CVS MODULE
PHYSIOLOGY (LECTURE 1)
Physiology of Cardiac Muscle I

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CARDIOVASCULAR SYSTEM

- The **cardiovascular (circulatory) system; CVS consists of the cardiac muscle (heart), blood vessels and blood.**
- It is a **closed system of vessels** inside which blood circulates continuously by the **pumping action** of the **heart** in **one direction** only by the action of **valves** present in the **heart** and **veins**.

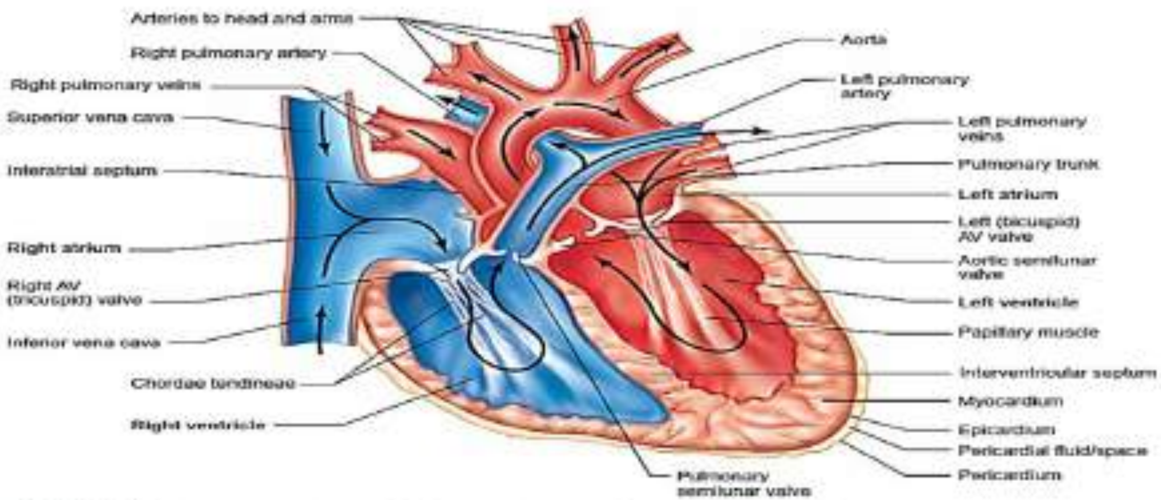


Figure 12.6 APR Diagrammatic section of the heart. The arrows indicate the direction of blood flow.

Functions of CVS

- The main functions of the cardiovascular system are:

gas transport, nutrient delivery, and waste removal.

- These functions are dependent on circulation of blood, which requires structural integrity of the heart and vessels.
- The cardiovascular system also is involved in several homeostatic functions:

It participates in the regulation of arterial blood pressure; it delivers regulatory hormones from the endocrine glands to their sites of action in target tissues; it participates in the regulation of body temperature; and it is involved in the homeostatic adjustments to altered physiologic states such as hemorrhage, and exercise.

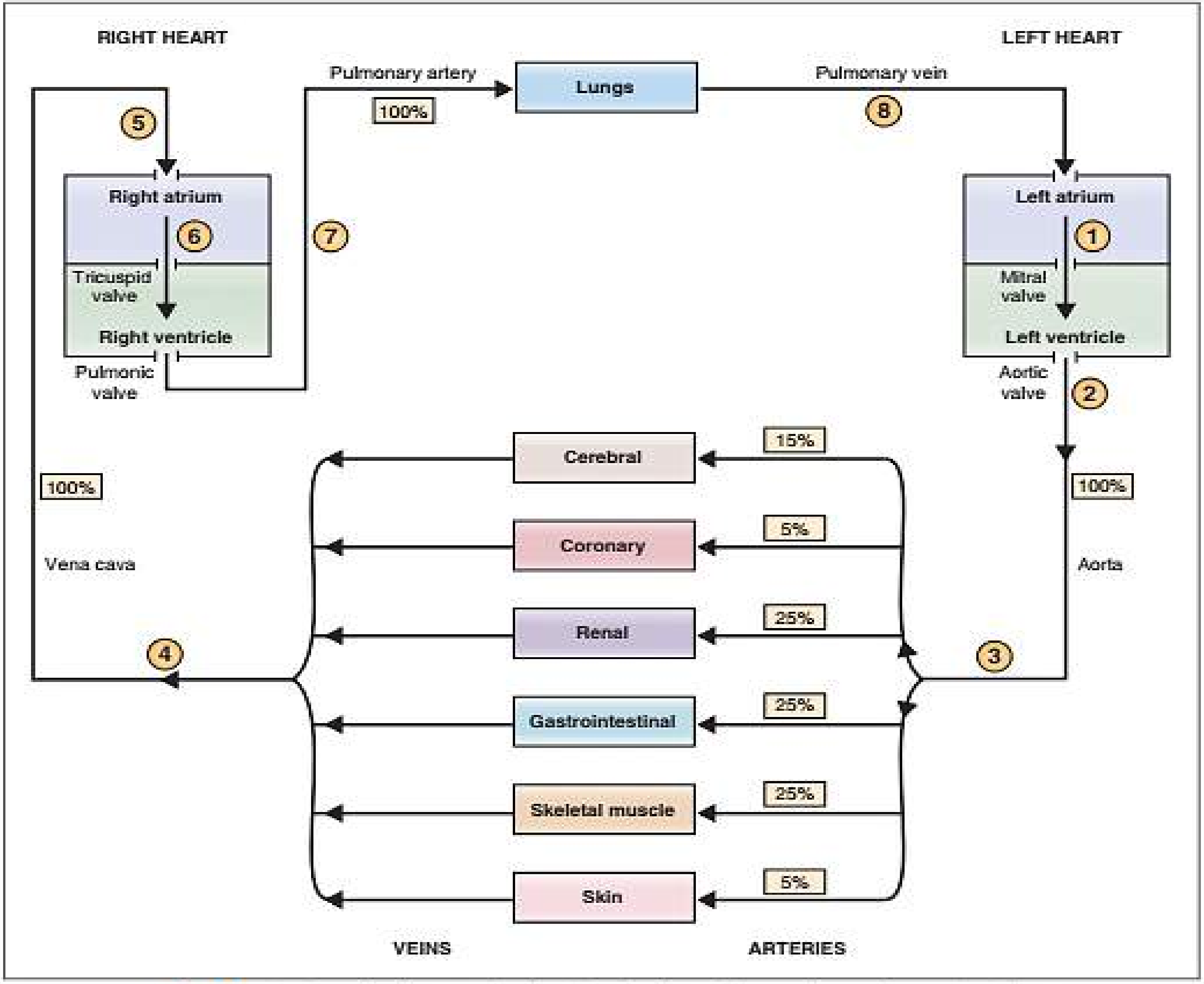


Fig. 4.1 A schematic diagram showing the circuitry of the cardiovascular system. The *arrows* show the direction of blood flow. Percentages represent the percent (%) of cardiac output.

CARDIAC MUSCLE

- Cardiac muscle is a **striated muscle** like the skeletal muscle, but it is **different** from the skeletal muscle in being **involuntary** and **syncytial**.

- **Syncytium:** It means that **cardiac muscle cells are able to excite and contract together** as one unit **due to** the presence of **gap junctions** between adjacent cardiac cells.

- **Both atria** contract together as one unit (**upper syncytium**) and **both ventricles** contract together as one unit (i.e. **lower syncytium**), which are **completely separated** from each other by the **fibrous A-V ring**. So, the excitation waves **cannot be directly transmitted** from one syncytium to the other.

Types of Cardiac Muscle cells

Myocardium of the heart is composed of two types of cardiac muscle cells (fibers):

A. Contractile Cells :

- Form about 98-99% of the cardiac muscle.
- Their action potential (AP) is called **fast AP**.

B. Non-contractile (auto-rhythmic) Cells:

- Form about 1-2 % of the cardiac muscle and are the cells that form **excitatory- conductive system** of the heart.
- Their AP is called **slow or pacemaker AP.**

Functions of the atria and ventricles

Atria:

- They are the entry-ways to the ventricles: they receive and store the venous return (VR) then pass it to the ventricles during ventricular diastole.
- The atrial walls contain stretch receptors the monitor changes in the intra-atrial pressure.
- Secret the atrial natriuretic peptide (ANP) which favors Na^+ and water excretion by the kidneys and also causes VD.

Ventricles:

- Chambers whose contractions produce the pressures that drive blood through the pulmonary and systemic vascular systems and back to the heart.
- The pumping action of ventricles is the main force that propels blood to the peripheral circulatory system, thus loss of such function is fatal.

Physiological Properties of the Cardiac Muscle

Cardiac muscle has four properties, due to which the heart is able to fulfill its function as a pumping organ.

They include:

1. **Automaticity & Rhythmicity (Chronotropism).**
2. **Excitability (Bathmotropism).**
3. **Conductivity (Dromotropism).**
4. **Contractility (Inotropism).**

1. Automaticity and Rhythmicity (Auto-rhythmicity)

✓ **Automaticity:**

It is the property of self-excitation; the ability of spontaneous generation of action potentials independent of any extrinsic stimuli.

✓ **Rhythmicity:**

The regular generation of these action potentials (the heart can beat regularly).

Spontaneous automaticity and **rhythmicity (auto-rhythmicity)** of the cardiac muscle is due to the existence of a specialized excitatory-conductive system, which is composed of modified **self-exciting**, **non- contractile** cardiac muscle cells called pacemaker cells.

Auto-rhythmicity is **myogenic** in origin (i.e. starts from the **muscle** itself **independent** from autonomic nerve supply that **only controls** the heart rate (either ↑ or ↓) **but don't initiate the beat.**

Pacemaker(s) of the heart:

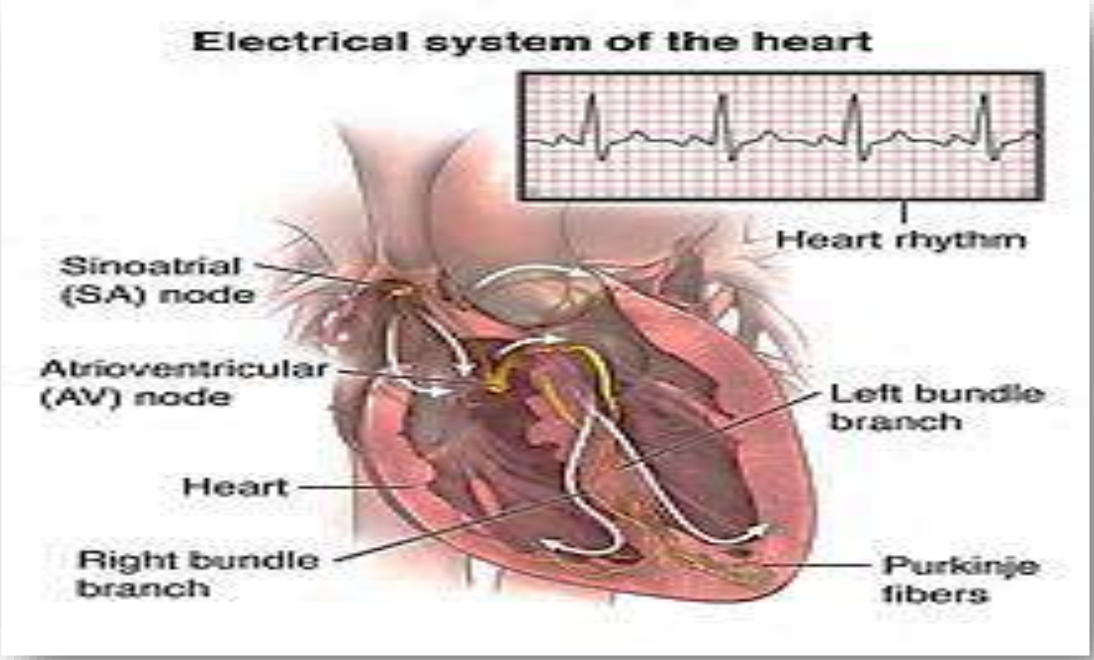
Pacemaker means the part of the heart that has the highest rhythmicity and the whole other parts of the heart follow its rhythm.

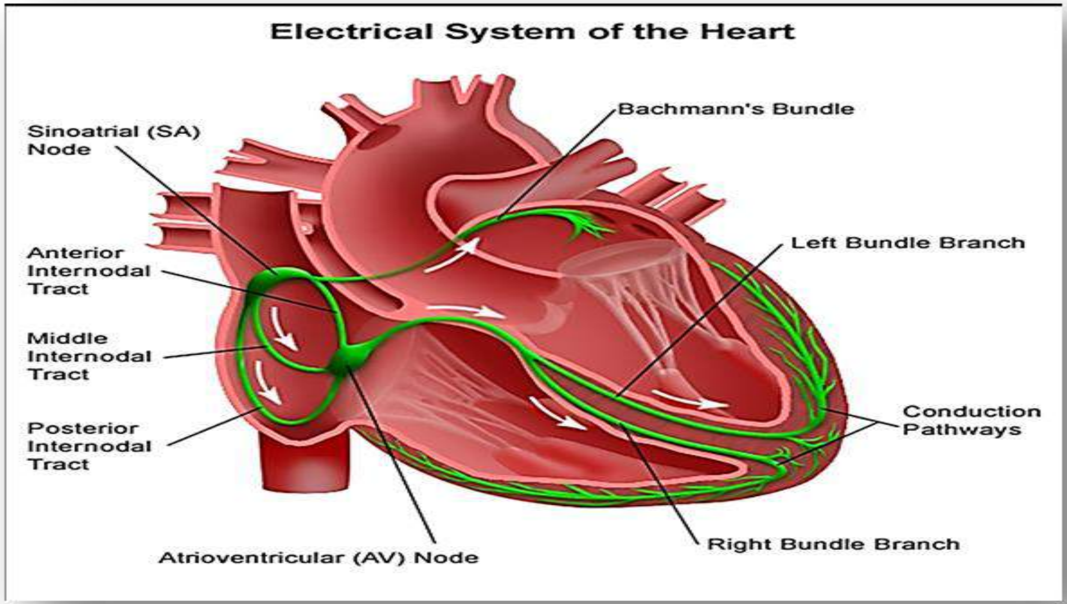
They include:

1. **Sinoatrial (auricular) (SA) Node (1ry; normal Pacemaker):**
- It has the **highest** rhythm **(90-110; 100/minute)**.
- So, it is called the **normal** or **1ry pacemaker** of the heart.
- Its rhythm is called **sinus rhythm**.

2. **Atrioventricular (AV) Node (2ry Pacemaker):**
- Its rate is **45-60 /minute.**
- It acts **only** if **SA node is damaged or blocked.**
- Its rhythm is called **nodal rhythm**.

3. **Purkinje fibres (3ry Pacemaker):**
- Its rate is **25-40 /minute.**
- It takes over **only** if the conduction in **AV node is completely blocked**.
- Its rhythm is called **idioventricular rhythm**.


Electrical system of the heart
Heart rhythm
Sinoatrial (SA) node
Atrioventricular (AV) node
Left bundle branch
Heart
Right bundle branch
Purkinje fibers


Electrical System of the Heart
Bachmann's Bundle
Sinoatrial (SA) Node
Anterior Internodal Tract
Left Bundle Branch
Middle Internodal Tract
Posterior Internodal Tract
Conduction Pathways
Right Bundle Branch
Atrioventricular (AV) Node

Mechanism of Autorhythmicity (Prepotential & Pacemaker AP)

- ✓ Pacemaker cells in the nodal tissue (SA node and AV node) have a resting membrane potential of about -60 mV. However, it is unstable.
- ✓ After each impulse, gradual depolarization occurs spontaneously till a firing level (threshold potential) is reached at which action potential (an impulse) is initiated.
- ✓ They can spontaneously and regularly initiate action potential and so they are responsible for automaticity and rhythmicity of the heart.
- ✓ This gradual depolarization is called prepotential or diastolic depolarization.

Phases of Pacemaker Potential

<u>Pacemaker action potential is composed of:</u>

<u>Phase 4; (Prepotential):</u>

- ✓ Resting membrane potential (RMP) is about - 60 mV **(unstable)**.
- ✓ At this potential, there is activation of a special type of Na^+ channels known as **Na^+ funny channels** → funny current; F (inward Na^+).
- ✓ Another type of channels called **transient** or **<u>T-type</u> Ca^{++} channels opens**→ Ca^{++} enters down its electrochemical gradient → depolarizes the cell to **- 40 mV**.

Phase 0; (Depolarization):

- As the membrane is depolarized to the **firing level; threshold potential** **(about - 40 mV)** another type of Ca^{++} channels opens. These are known as **long-lasting or L-type Ca^{++} channels** → entrance of Ca^{++}.

- Because the movement of Ca^{++} through these channels into the cell is not rapid, the rate of depolarization (slope of phase 0) is much slower than found in other types of cardiac cells. Therefore AP of pacemaker cells is called **slow response action potential.**

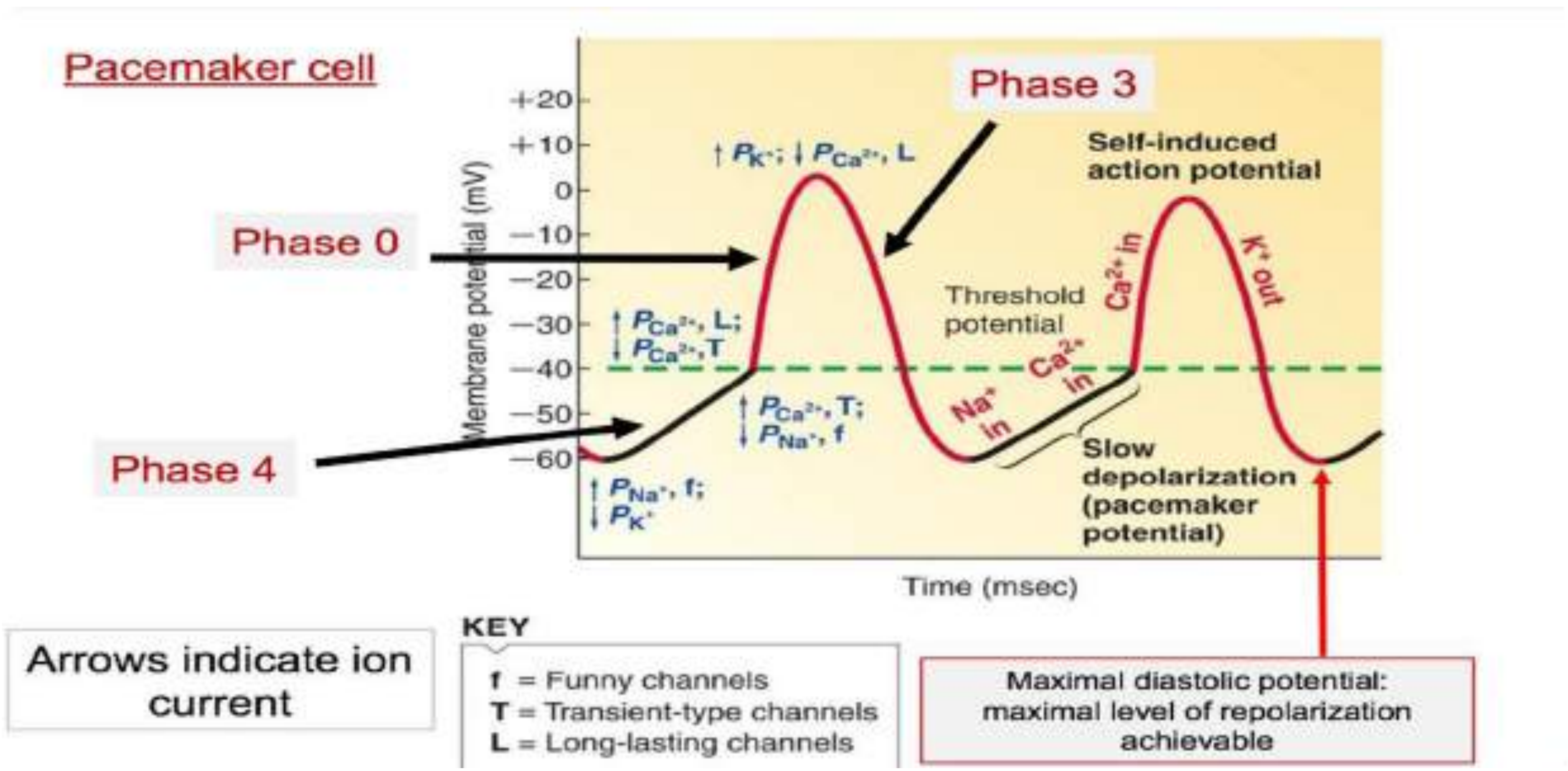
Phase 3; (Repolarization):

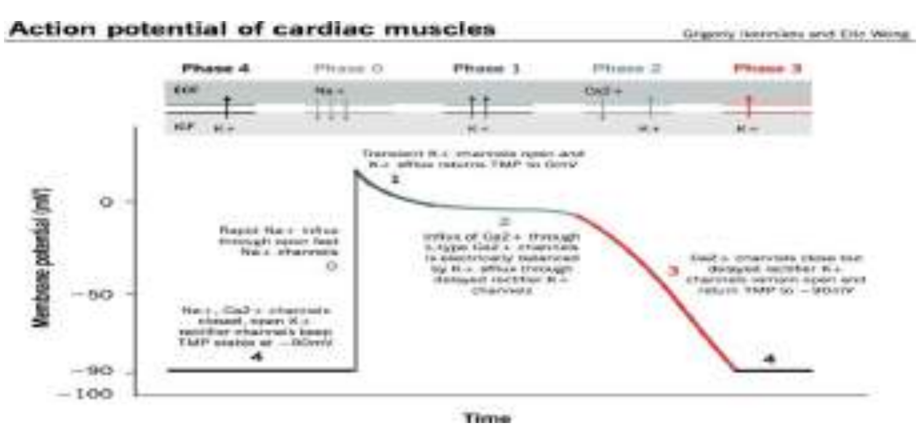
- It occurs due **to opening of K^+ channels**→ outward directed K^+ current along concentration and electric gradients. At the same time, L-type Ca^{++} channels become inactivated and close → stop entrance of Ca^{++}.
- Repolarization continues until the membrane potential reaches **-60 mV**. At this potential the **outward K^+ current becomes gradually inactivated**, while the inward Na^+ current becomes activated again due **to opening of funny current channels** and a new phase 4 is initiated and the whole cycle is spontaneously repeated.

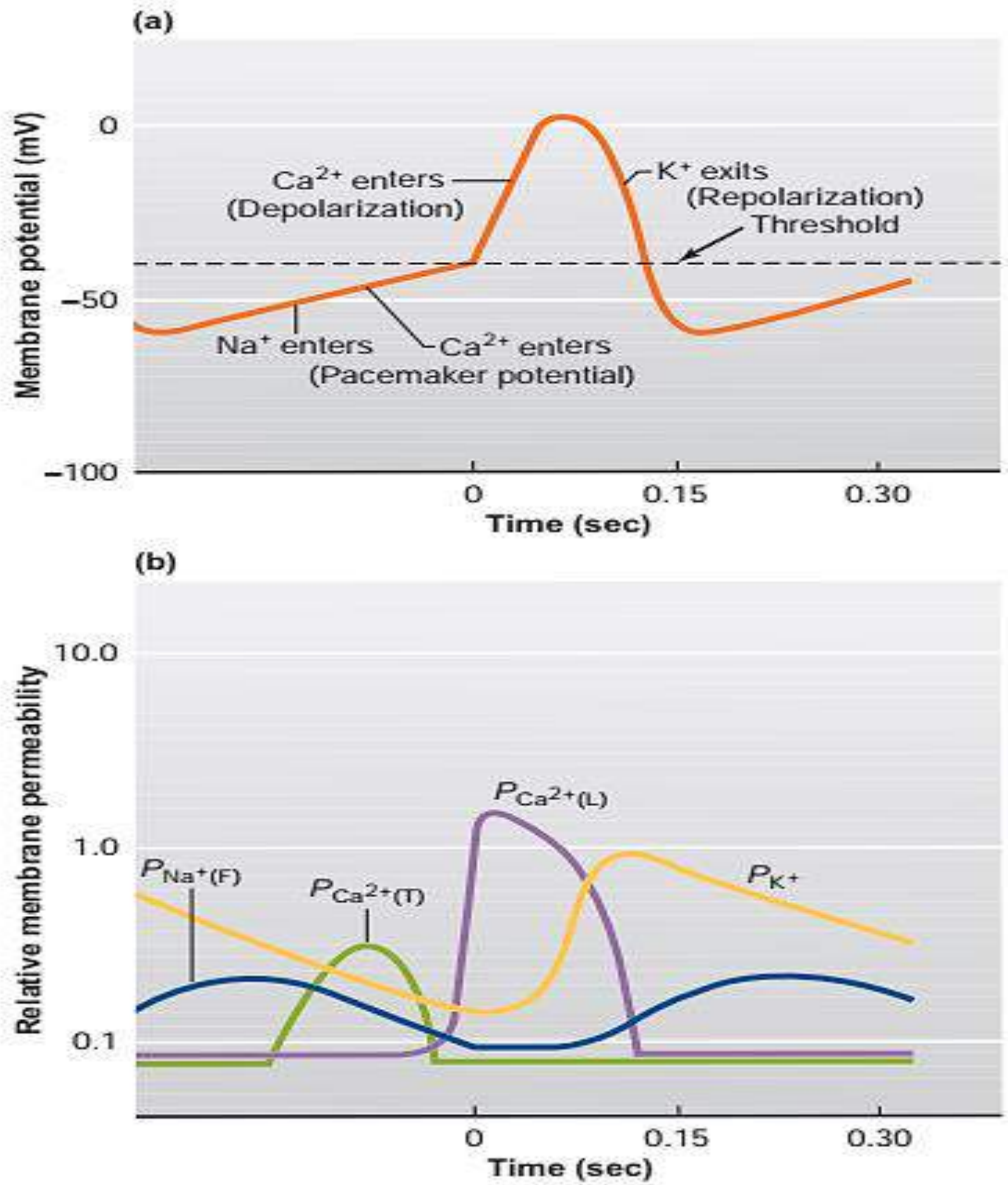
- **N.B.:**

Phase 1 and 2 (which are present in fast AP) are absent.

Pacemaker Potential







(a)
Membrane potential (mV)
0
−50
−100
Ca2+ enters
(Depolarization)
K+ exits
(Repolarization)
Threshold
Na+ enters
Ca2+ enters
(Pacemaker potential)
0
0.15
0.30
Time (sec)
(b)
Relative membrane permeability
10.0
1.0
0.1
PCa2+(L)
PNa+(F)
PCa2+(T)
PK+
0
0.15
0.30
Time (sec)

Characters of pacemaker (slow) action potential

- The RMP is about – 60 mV. It is unstable leading to prepotential (phase 4).
- Its upstroke (depolarization phase; phase 0) is slow, of small magnitude (up to +10 mV) and is due to Ca^{++} influx due to opening of L-type Ca^{++} channels.
- The action potential duration is about 200- 250 ms.
- There is no plateau.
- The falling phase (repolarization; phase 3) is one phase.

N.B.

Although the rhythmicity of the SA node is ~ **100 /min,** the resting heart rate is **only about 75 beat/min, why**?

ANSWER:

This is due the **continuous inhibitory discharge** from the vagus nerve on SA node **decreasing** its inherited rhythm from **100 to 75 beat/min**. This called **VAGAL TONE.**

The vagus nerve **supplies** the whole cardiac muscle **except** the **ventricles** (i.e. called **VAGAL ESCAPE PHENOMENON).** This phenomenon **protects** the ventricles from **abnormally high** vagal stimulation (which can cause **cardiac arrest**).

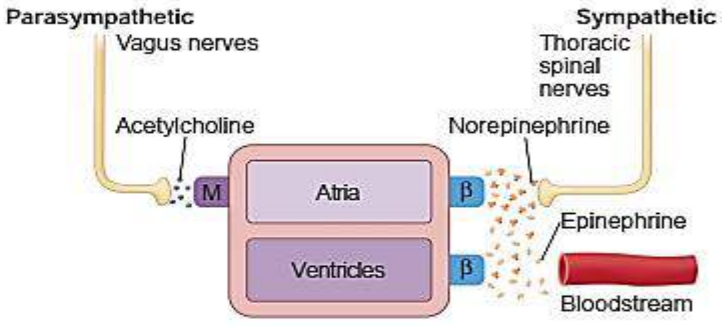


Figure 12.9 Autonomic innervation of heart. Neurons shown represent postganglionic neurons in the pathways. M 5 muscarinic-type ACh receptor; b 5 b-adrenergic receptor.

Factors affecting rhythmicity (chronotropism)

- **The effect of various factors on rhythmicity is called chronotropism.**
- **The factors that stimulate rhythmicity (accelerating the heart rate) are called positive (+ve) chronotropic factors, while factors that inhibit rhythmicity (slowing the heart rate) are called negative (-ve) chronotropic factors.**

A . Nervous Factors:

1. Parasympathetic (Vagal) stimulation via **acetylcholine (ACh) → -ve chronotropic effect.**

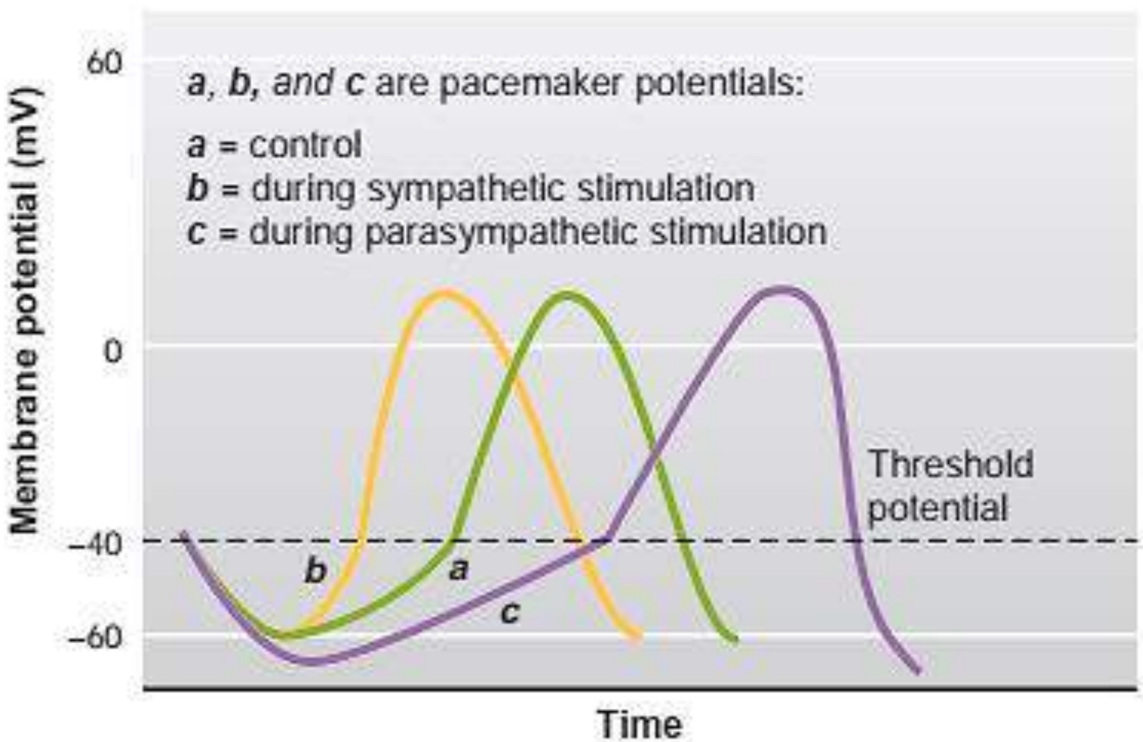
Mechanism:

ACh **stimulates M_2** cholinergic receptors → decreased level of cyclic-AMP → **inhibits F-type (Na^+) channel → ↓ slope of prepotentials →** ↓ rhythmicity. In addition, **Parasympathetic stimulation also hyperpolarizes the plasma membranes of SA node cells by increasing their permeability to K^+→ K^+ efflux. So, the rate of discharge is decreased (e.g. bradycardia).**

2. Sympathetic stimulation via **noradrenaline (NA) → (+ve) chronotropic effect.**

Mechanism:

NA stimulates B_1 adrenergic receptors → increased formation of cyclic-AMP→ **increase inward Na^+ current (F-type channel) & ↑ Ca^{2+} influx → ↑ slope of prepotentials → ↑** rhythmicity**. So, the rate of discharge is increased (e.g. tachycardia).**



B. Chemical Factors:

1. Catecholamines: ↑ rhythmicity.

2. Thyroxine: ↑ rhythmicity

3. Acetyl choline (ACh): ↓ rhythmicity.

4. Blood gases:

- **Mild to moderate O_2 lack (hypoxia) → ↑**rhythmicity.
- **Mild to moderate ↑ CO_2 (hypercapnia) or ↑ H+ (acidosis) → weakly inhibit SA node** rhythmicity but they increase heart rate (HR).
- **Severe O_2 lack (↓O_2), ↑ H+ or ↑ CO_2 →** ↓ rhythmicity.

5. Effect of Drugs:

- **Sympathomimetic drugs → ↑** rhythmicity.
- **Parasympathomimetic drugs (i.e. cholinergic drugs)** → **↓** rhythmicity.
- **Digitalis:** Although it increases myocardial contractility, it **inhibits** SA node activity and **decreases** HR (i.e. **vagal like effect**)

C. Physical Factors:

A rise of body temperature (e.g. in muscular exercise or fever)→ ↑ rhythmicity (heart rate; HR) due to increased rate of discharge of SA node.

Hypothermia→ ↓ rhythmicity due to decreased rate of discharge of SA node.

D. Mechanical factors:

Rise of right atrial pressure ....↑rhythmicity (Bainbridge reflex).

Right atrial distension may directly excite SA node leading to tachycardia (= Bainbridge effect).


THANK YOU