Lecture 23. Pumping function of the heart, its role in hemodynamics, physiological bases of research methods. The regulation of the heart activity

Lecture Outline

- Cardiovascular System Function
- Functional Anatomy of the Heart
- Myocardial Physiology
  - Autorhythmic Cells (Pacemaker cells)
  - Contractile cells
- **Cardiac Cycle**
- Cardiac Output Controls & Blood Pressure

Cardiac Cycle

Coordinating the activity

- Cardiac cycle is the sequence of events as blood enters the atria, leaves the ventricles and then starts over
- Synchronizing this is the Intrinsic Electrical Conduction System
- Influencing the rate (chronotropy & dromotropy) is done by the sympathetic and parasympathetic divisions of the ANS

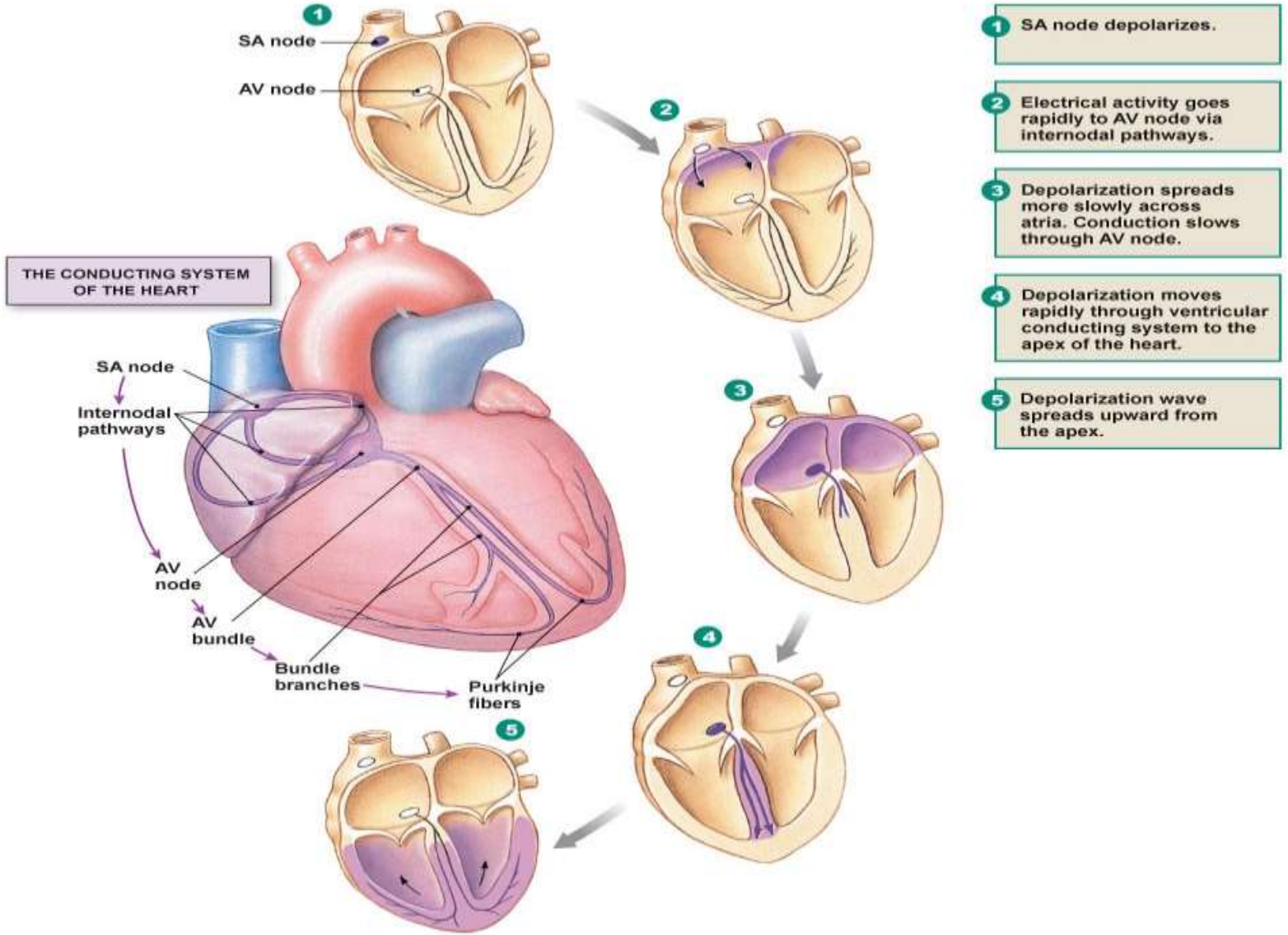
Cardiac Cycle

Coordinating the activity

- Electrical Conduction Pathway
  - Initiated by the Sino-Atrial node (SA node) which is myogenic at 70-80 action potentials/minute
  - Depolarization is spread through the atria via gap junctions and internodal pathways to the Atrio-Ventricular node (AV node)
    - The fibrous connective tissue matrix of the heart prevents further spread of APs to the ventricles
    - A slight delay at the AV node occurs
      - Due to slower formation of action potentials
      - Allows further emptying of the atria
  - Action potentials travel down the Atrioventricular bundle (Bundle of His) which splits into left and right atrioventricular bundles (bundle branches) and then into the conduction myofibers (Purkinje cells)
    - Purkinje cells are larger in diameter & conduct impulse very rapidly
      - Causes the cells at the apex to contract nearly simultaneously
        - » Good for ventricular ejection

Cardiac Cycle

Electrical Conduction Pathway



Cardiac Cycle

Phases

- Systole = period of contraction
- Diastole = period of relaxation
- Cardiac Cycle is alternating periods of systole and diastole
- Phases of the cardiac cycle
  1. Rest
     - Both atria and ventricles in diastole
     - Blood is filling both atria and ventricles due to low pressure conditions
  2. Atrial Systole
     - Completes ventricular filling
  3. Isovolumetric Ventricular Contraction
     - Increased pressure in the ventricles causes the AV valves to close… why?
       - Creates the first heart sound (lub)
     - Atria go back to diastole
     - No blood flow as semilunar valves are closed as well

Cardiac Cycle

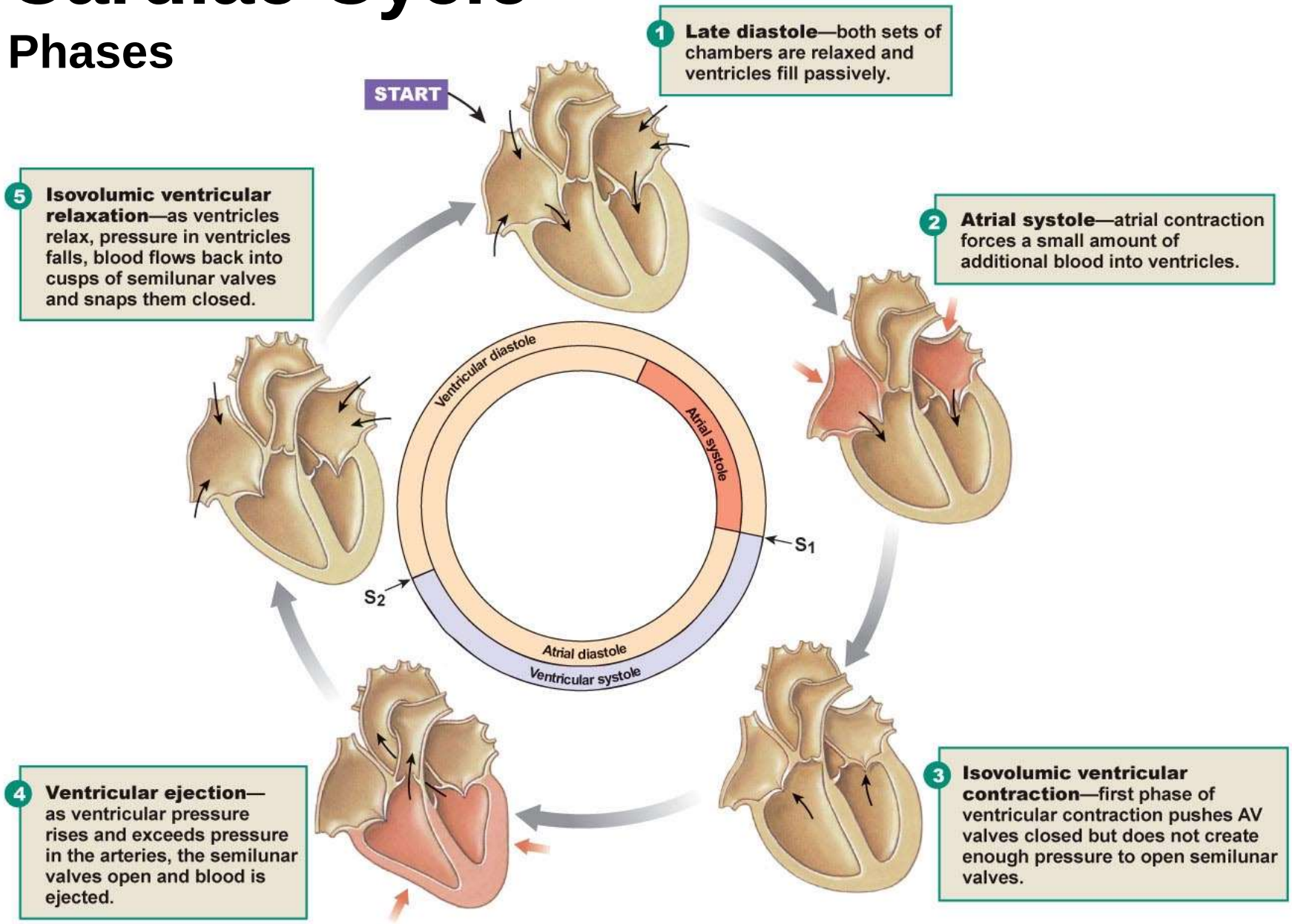
Phases

- Phases of the cardiac cycle
  4. Ventricular Ejection
     - Intraventricular pressure overcomes aortic pressure
       - Semilunar valves open
       - Blood is ejected
  5. Isovolumetric Ventricular Relaxation
     - Intraventricular pressure drops below aortic pressure
       - Semilunar valves close = second heart sound (dup)
     - Pressure still hasn't dropped enough to open AV valves so volume remains same (isovolumetric)

  Back to Atrial & Ventricular Diastole

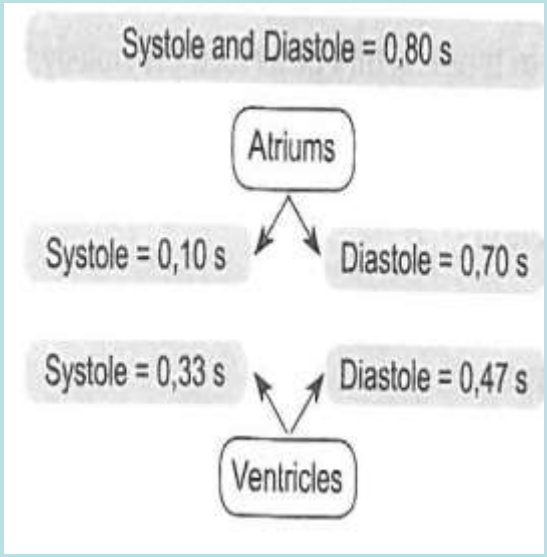
Cardiac Cycle

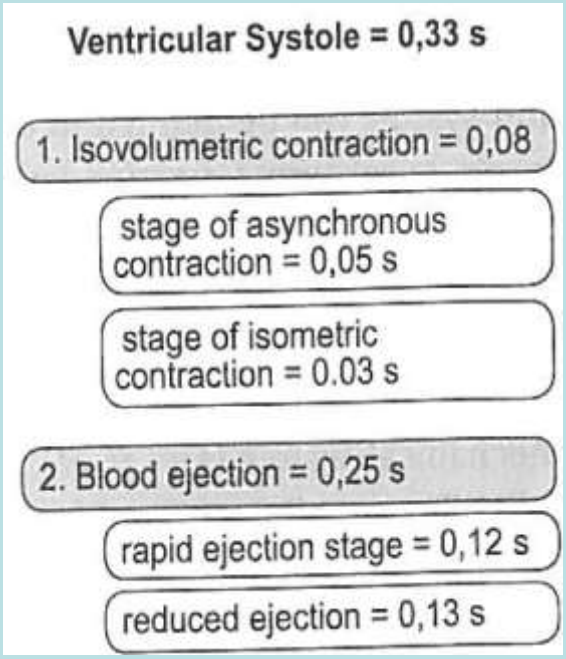
Phases

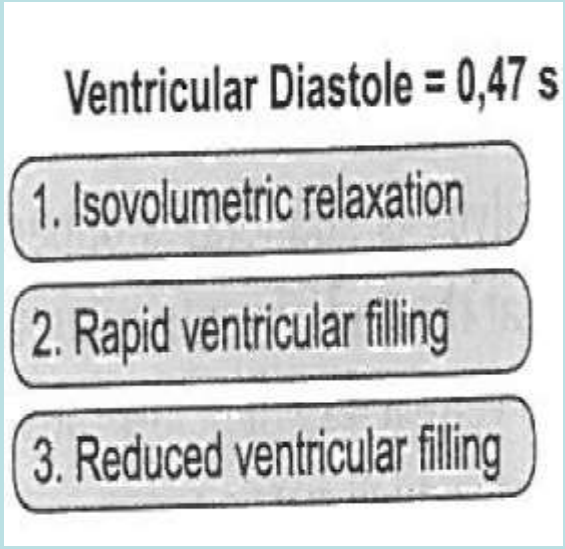


Cardiac Cycle (0,8 s)

- **The cardiac cycle is systole and diastole of all the heart' parts**. The duration of systole and diastole, and their phases, given here, are mean values encountered with a cardiac rate of 75 beats per minute. To know the C.C. duration is necessary **60 s/RCC.**



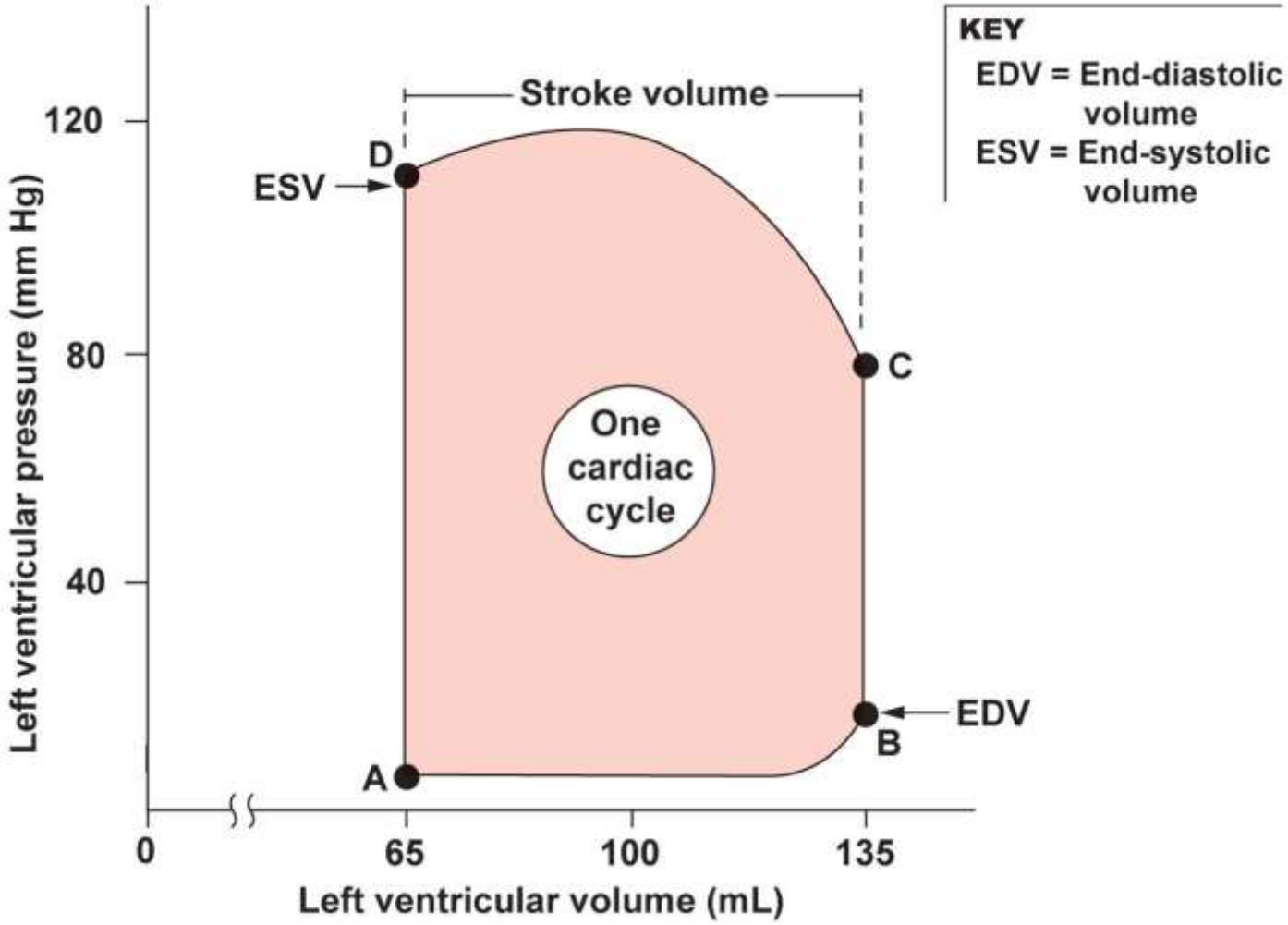




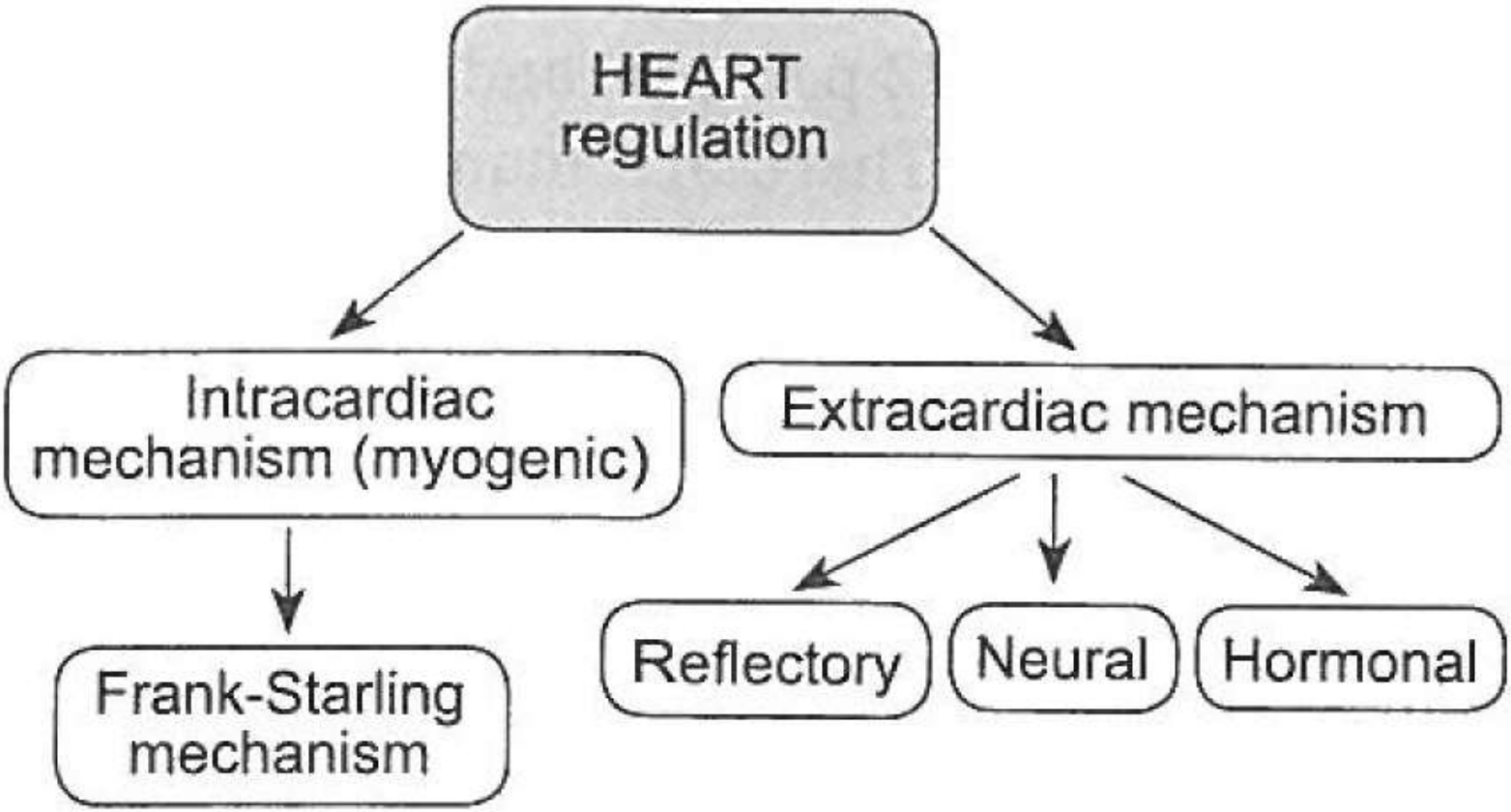
N	Phases of cardiac cycle	Duration 0,8 sec	State of valves		Sounds
			AV	Aortic, pulmonary	
1	Atrial systole	0,1	open	closed	S_4
2	Ventricles systole	0,33			
2.1.	Tension	0,08			
2.1.1.	asynchronous contraction	0,05	open	closed	
2.1.2.	isometric contraction	0,03	closed(at beginning)	closed	S_I
2.2.	Blood ejection	0,25			
2.2.1	rapid ejection	0,12	closed	open (at beginning)	
2.2.2.	reduced ejection	0,13	closed	open	
3.	Ventricular diastole	0,47			
3.1.	Protodiastolic period	0,04	closed	Closed (at the end)	S_2
3.2.	Isovolumetric relaxation	.0,08	closed	closed	
3.3.	Ventricular filling	0,25			
3.3.1	rapid	0,08	open (at beginning)	closed	S_3
3.3.2	reduced	0,17	open	closed	
	Presystolic=atrial systole	0,1	open	closed	

Cardiac Cycle

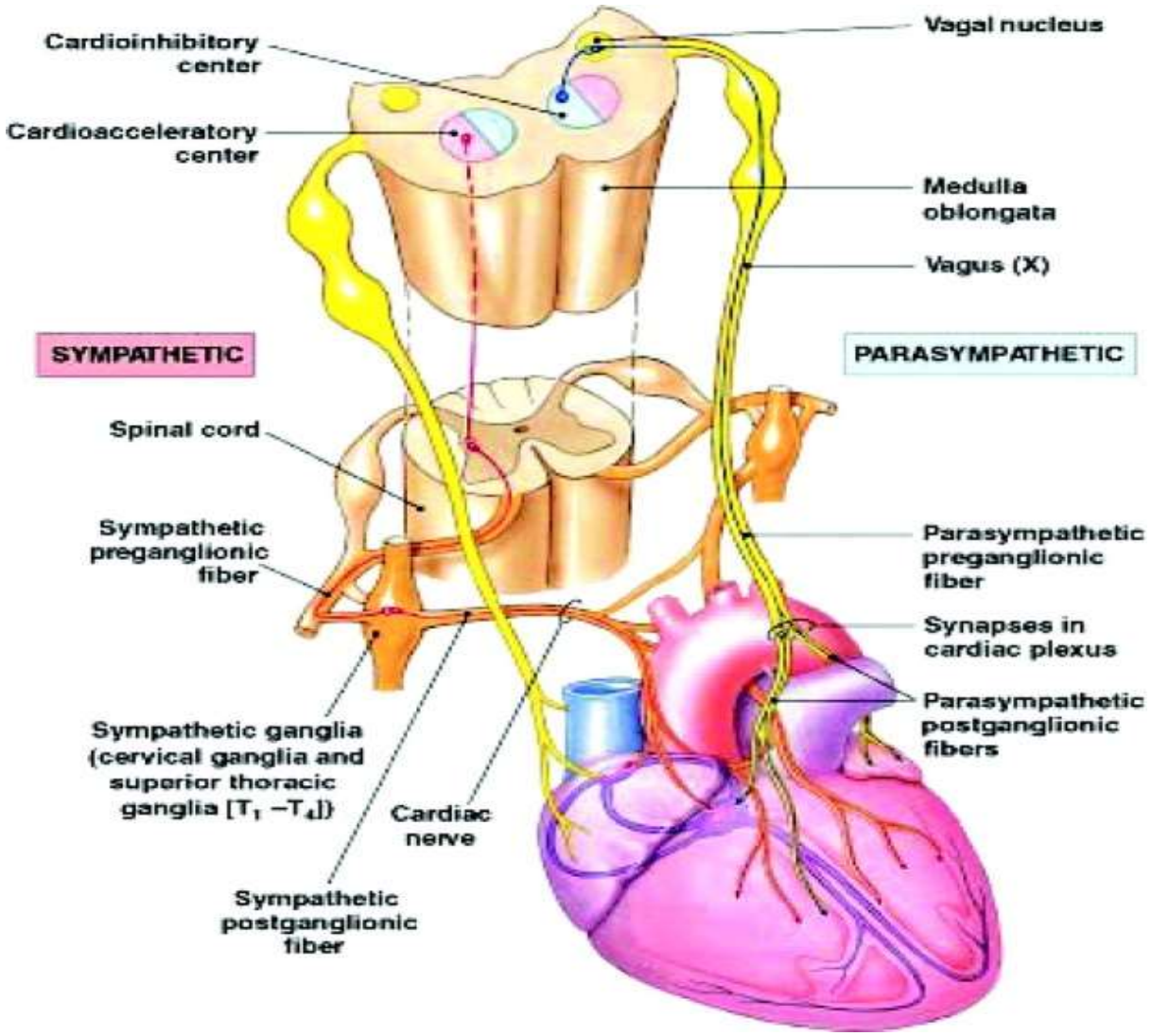
Blood Volumes & Pressure



Regulation of heart activity



Innervation of the heart



Heterometric regulation

- **Myogenic regulatory** mechanisms of the heart (Heterometric regulation) based on the special properties typical of cardiomyocytes. Their implementation leads to a variation of heart rate (stroke volume)
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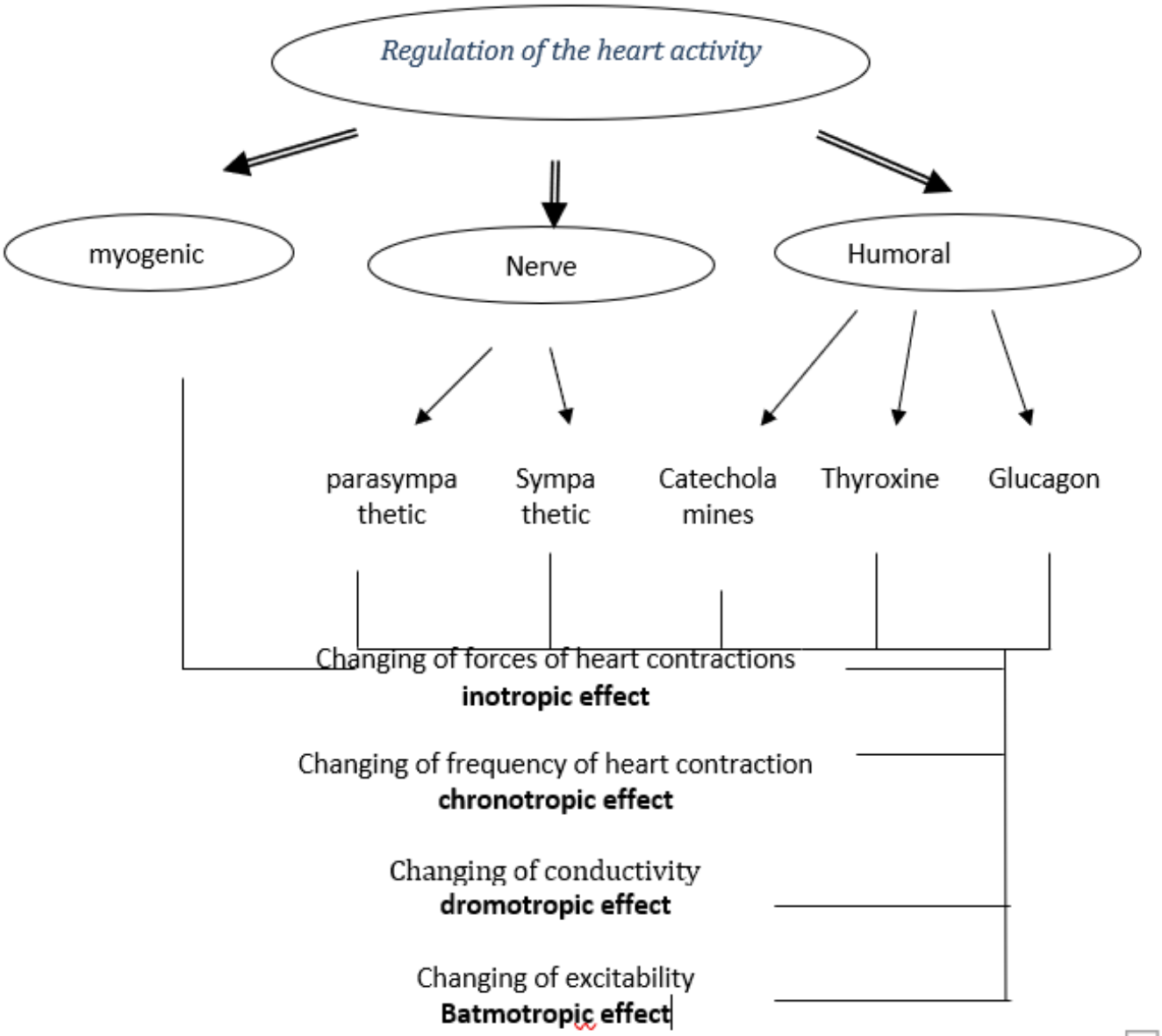
1. **Mechanism Frank - Starling** (the law of the heart) is to increase the strength of heart contractions (and stroke volume) with increasing initial length cardiomyocytes. Since the initial length of cardiomyocytes depends on the volume of blood flowing to the cavities of the heart during diastole (venous return), Frank-Starling mechanism provides the same amount of venous return of blood to the heart and systolic volume. This mechanism ensures the adaptation of the heart to changes in venous return of blood to the heart during diastole.
2. **Anrepa effect** is to increase the strength of heart contractions with increasing resistance captivity (blood pressure). If blood pressure is increase systolic volume (and cardiac output) is not reduced. Effect Anrepa provides adaptation of the heart to change the outflow of blood from the ventricles of the heart.

- 3. **Effect Boudycha** (hronoinotropna dependence) is to increase the strength of heart contractions at a significant increase in the frequency of contractions. This systolic volume does not decrease, despite the reduction in diastole. Thus, cardiac output increases in proportion to an increase in frequency. The man hronoinotropnyy mechanism is effective in increasing the heart rate to 180-200 times per minute. The increase in frequency over these values accompanied by a

Homometric regulation



Myocardial Physiology

Autorhythmic Cells (Pacemaker Cells)

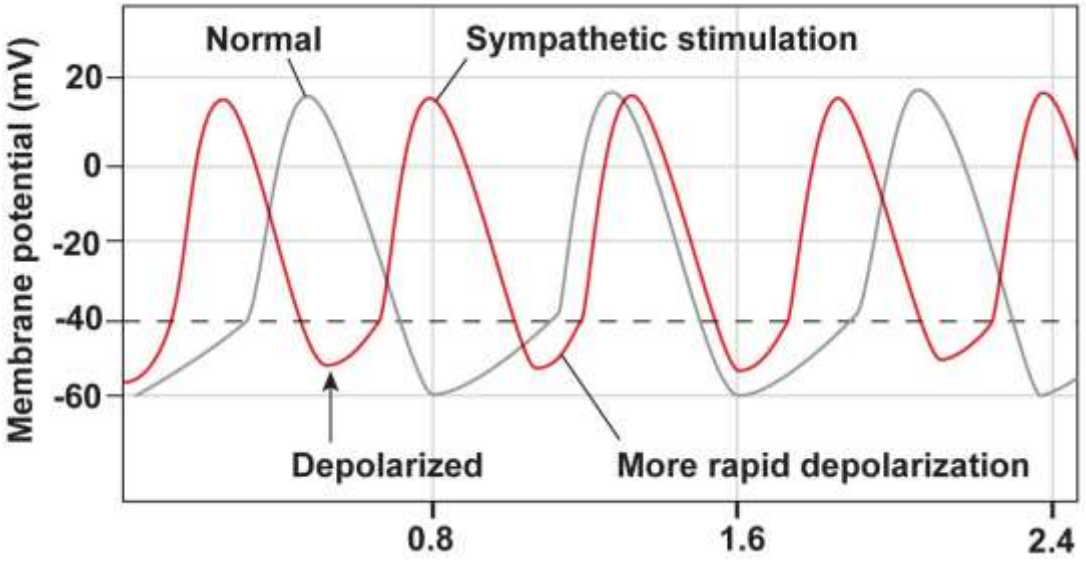
- Altering Activity of Pacemaker Cells
  - Sympathetic activity
    - NE and E increase I_f channel activity
      - Binds to β_1 adrenergic receptors which activate cAMP and increase I_f channel open time
      - Causes more rapid pacemaker potential and faster rate of action potentials

Sympathetic Activity Summary:

increased chronotropic effects
↑heart rate

increased dromotropic effects
↑conduction of APs

increased inotropic effects
↑contractility



Myocardial Physiology

Autorhythmic Cells (Pacemaker Cells)

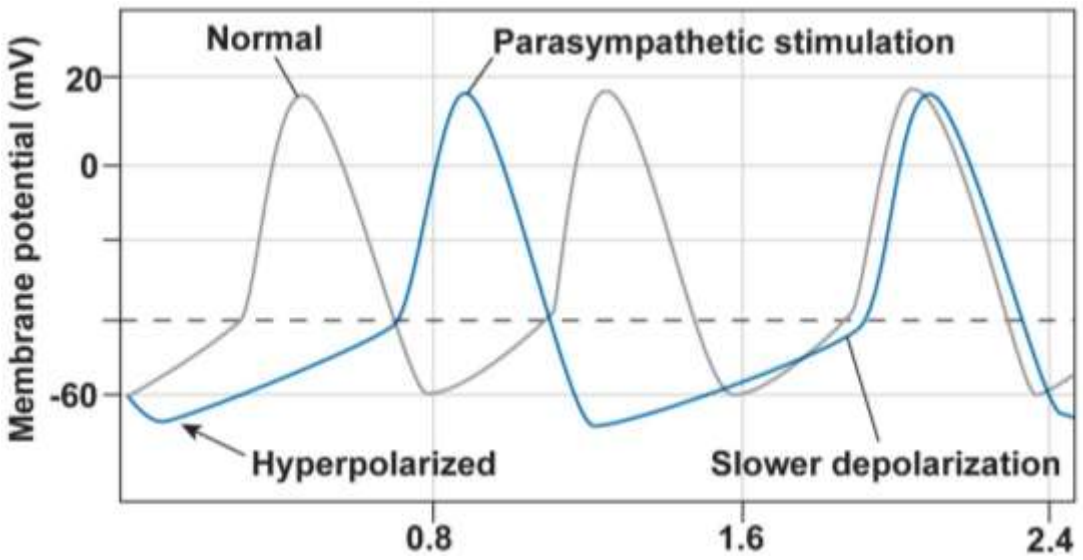
- Altering Activity of Pacemaker Cells
  - Parasympathetic activity
    - ACh binds to muscarinic receptors
      - Increases K^+ permeability and decreases Ca^{2+} permeability = hyperpolarizing the membrane
        - » Longer time to threshold = slower rate of action potentials

<u>Parasympathetic Activity Summary:</u>

decreased chronotropic effects
↓heart rate

decreased dromotropic effects
↓ conduction of APs

decreased inotropic effects
↓ contractility



Effect of changes in the concentration of ions in the blood plasma on heart activities

• The practical significance is the change in plasma concentrations of two ions, calcium and potassium.

• **Calcium ions**. Changing their concentration in plasma affects both the strength and the heart rate. For example, if a patient intravenously administered drugs containing calcium ions (calcium chloride, calcium gluconate), this leads to a rapid increase in their concentration in plasma and in the extracellular fluid. This increases the concentration gradient of calcium ions between the extracellular fluid and cytoplasm of cardiomyocytes. And when the excitement in their cardiomyocytes membrane calcium channels are opened, the entrance of calcium into the cytoplasm increases.

• - Increased entry of calcium ions to atypical cardiomyocytes Sino-atrialnoho node increases the rate of slow diastolic depolarization: pacemaker generates more action potentials and heart rate increases.

• - Increased entry of calcium ions to typical atrial and ventricular cardiomyocytes during phase "plateau" of AP leads to increased calcium volley (release of calcium from SPR); more calcium ions interacting with regulatory proteins called actin centers,

• **Potassium ions**. Hypokalemia develops most often with insufficient flow of potassium ions in the body or in their great loss. Because hypokalemia is accompanied by a decrease in content of potassium ions in the cytoplasm. Violation electrophysiological characteristics of cardiac myocytes in this case may be different. Impaired function of the heart is to develop arrhythmia (disturbance of the normal heart rate), tachycardia, there are typical changes in the ECG, cardiac arrest may occur.

• **High concentration of potassium ions**. Always leads to a decrease in the concentration gradient of potassium ions in the cytoplasm of cells and in the intercellular fluid, potassium ions out of the cell decreases, which leads to sustainable long membrane depolarization of excitable cells, including cardiomyocytes. Thus there are conditions for the manifestation of latent pacemakers activity (centers automaticity second, third order). After prolonged depolarization cardiomyocytes leads to atrial fibrillation and cardiac arrest in diastole.