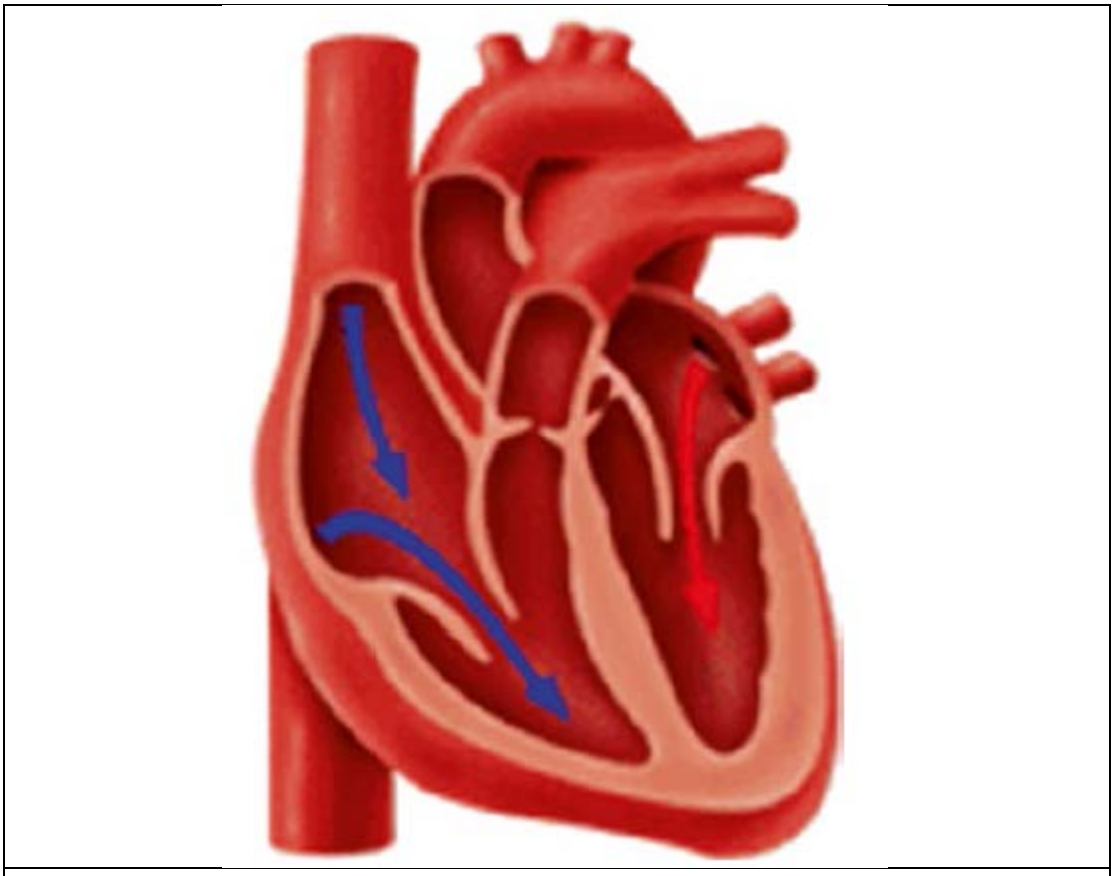

Figure 2: Flow through the heart

Once the right ventricle is sufficiently filled with blood, it contracts, pumping blood via the pulmonary arteries into the pulmonary circuit of the cardiovascular system.

Newly oxygenated blood enters the left atrium of the heart through pulmonary veins. Once this chamber is filled with blood, the left atrial wall will contract, pushing blood into the left ventricle through the bicuspid

valve. After the left ventricle is filled with blood, it contracts, forcing blood out of the ventricle and into the aorta. From the aorta, blood travels through the systemic circuit of the blood vessels, bringing oxygen to tissue cells throughout the body.

The internal cavity of the heart is divided into four chambers: **right atrium, right ventricle, left atrium, and left ventricle.** The two atria are thin-walled chambers that receive blood from the veins. The two ventricles are thick-walled chambers that forcefully pump blood out of the heart. Differences in thickness of the heart chamber walls are due to variations in the amount of myocardium present, which reflects the amount of pressure each chamber is required to generate. The right atrium receives deoxygenated blood from systemic veins; the left atrium receives oxygenated blood from the pulmonary veins.

3. Heart Valves

Heart valves act as one-way gates that allow blood to pass between heart chambers or from heart chambers to their associated blood vessels. They include the tricuspid and pulmonary valves for the right chambers and the bicuspid (or mitral) and aortic valves for the left chambers.

The tricuspid valve: The tricuspid valve is located between the atrium and ventricle on the right side of the heart. When this valve is open, blood passes from the right atrium into the right ventricle. The tricuspid valve prevents the reverse of blood flow back into the atrium by closing during ventricular contraction. As its name suggests, the tricuspid valve is made up of three leaves, or cusps (Figure 3).

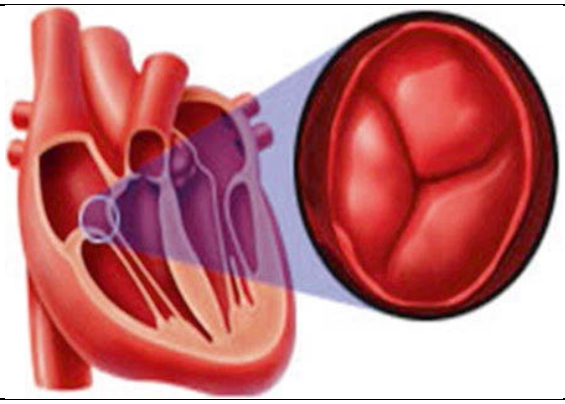

Figure 3: The tricuspid valve

The pulmonary valve: With the tricuspid valve closed, the only outlet for blood in the right ventricle is through the pulmonary trunk. The pulmonary trunk splits into the left and right pulmonary arteries, which connect to the left and right lungs, respectively. The entrance to the pulmonary trunk is guarded by the pulmonary valve. The pulmonary valve is made up of three leaves that open when the right ventricle contracts and close when this chamber relaxes, allowing blood to flow from the right ventricle into the pulmonary arteries but not the reverse (Figure 4).

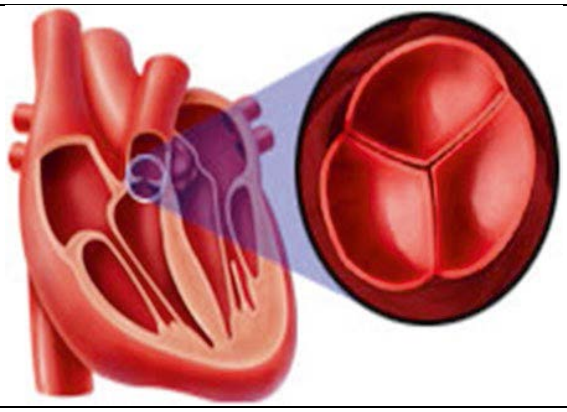

Figure 4: The tricuspid valves closed

The bicuspid valve: The bicuspid or mitral valve regulates the flow of blood from the left atrium to the left ventricle. Like the tricuspid valve, the bicuspid valve closes during ventricular contraction. The bicuspid valve is composed of two leaves (Figure 5).

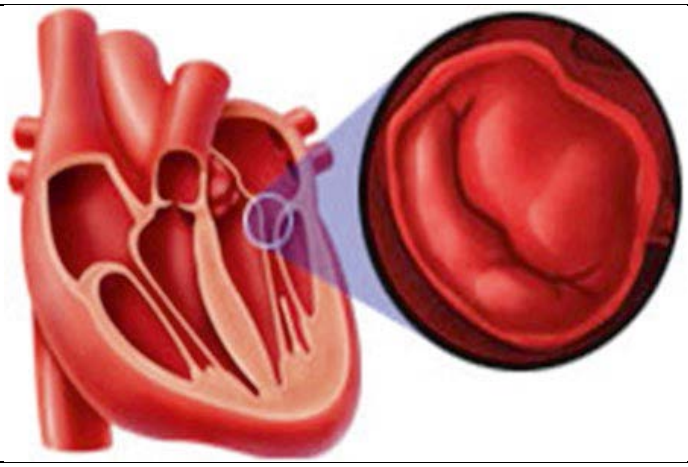

Figure 5: The bicuspid valve

The aortic valve: The aortic valve consists of three leaves found at the entrance to the aorta. This valve lets blood out of the left ventricle as it

contracts and blocks the pathway of blood from the aorta back into the left ventricle when this chamber relaxes (Figure 6).

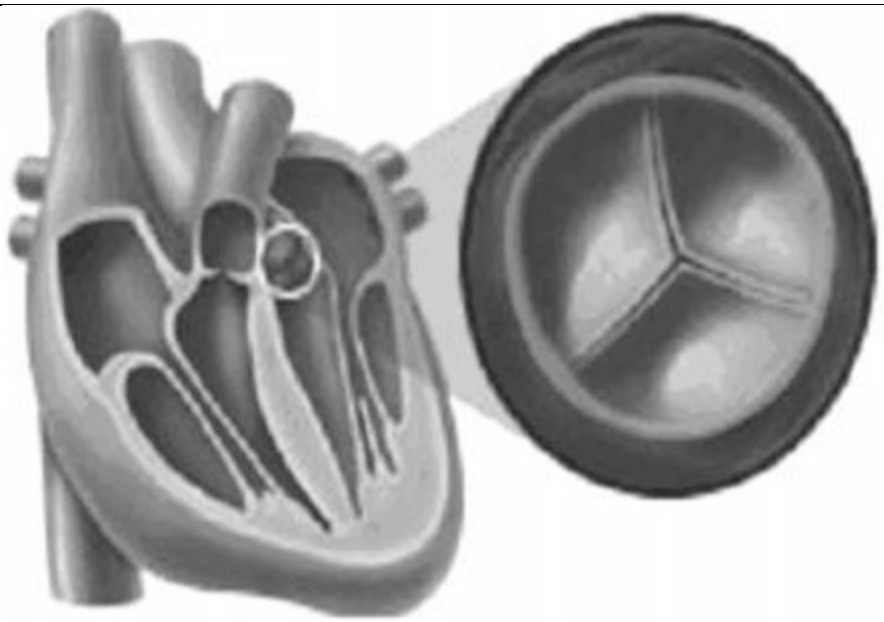

Figure 6: The aortic valve

4. Cardiac Cycle

The cardiac cycle consists of two parts: systole (contraction of the heart muscle) and diastole (relaxation of the heart muscle). Atria contract while ventricles relax. The pulse is a wave of contraction transmitted along the arteries.

Valves in the heart open and close during the cardiac cycle. Heart muscle contraction is due to the presence of nodal tissue in two regions of the heart:

The SA node (sinoatrial node) initiates heartbeat.

The AV node (atrioventicular) causes ventricles to contract. The AV node is sometimes called the pacemaker since it keeps the heart beating regularly.

The heartbeat is also controlled by the autonomic nervous system (Figure 7). Blood flows through the heart from veins to atria to ventricles and goes out via arteries. Heart valves limit the blood flow to a single direction. One heartbeat, or cardiac cycle, includes atrial contraction and relaxation, ventricular contraction and relaxation, and a short pause. Normal cardiac cycles (at rest) take 0.8 s. Blood from the body flows into the vena cava,

which empties into the right atrium. At the same time, oxygenated blood from the lungs flows from the pulmonary vein into the left atrium. The muscles of both atria contract, forcing blood downward through each AV valve into each ventricle.

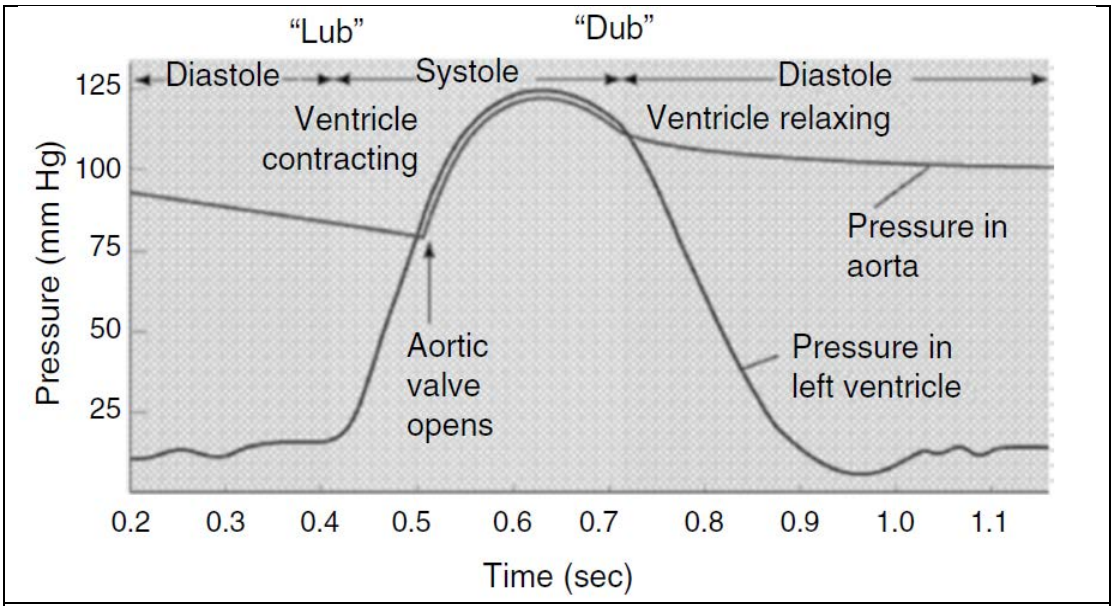

Figure 7: The cardiac cycle.

Diastole is the filling of the ventricles with blood. Ventricular systole opens the SL valves, forcing blood out of the ventricles through the pulmonary artery or aorta. The sound of the heart contracting and the valves opening and closing produces a characteristic "lub-dub" sound. Lub is associated with closure of the AV valves; dub is the closing of the SL valves.

Human heartbeats originate from the sinoatrial node (SA node) near the right atrium. Modified muscle cells contract, sending a signal to other muscle cells in the heart to contract. The signal spreads to the atrioventricular node (AV node). Signals carried from the AV node, slightly delayed, through bundles of His fibers and Purkinje fibers cause the ventricles to contract simultaneously (Figure 8).

An electrocardiogram (ECG) measures changes in electrical potential across the heart and can detect the contraction pulses that pass over the surface of the heart. There are three slow, negative changes, known as P, R, and T. Positive deflections are the Q and S waves. The P wave represents the contraction impulse of the atria, the T wave the ventricular contraction. ECGs are useful in diagnosing heart abnormalities (Figure 9).

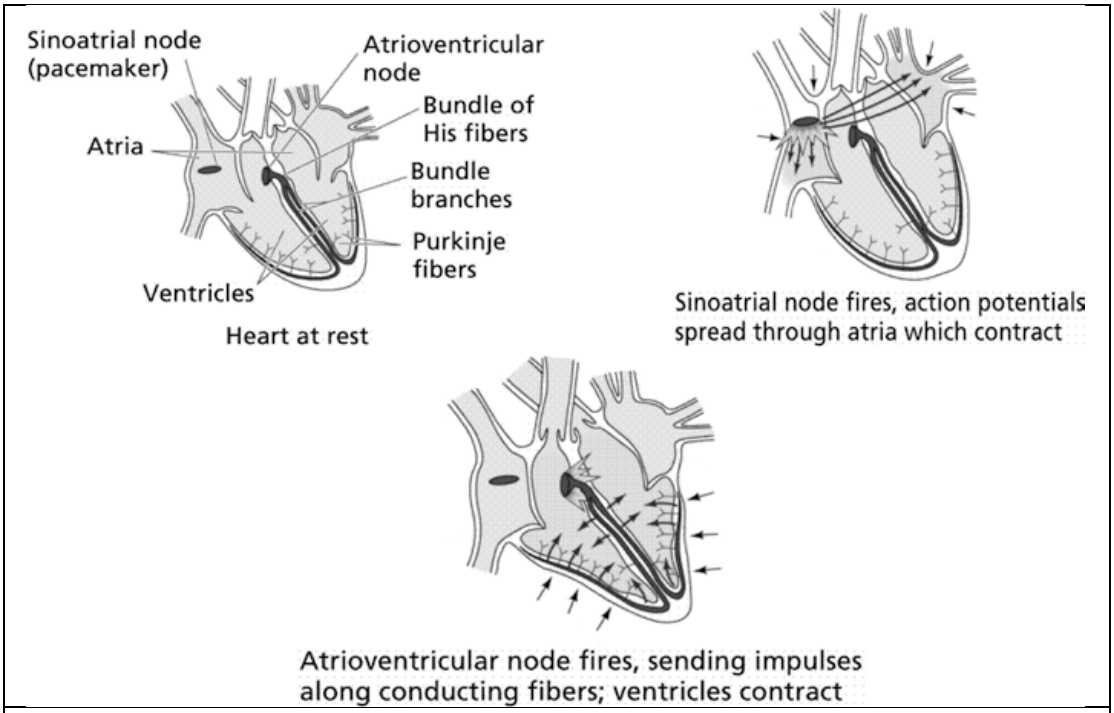

Figure 8: The contraction of the heart and the action of the nerve nodes located on the heart

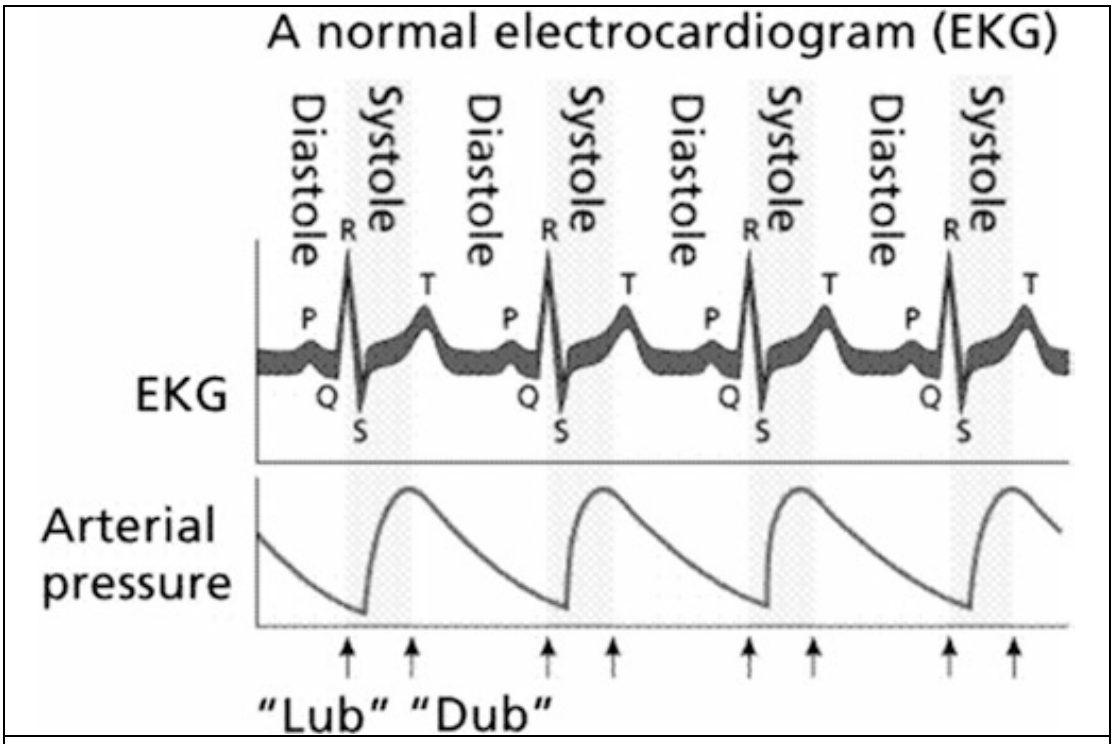

Figure 9: Normal ECG/EKG pattern and arterial pressure variation

5. Cardiac Output

Cardiac output (CO) is the product of the heart rate (HR) and stroke volume (SV):

$$CO = HR\ SV.$$

For a 70-kg man, normal values are HR = 70/min and SV = 70 ml, giving a cardiac output of about 5 l/min. The cardiac index is the cardiac output per square meter of body surface area; normal values range between 2.5–4.0 l/min/m2.

Heart rate is determined by the rate of spontaneous depolarization at the sinoatrial node (see above) but can be modified by the autonomic nervous system. The vagus nerve acts on muscarinic receptors to slow the heart, whereas the cardiac sympathetic fibers stimulate beta-adrenergic receptors and increase heart rate.

Stroke volume is determined by three main factors: preload, and contractility and afterload. These will be considered in turn:

Preload is the ventricular volume at the end of diastole. An increased preload leads to an increased stroke volume. Preload is mainly dependent on the return of venous blood from the body. Venous return is influenced by changes in position, intrathoracic pressure, blood volume, and the balance of constriction and dilatation (tone) in the venous system. The relationship between ventricular end-diastolic volume and stroke volume is known as Starling's law of the heart, which states that the energy of contraction of the muscle is related, or proportional, to the initial length of the muscle fiber. This is graphically illustrated in Figure10 by a series of "Starling curves."

Curves A and B illustrate the rise in cardiac output with increases in ventricular end-diastolic volume (preload) in the normal heart. Note that with an increase in contractility, there is a greater cardiac output for the same ventricular end-diastolic volume.

In the diseased heart (C and D), cardiac output is less and falls if ventricular end-diastolic volume rises to high levels, as in heart failure or overload.

As the volume at the end of diastole (end-diastolic volume) increases and stretches the muscle fiber, so the energy of contraction and the stroke

volume increase, until a point of overstretching when the stroke volume may actually decrease, as in the failing heart. Cardiac output will also increase or decrease in parallel with stroke volume if there is no change in heart rate.

The curves show how the heart performs at different states of contractility, ranging from the normal heart to one in cardiogenic shock. This is a condition where the heart is so damaged by disease that cardiac output is unable to maintain tissue perfusion. Also shown are increasing levels of physical activity, which require a corresponding increase in cardiac output.

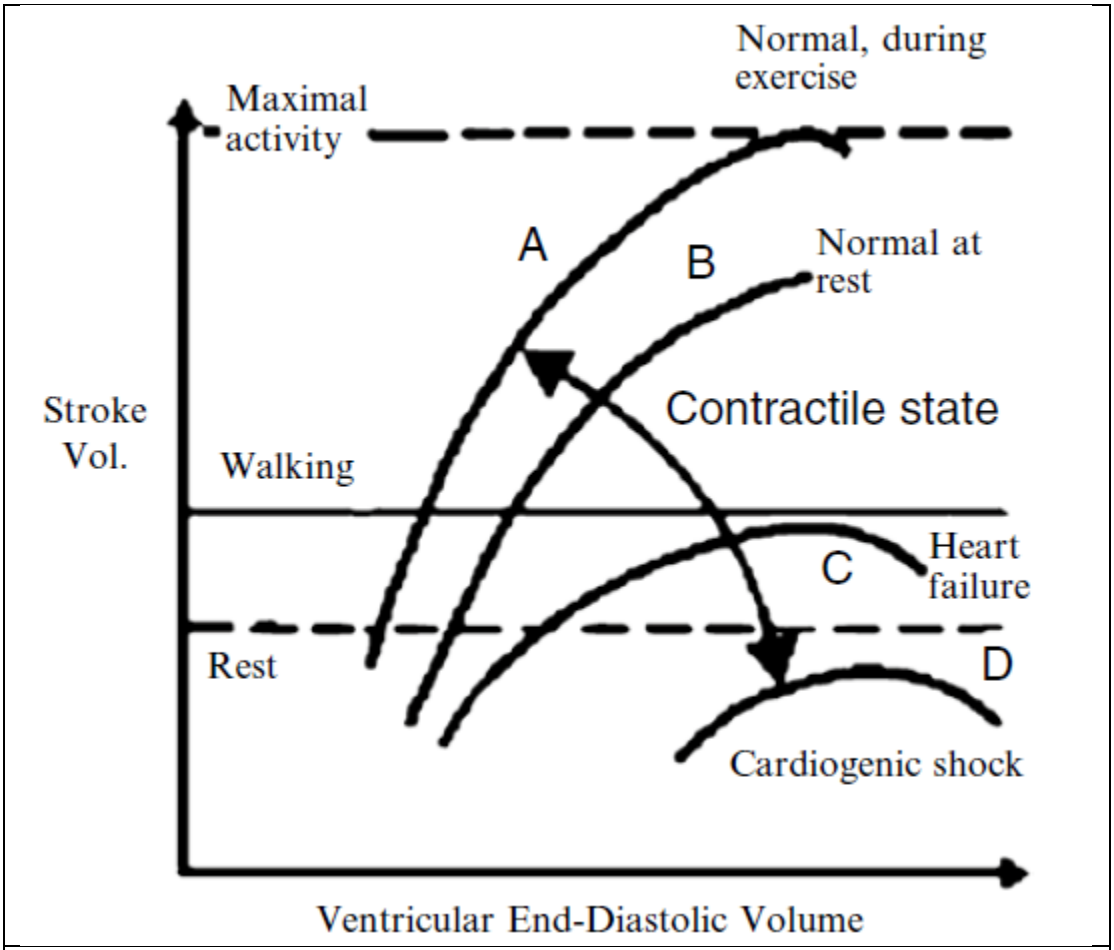

Figure 10: Stroke volume vs. VED volume

Afterload is the resistance to ventricular ejection. This is caused by the resistance to flow in the systemic circulation and is the **systemic vascular resistance**. The resistance is determined by the diameter of the arterioles and precapillary sphincters; the narrower or more constricted this diameter is, the higher the resistance is. The level of systemic vascular resistance is

controlled by the sympathetic system, which, in turn, controls the tone of the muscle in the wall of the arteriole, and hence the diameter. The resistance is measured in units of dyne·s/cm5. A series of Starling curves with differing afterloads is shown in Figure 11 demonstrating a fall in the stroke volume as the afterload increases.

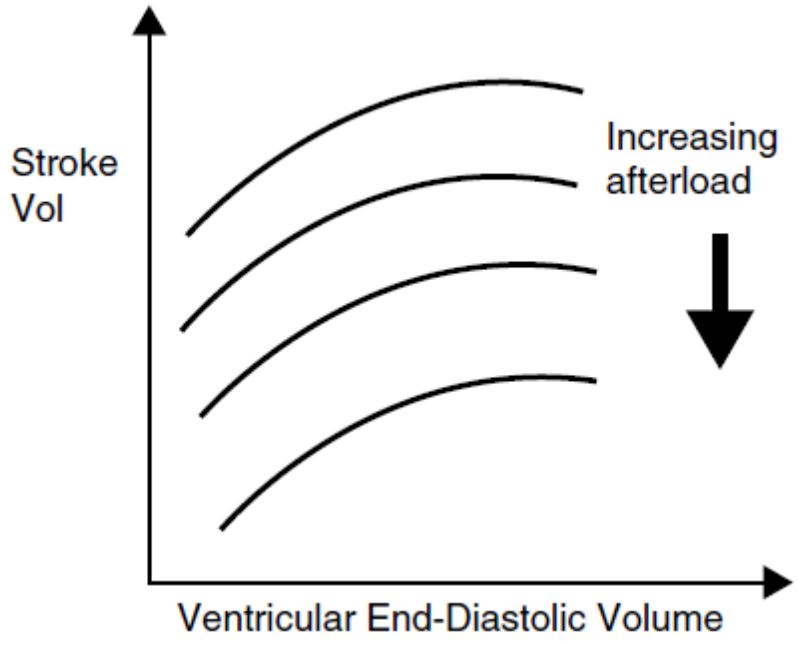

Figure 11: Stroke volume vs. ventricular end-diastolic volume

A series of curves illustrates the effects of increasing the afterload on systemic vascular resistance. As the afterload increases, the patient moves to a lower curve, with a lower stroke volume for the same ventricular end-diastolic volume (preload).

The relationship between the systemic vascular resistance and the control of arterial pressure is discussed below.

Contractility describes the ability of the myocardium to contract in the absence of any changes in preload or afterload. In other words, it is the "power" of the cardiac muscle. The most important influence on contractility is the sympathetic nervous system. Beta-adrenergic receptors are stimulated by noradrenalin released from nerve endings, and contractility increases. A similar effect is seen with circulating adrenaline and drugs such as ephedrine, digoxin, and calcium. Contractility is reduced

by acidosis, myocardial ischemia, and the use of beta-blocking and anti-arrhythmic agents.

Cardiac output will change to match changing metabolic demands of the body. The outputs of both ventricles must be identical and also equal the venous return of blood from the body. The balancing of cardiac output and venous return is illustrated during the response to exercise. Blood vessels dilate in exercising muscle groups because of increased metabolism, and the blood flow increases. This increases the venous return and right ventricular preload. Consequently, more blood is delivered to the left ventricle, and the cardiac output increases. There will also be increased contractility and heart rate from the sympathetic activity associated with exercise, further increasing cardiac output to meet tissue requirements.

Cardiac Reserve

- Cardiac reserve is the difference between cardiac output at rest and the maximum volume of blood the heart is capable of pumping per minute.
- It permits cardiac output to increase dramatically during periods of physical activity.

Flow Rate Through Blood Vessels

- Directly proportional to the pressure gradient
- Inversely proportional to vascular resistance

Flow = difference in pressure/resistance

Pressure gradient = difference in pressure between beginning and end of vessel (pressure = force exerted by blood against vessel wall and measured in millimeters of mercury).

Blood Flow

The relationship between the flow and the driving pressure is given by the Hagen– Poisseuille formula. This states that the flow rate Q in a tube is proportional to

$Q \alpha$ driving pressure × Radius / (Length × Viscosity).

In blood vessels, the flow is pulsatile rather than continuous, and the viscosity varies with the flow rate, so the formula is not strictly applicable,

but it illustrates an important point; small changes in radius result in large changes in flow rate. In both arterioles and capillaries, changes in flow rate are brought about by changes in tone and therefore in vessel radius.

Viscosity describes the tendency of a fluid to resist flow. At low flow rates, the red blood cells stick together, increasing viscosity, and remain in the center of the vessel. The blood closest to the vessel wall (which supplies side branches) therefore has a lower hematocrit. This process is known as plasma skimming. Viscosity is reduced in the presence of anemia, and the resulting increased flow rate helps maintain oxygen delivery to the tissues.

6. Artificial Heart Valves

An artificial (mechanical) heart valve is a manmade device that is used to replace one of a patient's own damaged or diseased heart valves that cannot be repaired. A biological valve, from either an animal (xenograft) or a deceased human donor (allograft), may also be used to replace the patient's damaged or diseased valve. In most cases, the use of an artificial heart valve can lengthen or even save a patient's life. The valves are durable and can last 30 years or longer. However, there is a risk of complications, and most patients will need to take anticoagulants for the rest of their lives to reduce the risk of blood clot formation. An artificial heart valve is inserted into the patient's heart as part of an open-heart surgery called heart valve replacement (Figure 12).

Any of the patient's four heart valves (aortic valve, mitral valve, pulmonic valve, or tricuspid valve) may be replaced with an artificial heart valve. However, artificial heart valves tend to last about 30 years or even more, which is significantly longer than biological valves last. Usually, a porcine (pig) valve suits the human body. The weight of the pig has to be approximately 60–70 kg to attain a suitable size for a human valve. It is estimated that each year, almost 100,000 people will need a heart valve replacement. In India, at the start of this century, the current requirement was about 6,000–8,000 or more valves annually. It will increase at a much faster rate in the future due to the increase in aging patients, the adoption of a Western lifestyle, and the expectation for a good-quality life.

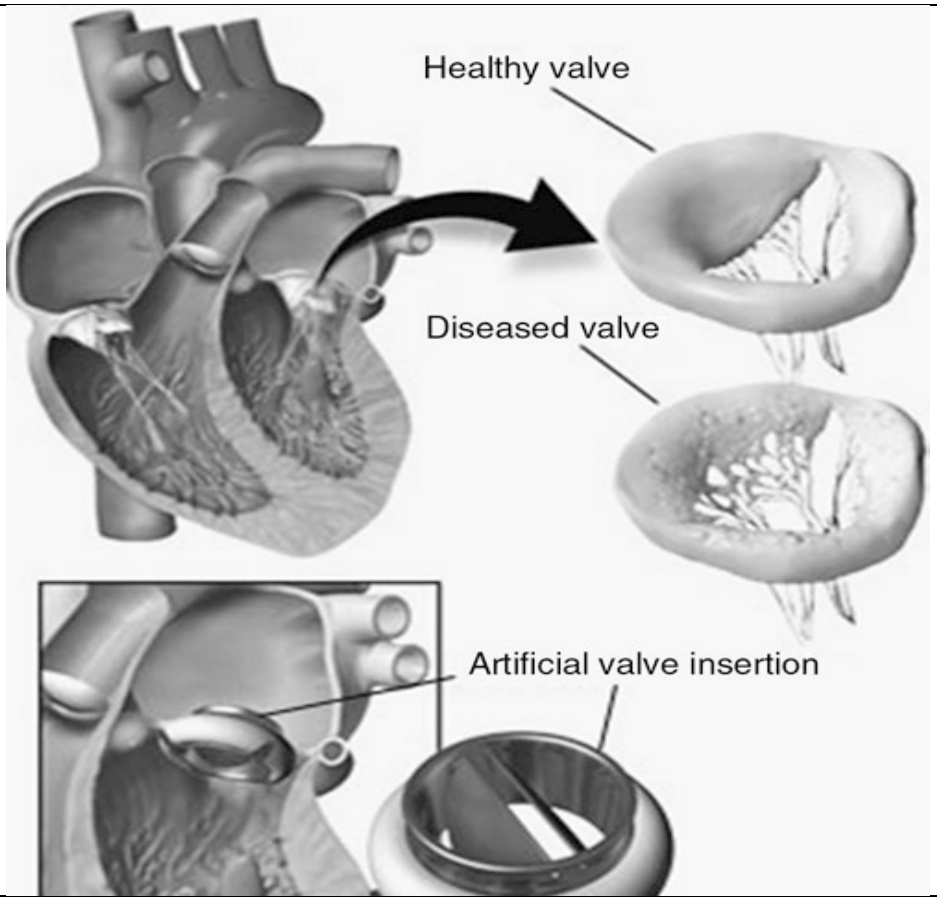

Figure 12: Artificial heart valve and its placement

5.1 Potential Risks of Artificial Heart Valves

In most cases, replacing a diseased or damaged **heart valve** can lengthen or even save a life. Untreated, **valvular heart disease** can lead to heart failure and death.

There are a number of rare but possible complications that could arise from the surgery needed to insert the artificial heart valve (see **Heart Valve Replacement**). Rarely, there may be a problem with the artificial heart valve itself. Patients are encouraged to document the make, model, and serial number of their artificial heart valve in case any problems with it are announced in the future.

5.2 Lifestyle Considerations

People with artificial heart valves will need follow-up visits to have a painless echocardiogram, which can reveal any malfunction or leaking of the valve. People with artificial heart valves face an increased risk of

developing a potentially dangerous inflammation called bacterial endocarditis. This risk is increased when bacteria enter the bloodstream, such as during a dental, medical, or surgical procedure, and infect the tissue surrounding the valve, or the valve itself. Therefore, people with artificial heart valves are generally advised by their physicians to take antibiotics before any of these procedures to minimize their risk of bacterial endocarditis. All people with artificial heart valves will also need to take medications called anticoagulants for the remainder of their lives. These medications help prevent the body's natural response of forming blood clots around a foreign object, such as an artificial heart valve. Anticoagulants reduce the risk of blood clot formation, thus reducing the patient's risk of valve malfunction, stroke, and other potentially dangerous complications. Patients taking anticoagulants may need to undergo regular blood tests to monitor their medication dosage. It is also wise for patients carry a form of identification (card, bracelet) stating that they have an artificial heart valve, in case of emergency.

5.3 Longevity and Replacements

On average, an artificial heart valve can last 30 years or more. The patient will need to see his or her physician for regular follow-up to detect any signs that the artificial heart valve is beginning to wear out. These signs include an unusual heart murmur or leakage that is growing more severe. As the leakage gets more severe, the patient may begin to notice symptoms such as shortness of breath or chest pain. Patients are urged to contact their physician if they begin to notice these types of symptoms (Figure 13).

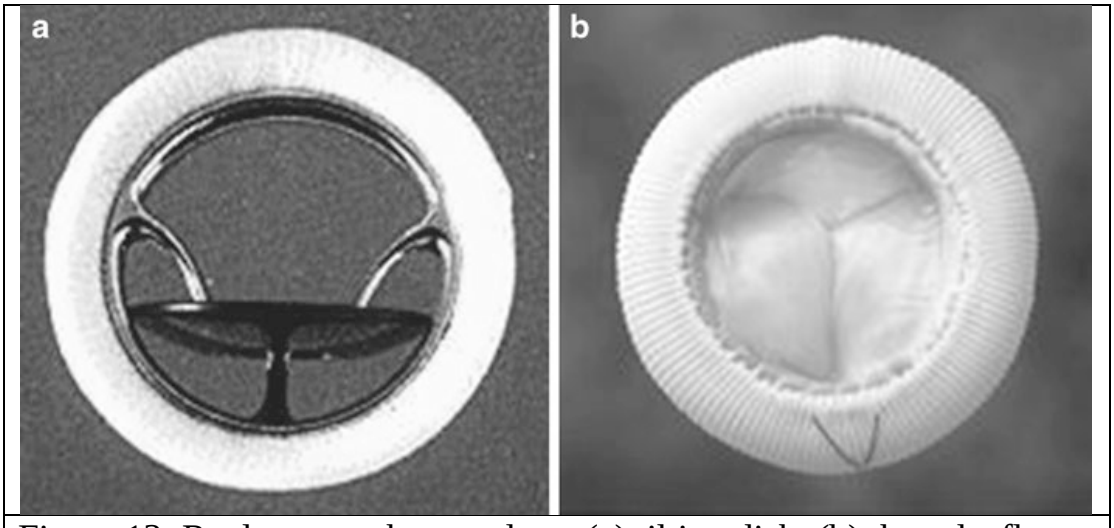

Figure 13: Replacement heart valves: (a) tilting disk; (b) three-leaflet

5.4 Design of Valves

By comparison with the revolutionary changes in many other fields of medicine, the development of artificial heart valves has progressed rather gradually since its beginning in 1960. The main problems recognized with the first designs, thromboembolism in mechanical valves and structural deterioration in tissue valves, were reduced to an acceptable level by 1965–1970 and were not substantially reduced further in the ensuing 25 years. However, the years since 1995 have seen a resurgence in the development cycle, and many new devices are currently under investigation.

Heart valves can be classified into two broad categories according to the origin of their occluding mechanism: **(1) mechanical** and **(2) biological**. The introduction of the heart-lung machine made valve replacement a possibility in 1955; many replacement devices were attempted. Mechanical and biological valves of all kinds were tried, including designs with one, two, three, and four leaflets. The first valve replacements that led to long-term survivors were mechanical cage ball valves used by the Harken in the aortic position and Starr in the mitral position, both in 1960.

Once the early problems of valve fixation and durability were solved, the major concern that drove mechanical valve development was the reduction of thromboembolic complications. One favorite design was the tilting disk valve, several varieties of which exist. The original versions of the bileaflet valve did not endure, but this design was successfully implemented in 1977 on the basis of the transfer of pyrolytic carbon technology from spacecraft to heart valves. Thus, the mechanical valve designs that have prevailed until today are the **ball valve, tilting disk valve, and the bileaflet valve**. Current development of mechanical valves is concentrated in attempting to enhance the bileaflet valve design.

The first biological valves used successfully were transplants from human cadavers, called allografts, pioneered by Ross and Barratt-Boyes in 1962. The successful use of autologous grafts was begun with the pulmonary autograft in 1967. The goal of these biological valves was to reduce the complications associated with thromboembolism and the need for anticoagulation. Several homologous and heterologous materials were used to fabricate tissue valves but were eventually abandoned. During the 1960s, a major advance was the use of glutaraldehyde for the preservation of porcine valves pioneered by Carpentier and coworkers. Glutaraldehyde-

fixed valves currently in use are aortic porcine valves and, in resurgence, valves fabricated of bovine pericardium. Current developments in tissue valve technology include improved methods of fixation, calcification mitigation treatments, and stentless designs.