Al-Mustaqbal University College

Pharmacy Department / Second Stage

PHYSIOLOGY

CARDIOVASCULAR SYSTEM, L1

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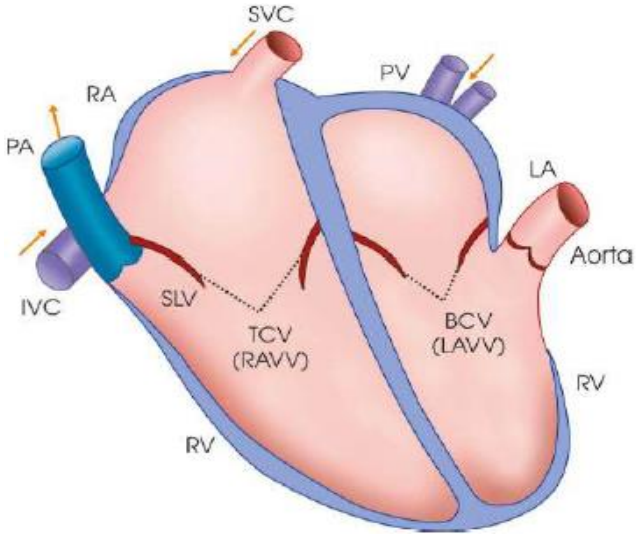
The primary function of the cardiovascular system is to provide an adequate supply of oxygen and nutrients to all cells of the body and carry away the waste products of their metabolism. The principal components of the cardiovascular system are:

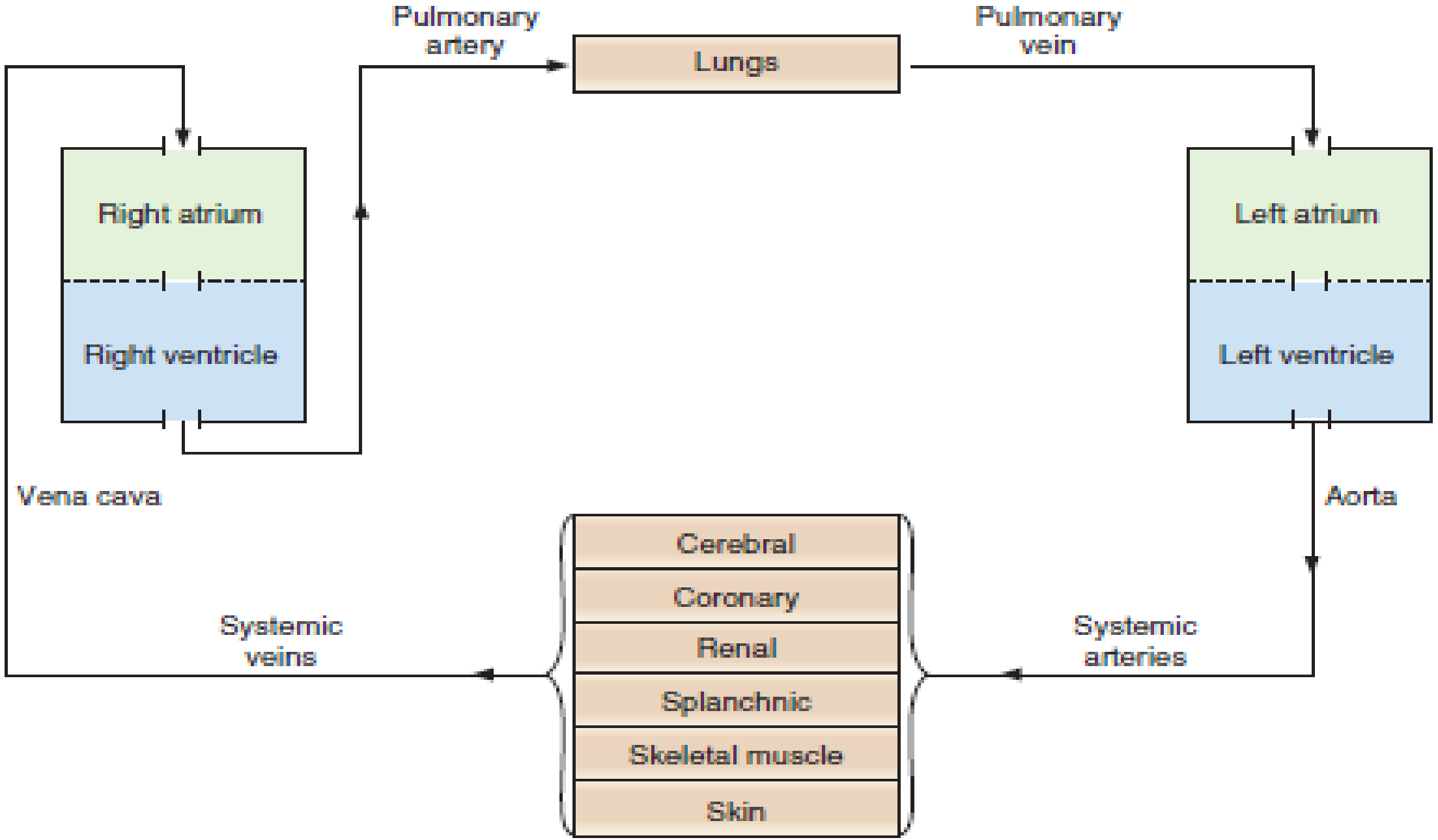
1- blood 2-blood vessels 3-the heart.

Blood carries materials to and from the tissues, blood vessels are conduits that bring blood close to cells, and the heart is used to create the pressure that is needed to propel blood around the system.

The heart has four chambers two ventricles and two atria: both right and left. The two left chambers are separated from the two right ones, by a continuous partition, the atrial portion of which is called the interatrial septum (fibrous). The ventricular part is known as the interventricular septum

- From the left ventricle arises the aorta, carrying oxygenated blood to the tissues.
- From the right ventricle, which is less muscular than the left, arises the pulmonary trunk, carrying reduced blood to the lungs.
- The right atrium receives all the venous blood from the body through three veins; the inferior and the superior venae cavae, and the coronary sinus.
- The left atrium receives all the oxygenated blood from the lungs through pulmonary veins.




Pulmonary artery
Lungs
Pulmonary vein
Right atrium
Right ventricle
Left atrium
Left ventricle
Vena cava
Aorta
Cerebral
Coronary
Renal
Splanchnic
Skeletal muscle
Skin
Systemic veins
Systemic arteries

Heart structure

Blood is propelled around the vasculature by two muscular pumps, one on each side of the heart Each pump contains two chambers: one **atrium** and one **ventricle**. The left heart pumps blood through the **aorta** to the organs of the systemic circulation. It returns to the heart via the **vena cavae**. The right side of the heart perfuses the pulmonary circulation. Blood exits the right ventricle via the **pulmonary artery**, passes through the lungs, and then enters the left heart via the **pulmonary vein**.

Cardiac chambers

1. Atria: Accumulated blood is transferred to the ventricles by atrial contraction at the beginning of each cardiac cycle. Minimal amounts of pressure are required to push blood into the ventricles and, therefore, atrial walls contain relatively small amounts of muscle and are thin.

2. Ventricles :

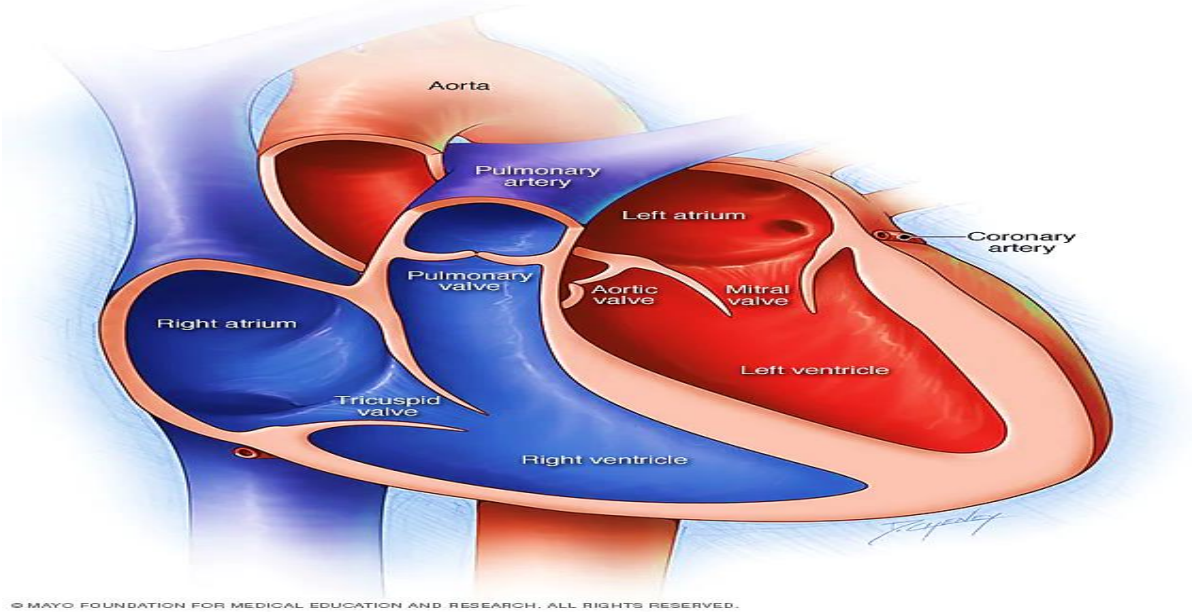
Ventricles drive blood at high pressure through vast networks of vessels, made possible by chamber walls that are thick with cardiac muscle. The left ventricle (LV) typically generates peak pressures of 120 mm Hg. The right ventricle (RV) pumps blood through a system of relatively low-resistance vessels and, therefore, its walls are less muscular than those in the LV. The RV generates peak pressures of about 20 mm Hg.

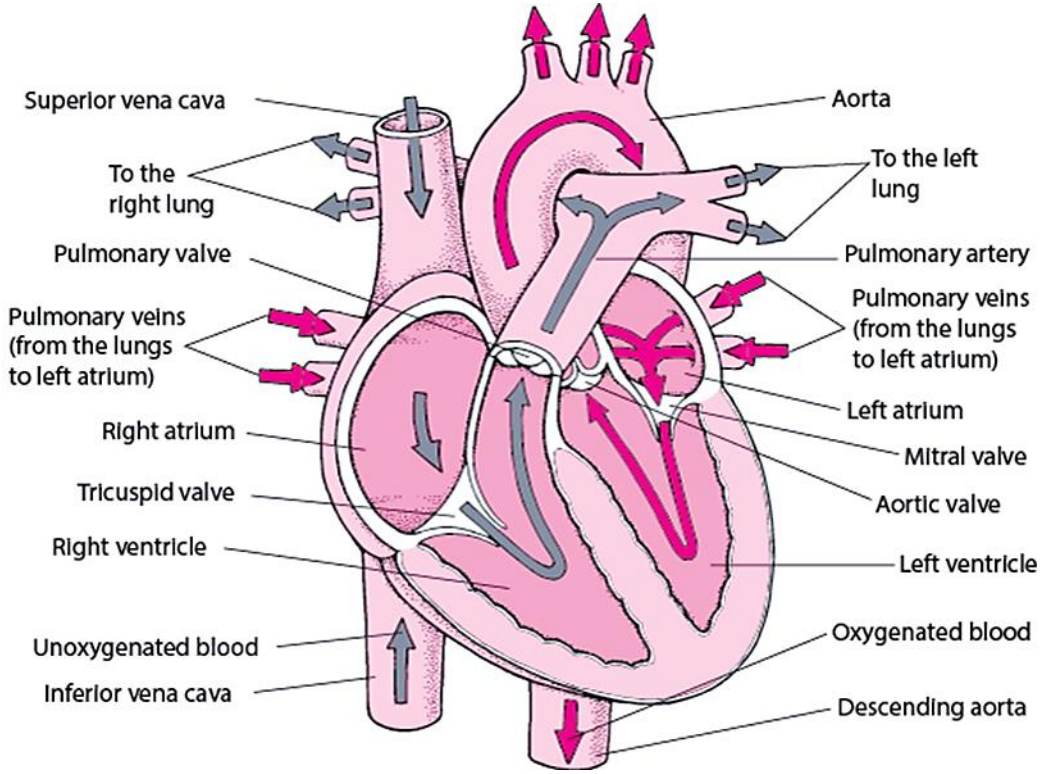
Valves :

One-way valves situated between atria and ventricles (**atrioventricular** [**AV**] **valves**) and between ventricles and their outlets (**semilunar valves**) help ensure that flow around the cardiovascular system is unidirectional(to prevent regurgitation of blood)

1. Atrioventricular: The **tricuspid** (right side) and **mitral** (left side) valves allow blood to pass from atrium to ventricle and close when ventricular contraction begins.

2. Semilunar: The **pulmonary** (right side) and **aortic** (left side) valves prevent back flow from the arterial system into the ventricles.





circulatory system has been divided into two functionally opposite parts:

l. *Systemic circulation*

2. *Pulmonary circulation*

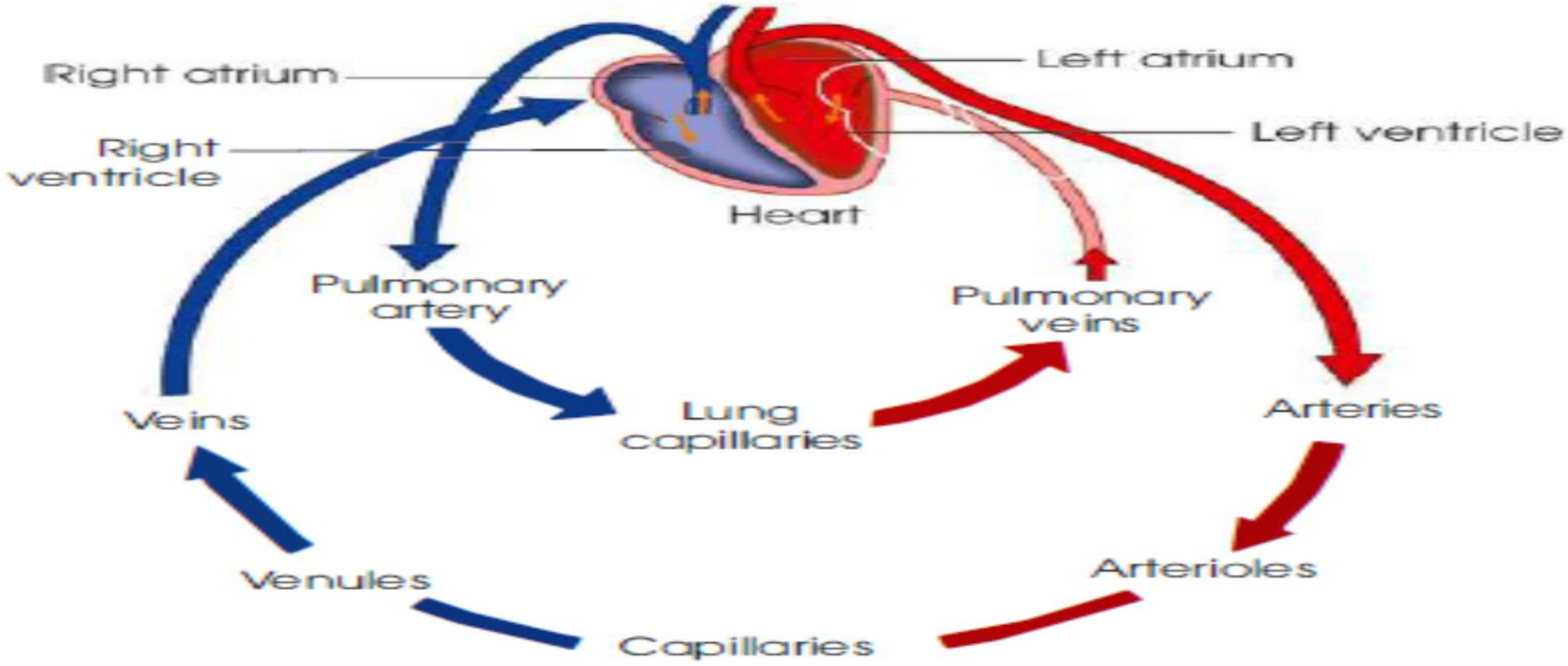


Fig. 29.2: Relationship between systemic and pulmonary circulation

The systemic circulation, begins in the left ventricle and ends in the right atrium.

The pulmonary circulation starts in the right ventricle and ends in the left atrium. The right half of the heart is concerned with reduced blood(deoxygenated), while the left half with oxygenated blood.

CARDIAC CYCLE : The cardiac events that occur from the beginning of one heartbeat to the beginning of the next are called the *cardiac cycle.* Each cycle is initiated by spontaneous generation of an action potential in the *sinus node.*

Diastole and Systole : The cardiac cycle consists of a period of relaxation called *diastole,* during which the heart fills with blood, followed by a period of contraction called *systole*

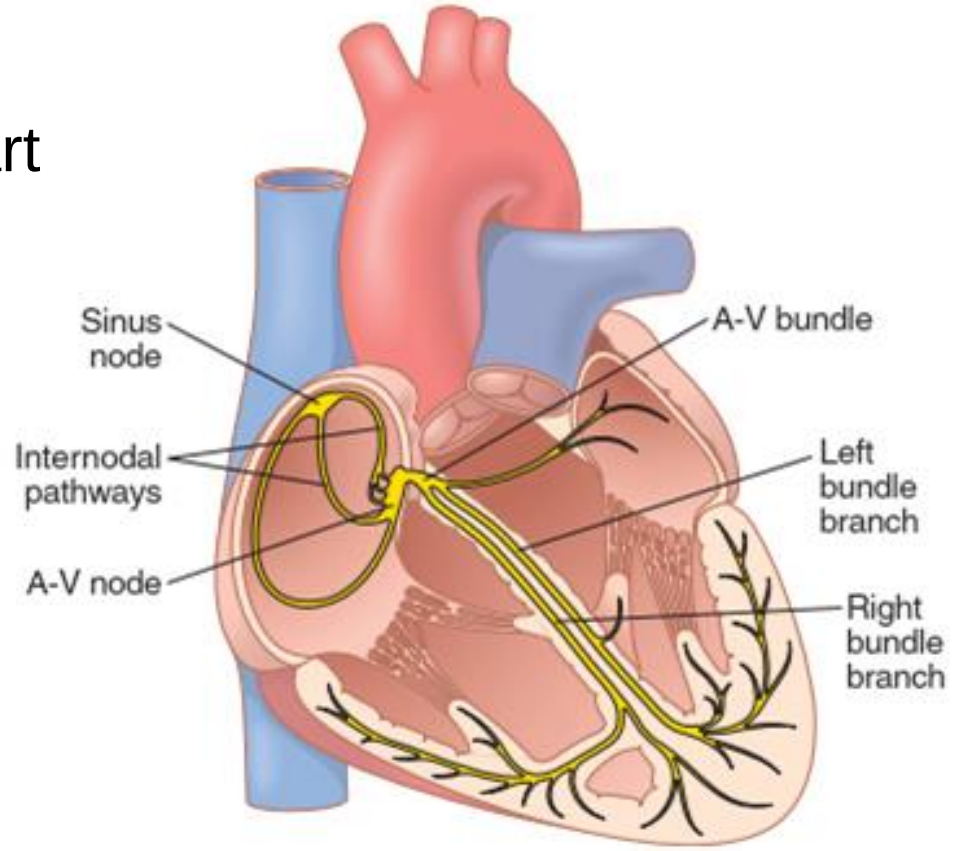
The resting membrane potential is the result of the movement of several different ions through various ion channels and transporters (uniporters, cotransporters, and pumps) in the plasma membrane. These movements result in different electrostatic charges across the cell membrane. Neurons and muscle cells are excitable such that these cell types can transition from a resting state to an excited state. In the resting state, K ions are found mainly intracellular, while Na and Ca ions are found extracellular making the interior of the cells electrically negative.

Unlike the action potential in skeletal muscle cells, the cardiac action potential is not initiated by nervous activity. Instead, it arises from a group of specialized cells known as pacemaker cells, that have automatic action potential generation capability.

The Conductive System of the Heart

The human heart has a special system for rhythmic self- excitation and repetitive contraction approximately 100,000 times each day, or 3 billion times in the average human lifetime.

This rhythmical and conductive system of the heart is susceptible to damage by heart disease, especially by ischemia of the heart tissues resulting from poor coronary blood flow. The effect is often a bizarre heart rhythm or abnormal sequence of contraction of the heart chambers, and the pumping effectiveness of the heart often is affected severely, even to the extent of causing death.



Sinus node and the Purkinje system of the heart, showing also the atrioventricular *(A-V)* node, atrial internodal pathways, and ventricular bundle branches.

A. Pacemaker(SA node)

The SA node comprises a group of specialized cardiac myocytes located near the superior vena cava in the wall of the right atrium generate spontaneous APs. The rate at which APs are initiated and, thus, heart rate (HR), is under simultaneous control of both arms of the **autonomic nervous system** (**ANS**). The **sympathetic nervous system** (**SNS**) increases HR, whereas the **parasympathetic nervous system** (**PSNS**) decreases it.

B- Atrioventricular node(AV node)

The spreading wave of depolarization is arrested before it can reach the ventricles by a plate of cartilage and fibrous material located at the AV junction. The plate provides structural support for the heart valves, but it also acts as an electrical insulator. By halting the wave, it allows time for the rapidly moving electrical events to be transduced into slower mechanical events and for blood to move from atria to ventricles.

C-His–Purkinje system

Once the wave of excitation migrates through the AV node, the ventricular walls must be stimulated to contract in a sequence that squeezes blood upwards toward the outlets: **septum** → **apex** → **free walls** → **base**. This is made possible using tracts of tissue comprising myocytes that are specialized to deliver the wave of depolarization at high speeds to the different regions of the ventricles.

ELECTROPHYSIOLOGY OF NORMAL CARDIAC RHYTHM

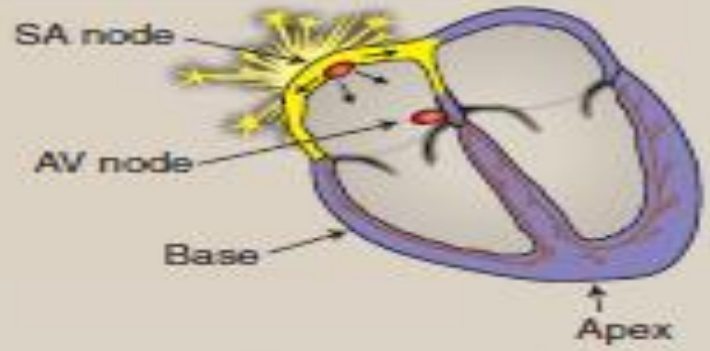
The electrical impulse that triggers a normal cardiac contraction originates at regular intervals in the sinoatrial (SA) node, usually at a frequency of 60–100 bpm.This impulse spreads rapidly through the atria and enters the atrioventricular (AV) node, which is normally the only conduction pathway between the atria and ventricles. Conduction through the AV node is slow, requiring about 0.15 seconds. (This delay provides time for atrial contraction to propel blood into the ventricles.)

The impulse then propagates over the His-Purkinje system and invades all parts of the ventricles, beginning with the endocardial surface near the apex and ending with the epicardial surface at the base of the heart. Ventricular activation is complete in less than 0.1 seconds; therefore, contraction of all of the ventricular muscle is normally synchronous and hemodynamically effective.

Arrhythmias consist of cardiac depolarizations that deviate from the above description in one or more aspects: there is an abnormality in the site of origin of the impulse, its rate or regularity, or its conduction.

The rapid pulse is referred to as *Tachycardia*, while the slow pulse is referred to as *Bradycardia*.

1 SA node fires. Wave of depolarization spreads over atria.



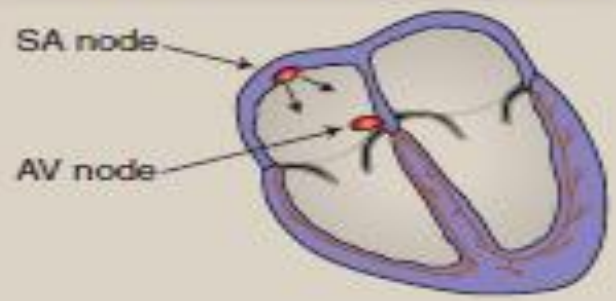
2 Atria are depolarized. AV node relays wave of excitation.

3 Purkinjes relay wave to septum. Septum depolarizes.

4 Apical region and ventricular free walls depolarize.

5 Base of ventricle depolarizes.

6 Atria and ventricles recover in the period between beats.



Overview of Heart Conduction

- The sequence of electrical events during one full contraction of the heart muscle:
- An excitation signal (an action potential) is created by the **sinoatrial (SA) node**.
- The wave of excitation spreads across the **atria,** causing them to contract.
- Upon reaching the **atrioventricular (AV) node**, the signal is delayed.
- It is then conducted into the **bundle of His**, down the interventricular septum.
- The bundle of His and the **Purkinje fibres** spread the wave impulses along the ventricles, causing them to contract.

Autonomic effects on heart rate and conduction velocity

a. Chronotropic effects : produce changes in heart rate.

- A **negative chronotropic effect** decreases heart rate by decreasing the firing rate of the SA node.
- A **positive chronotropic effect** increases heart rate by increasing the firing rate of the SA node.

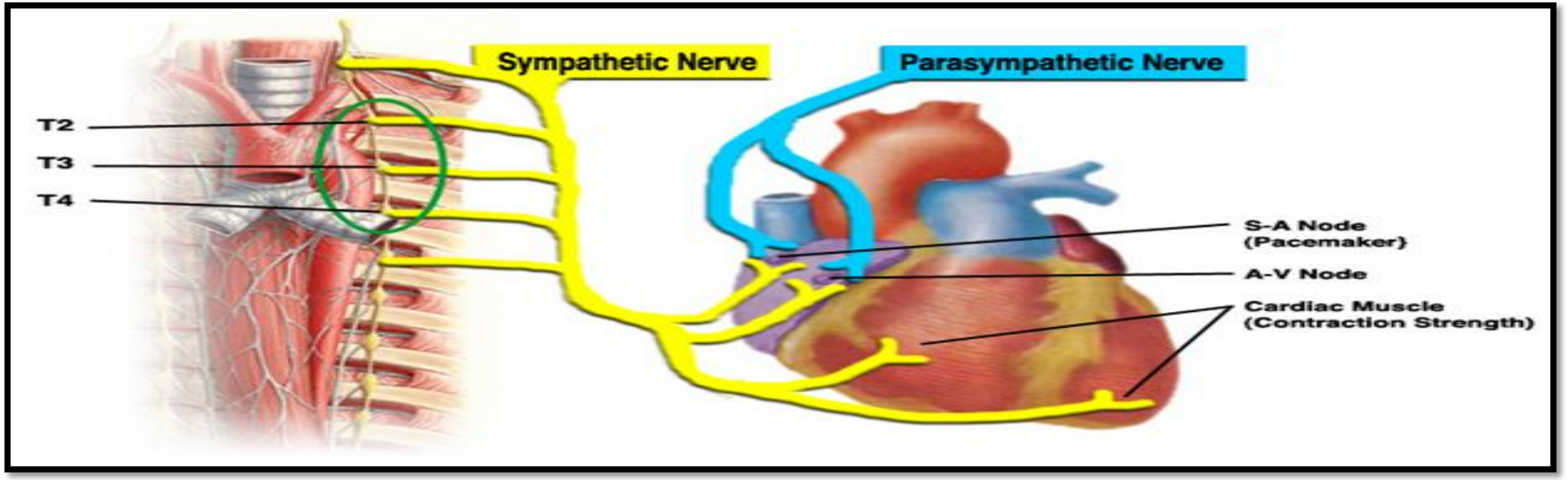
b. Dromotropic effects : produce changes in conduction velocity, primarily in the AV node.

- A **negative dromotropic effect** decreases conduction velocity through the AV node, slowing the conduction of action potentials from the atria to the ventricles.

- A **positive dromotropic effect** increases conduction velocity through the AV node, speeding the conduction of action potentials from the atria to the ventricles.

Neural regulation of the heart rate

- Sympathetic nerves: supply all parts of the heart (atria, ventricles, and conduction system). When activated they increase Heart rate (+ve chronotropic effect).
- Parasympathetic nerves: supply atria, SA & AV nodes but not the ventricles. When activated, they decrease Heart rate (-ve chronotropic effect).



Contractibility

is also called **inotropism**

Positive inotropic agents produce an increase in contractility.

Negative inotropic agents produce a decrease in contractility.

table 3.1 Autonomic Effects on the Heart and Blood Vessels

	Sympathetic		Parasympathetic	
	Effect	*Receptor*	*Effect*	*Receptor*
Heart rate	↑	β_1	↓	Muscarinic
Conduction velocity (AV node)	↑	β_1	↓	Muscarinic
Contractility	↑	β_1	↓ (Atria only)	Muscarinic
Vascular smooth muscle				
Skin, splanchnic	Constriction	α_1		
Skeletal muscle	Constriction	α_1		
	Relaxation	β_2		

AV = atrioventricular.

Cardiac Electrophysiology

A. Electrocardiogram (ECG)

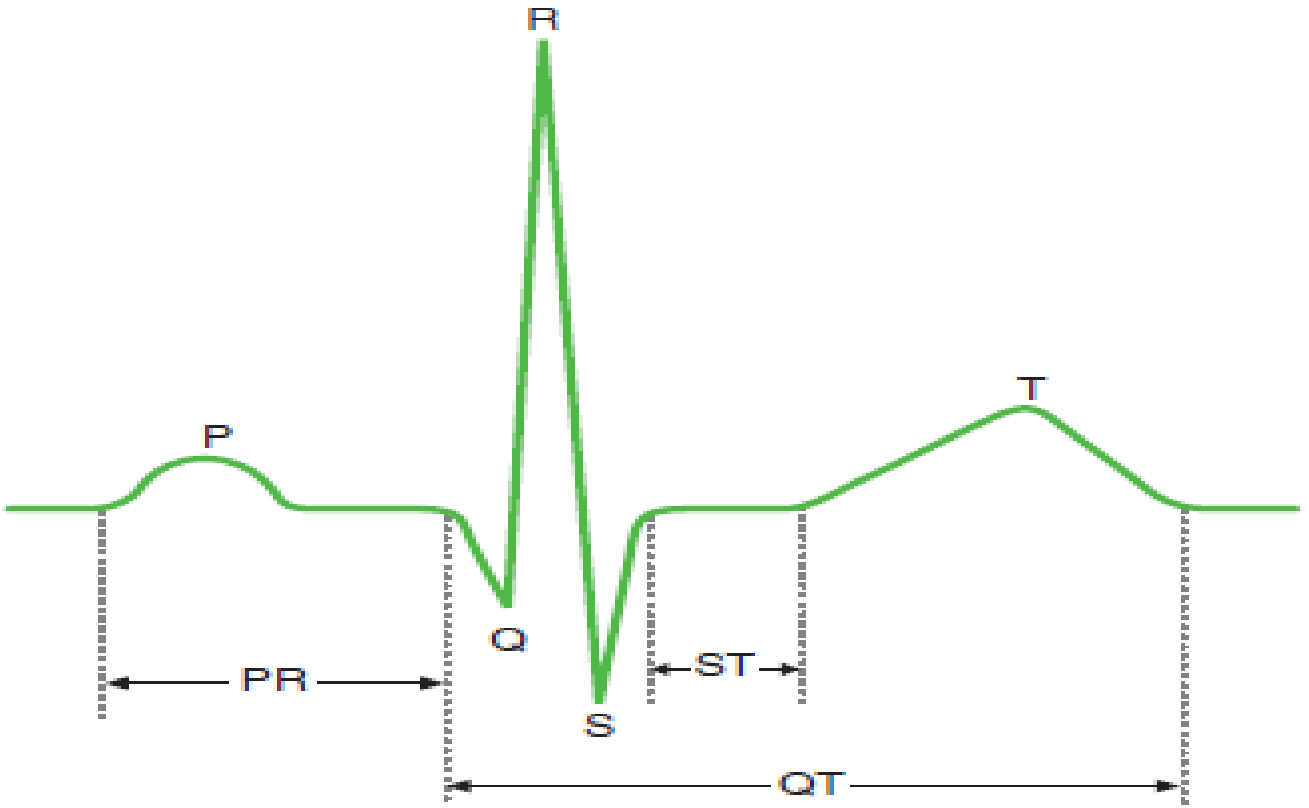
1. P wave represents atrial depolarization.

2. QRS complex represents depolarization of the ventricles.

3. QT interval is the interval from the beginning of the Q wave to the end of the T wave, represents the entire period of depolarization and repolarization of the ventricles.

4. ST segment is the segment from the end of the S wave to the beginning of the T wave, is isoelectric, represents the period when the ventricles are depolarized.

5. T wave represents ventricular repolarization.



CONDUCTING SYSTEM DEFECTS

- The defect/damage of conducting system causes cardiac **arrhythmias**.
- If the AV bundle fails to conduct normal impulses, there takes place alteration in the rhythmic contraction of the ventricles (arrhythmias). If complete bundle block takes place there is complete dissociation in the rate of contraction of atria and ventricles. The common cause of defective conduction via AV bundle is **atherosclerosis** of the coronary arteries which leads to diminished blood supply to the conducting system.
- The rapid pulse is referred to as **tachycardia**, the slow pulse is named **bradycardia** on the other hand irregular pulse is named arrhythmia.

Cardiac output

- The pumping ability of the heart is a function of the beats per minute HR **(heart rate)** and the volume of blood ejected per beat **SV (stroke volume).**
- The amount of blood pumped/minute by each ventricle, expressed in liters/minute. Normally, it is about 5 liters per minute.
- Determined through multiplying the heart rate (HR) by the stroke volume (SV).

CO = HR X SV

Control of cardiac output:

- Venous return (preload).
- Afterload.
- Heart rate (HR)
- Myocardial contractility.

Cardiac Output (CO)

volume of blood pumped by each ventricle per minute.

Average value: 5 liters/minute = Total blood volume 5 Liters.

Cardiac output increases in the following conditions:

1- Physical exercise (up to 700%).
2- Anxiety and excitement (up to 100%).

3- After meals (30%).
4- High environmental temperature.

5- Pregnancy.

Cardiac output decreases in the following conditions:

1- Standing from supine position (30%).

2- Rapid cardiac arrhythmias.

Control of cardiac output:

- **Venous return (preload)** : It is the amount of the blood flowing into the heart via the veins (right or left atrium) each minute (preload).
- VR and CO are equal (per minute).

  CO = VR

- Dependent on:
  - Blood volume & venous pressure.
  - Vasoconstriction (Sympathetic).
  - Skeletal muscle pumps.
  - Pressure drop during inhalation.

Control of CO by Venous return

- *Frank-Starling law;* the heart pumps automatically whatever amount of blood flows into the right atrium from the veins.
- The Frank-Starling Law is the description of cardiac hemodynamics as it relates to myocyte stretch and contractility.

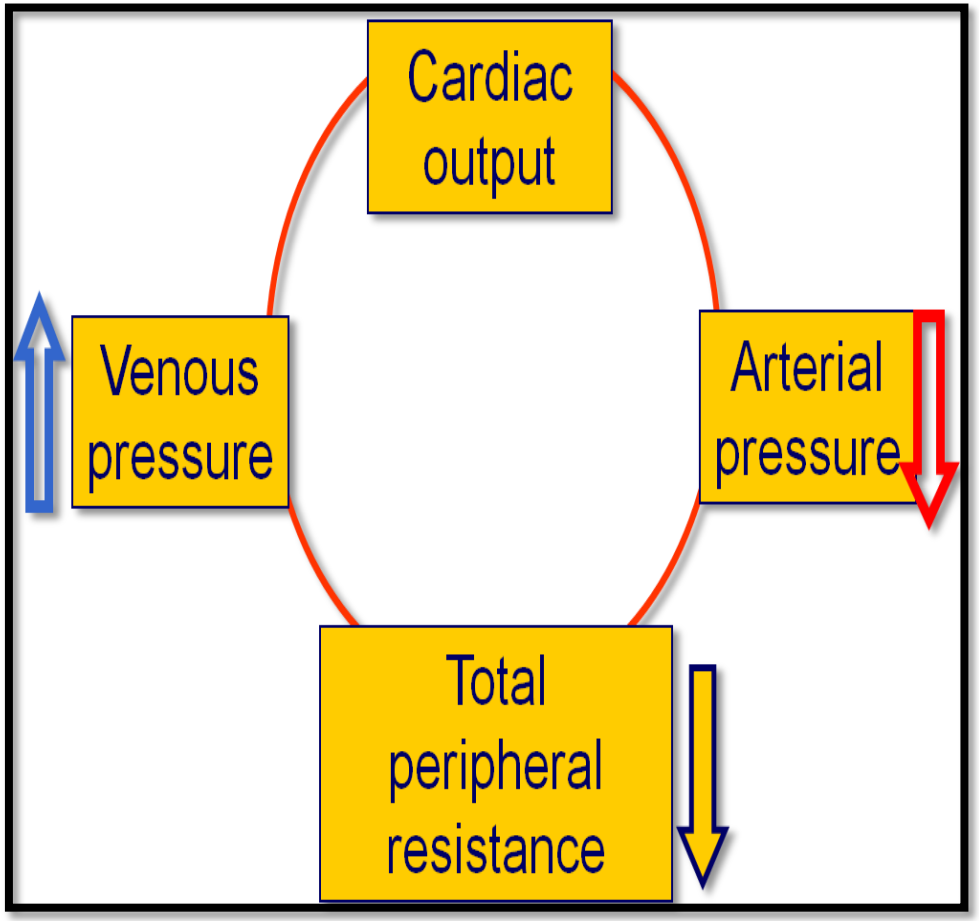
- The Frank-Starling Law states that the stroke volume of the left ventricle will increase as the left ventricular volume increases due to the myocyte stretch causing a more forceful systolic contraction. This assumes that other factors remain constant

- *heart rate;* (stretching of the heart), Stretch of the SA node in the wall of the right atrium has a direct effect on the rhythmicity of the SA node itself to increase heart rate 10 – 15%.

- *Bainbridge reflex;* (stretch of right atrium) vasomotor center → sympathetic nerves → SA node → increases of heart rate.

Afterload & COP

- It is the resistance that oppose cardiac output, e.g., increased arterial systolic pressure.
- increased afterload will reduce cardiac output.
- reduced afterload (reduced total peripheral resistance) causes high cardiac output.



Effect of changes in heart rate & stroke volume on cardiac output

CO = SV X HR

- In resting state = constant VR
- During muscular exercise = BOTH HR and SV increase

In resting state, (the venous return is constant), changes in heart rate between 100-200 beats/min., not affect CO markedly.

High heart rate (more than 200 beats/minute) may affect CO to be insufficient (duration of ventricular diastole).

Slow heart rate may also reduce CO (complete heart block disease (HR < 40 beats/minute)).

In exercise, (the venous return is increased), cardiac output is increased by increasing in both heart rate and stroke volume.

Myocardial Contractility

- defined as the strength of contraction at any given EDV.
- It is measured by Ejection Fraction (EF%).

Myocardial contractility affected by:

- The preload (i.e., EDV): Frank-Starling's law.
- The afterload (i.e., aortic impedance): An increase in the afterload reduces the cardiac pumping power, and vice versa.
- Sympathetic nerve supply.
- Ventricular hypertrophy; (athletes) can increase the cardiac pumping power up to about 35 litres/ minute.

End-Diastolic Volume, End-Systolic Volume, and Stroke Volume Output.

During diastole, normal filling of the ventricles increases the volume of each ventricle to about 110 to 120 milliliters. This volume is called the ***end diastolic volume***.

Then, as the ventricles empty during systole, the volume decreases about 70 milliliters, which is called the ***stroke volume output***. The remaining volume in each ventricle, about 40 to 50 milliliters, is called the ***endsystolic volume***.

The fraction of the end-diastolic volume that is ejected is called the ***ejection fraction***— usually equal to about 0.6 (or 60 percent).


Thank you.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