الفسلجة
مرحلة ثانية

الأستاذ المساعد الدكتور
خالد سعود صالح

CARDIOVASCULAR SYSTEM

Autorhythmicity

Automaticity:

Ability of the heart to beat independent of extrinsic stimuli

Rhythmicity:

Ability of the heart to beat in regular cycle

Autorhythmicity*:*

Ability of the heart to beat regularly independent on extrinsic stimuli

It is a myogenic property independent of cardiac innervation:

Completely denervated hearts continue beating rhythmically.

The transplanted hearts have no nerve supply but they beat regularly.

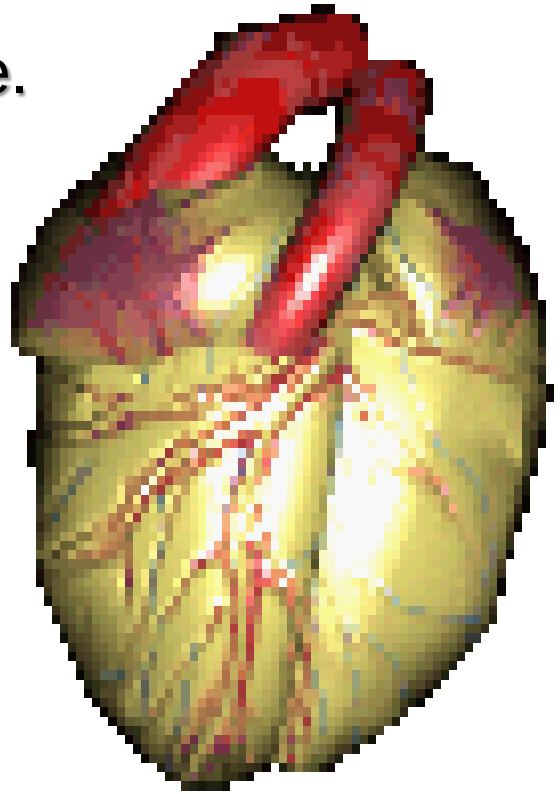
Electrophysiology of the Heart

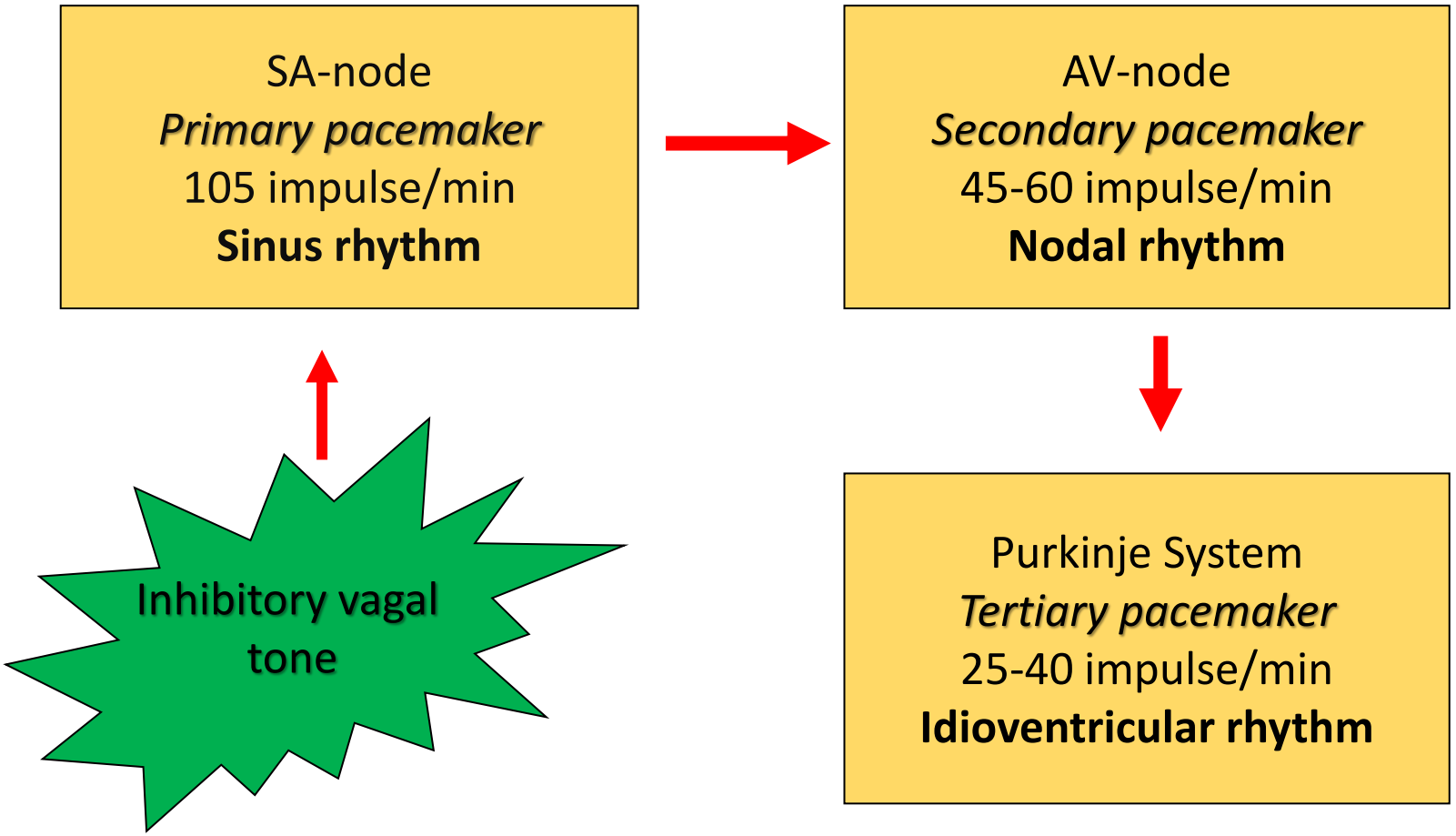
The heart is composed of muscle where the contraction is coupled to the generation of action potentials within its cells. However, cardiac muscle does not require action potentials from nerves to activate its own electrical activity (a fact that makes heart transplantation operations possible). Although both branches of the autonomic nervous system (ANS) innervate the heart, the ANS modulates cardiac function rather than initiates it. Furthermore, there are no anatomic or functional correlates of neuromuscular motor units in the heart. Therefore, the heart cannot recruit neuromuscular units to enhance its force-generating capacity.

Many regions within the heart are capable of originating APs & function as **pacemakers**.
In a normal heart, however, there is only one pacemaker . This is the SA node.

Specialized excitatory and conductive system consists of:

1. Sinus node, sinoatrial node "SA" node.
2. Internodal atrial pathways.
3. The AV node (atrioventricular node).
4. The AV bundle (bundle of His).
5. The left and right bundle branches.
6. The Purkinje fibers.




SA-node
Primary pacemaker
105 impulse/min
Sinus rhythm
AV-node
Secondary pacemaker
45-60 impulse/min
Nodal rhythm
Inhibitory vagal tone
Purkinje System
Tertiary pacemaker
25-40 impulse/min
Idioventricular rhythm

Conductive System of the Heart

1. "SA" node:

located in the right atrium, near the opening of the superior vena cava. The pacemaker of the heart.

2. Internodal atrial pathways.

3. The AV node:

Located in the posterior septal wall of the right atrium. Impulses from the atria are delayed before passing into the ventricles.

4. The AV bundle (bundle of His):

Conducts impulse from the atria into ventricles.

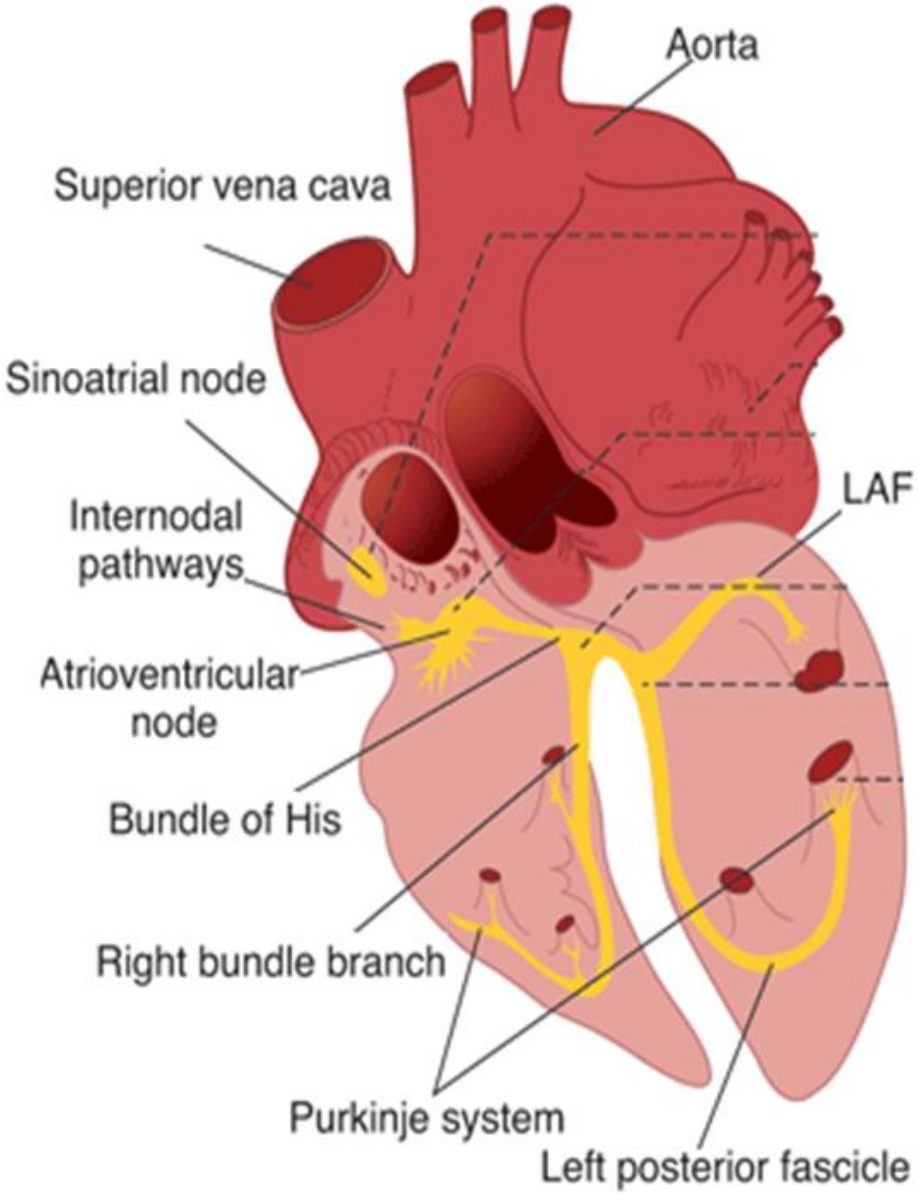
5. Left and right bundle branches:

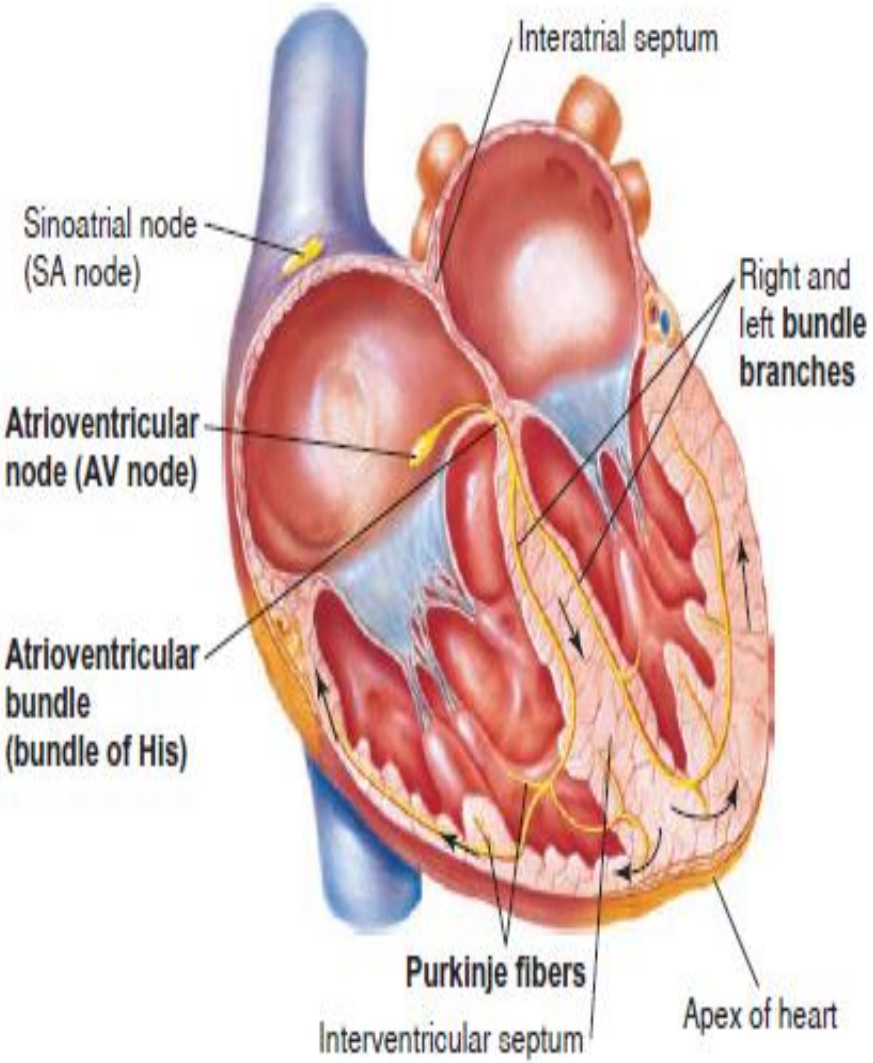
Conduct impulse to all parts of the ventricles.

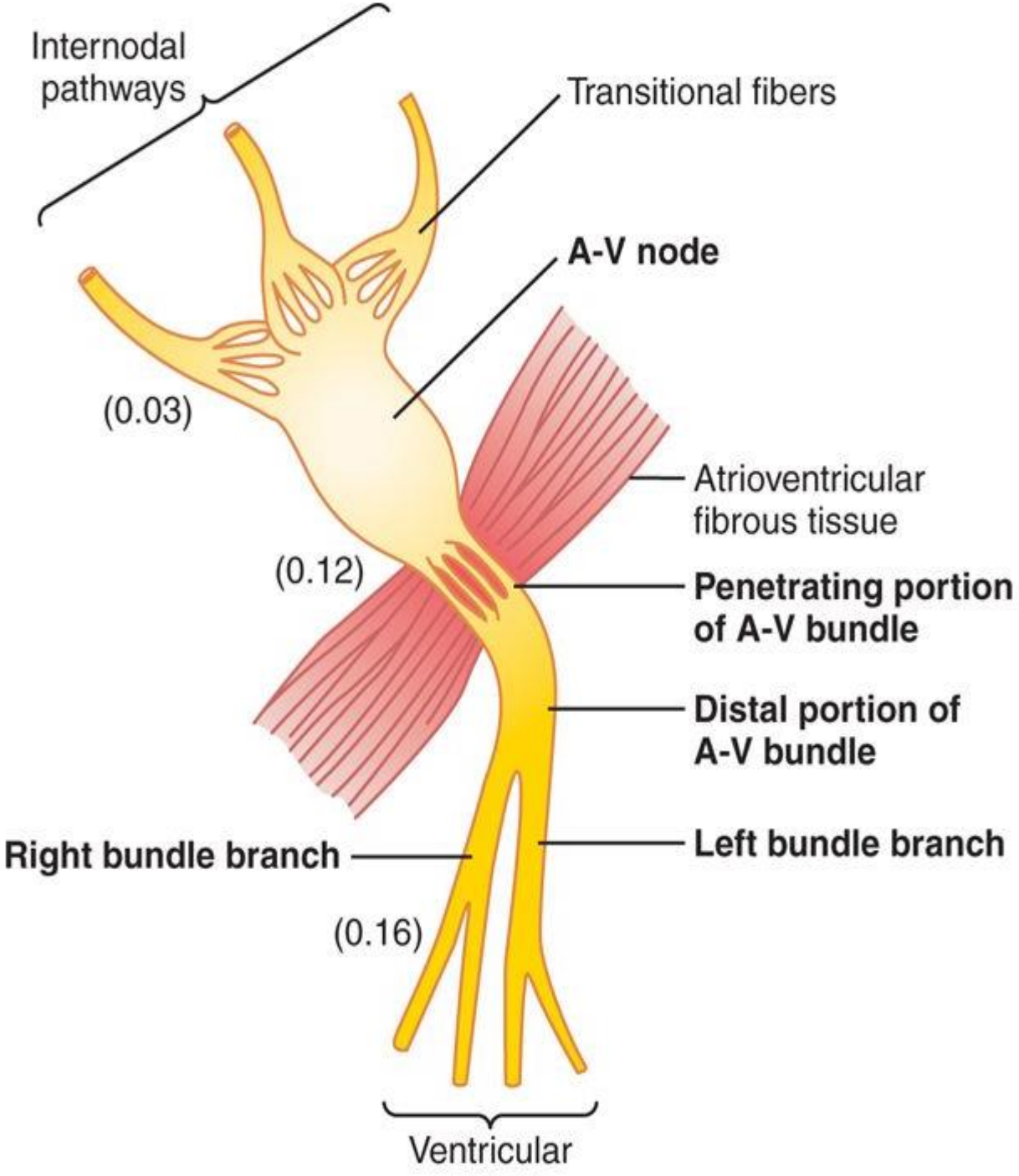
The left bundle branch divides into an anterior and a posterior fascicle. The branches run subendocardially down either side of the septum and contact with the Purkinje system.

6. The Purkinje fibers:

distribute the electrical excitation to the myocytes in all ventricular myocardium.

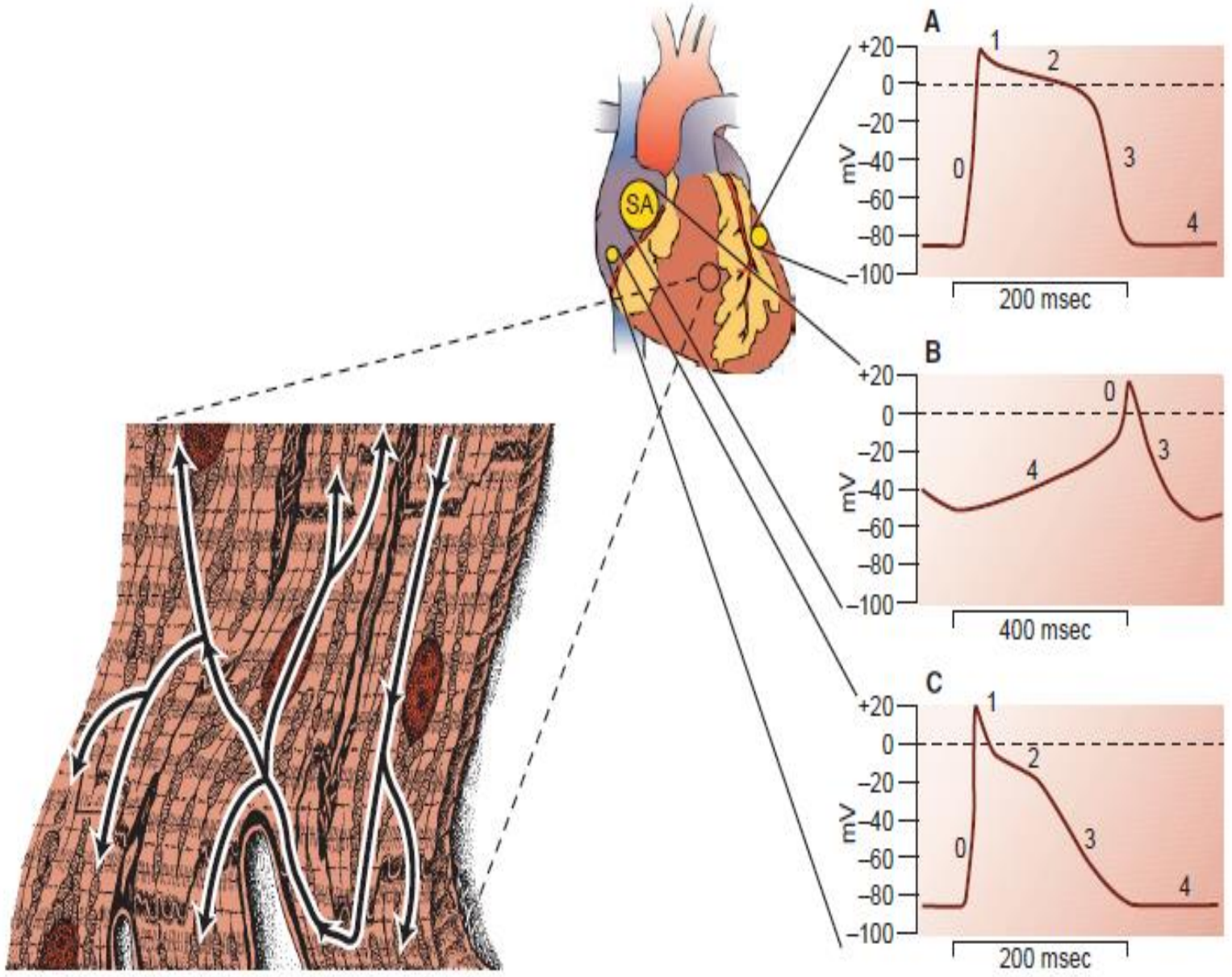

Aorta
Superior vena cava
Sinoatrial node
Internodal pathways
LAF
Atrioventricular node
Bundle of His
Right bundle branch
Purkinje system
Left posterior fascicle


Interatrial septum
Sinoatrial node (SA node)
Right and left bundle branches
Atrioventricular node (AV node)
Atrioventricular bundle (bundle of His)
Purkinje fibers
Interventricular septum
Apex of heart


Internodal pathways
Transitional fibers
A-V node
(0.03)
Atrioventricular fibrous tissue
(0.12)
Penetrating portion of A-V bundle
Distal portion of A-V bundle
Right bundle branch
Left bundle branch
(0.16)
Ventricular

Cardiac cells are electrically connected and can generate their own action potentials.

All myocardial cells are coupled electrically through gap junctions at points called *nexi* . This allows the generation of an action potential in one myocardial cell to spread rapidly to all cells in the heart. This means that, electrically, the heart behaves as a **functional syncytium**, or as if it was one large cell. The advantage of this electrical connectivity between all cells is that it helps the heart contract as a large, coordinated mechanical unit for the purpose of pumping blood. The heart could not function as a pump if its millions of cells activated randomly.


SA
A
+20
0
–20
–40
–60
–80
–100
mV
0
1
2
3
4
200 msec
B
+20
0
–20
–40
–60
–80
–100
mV
0
3
4
400 msec
C
+20
0
–20
–40
–60
–80
–100
mV
0
1
2
3
4
200 msec

the syncytial character of the myocardium also means that the contractile force of the heart as a whole cannot be modulated by the recruitment of motor units, as occurs in skeletal muscle groups. During systole, all cardiac cells are activated; there are no cells left to recruit. In addition, the electrical connectivity of all myocardial cells means that the activation of any cell in the heart can inadvertently activate the heart as a whole

Cardiac Muscle potential:

- AP recorded in a cardiac ventricular muscle fiber, averages about 105 mV.
- After the initial spike, the membrane remains depolarized for about (0.2 sec), "plateau".
- Plateau is followed by abrupt repolarization.
- *Because of plateau*, AP last about 15 times as long as in skeletal muscle.

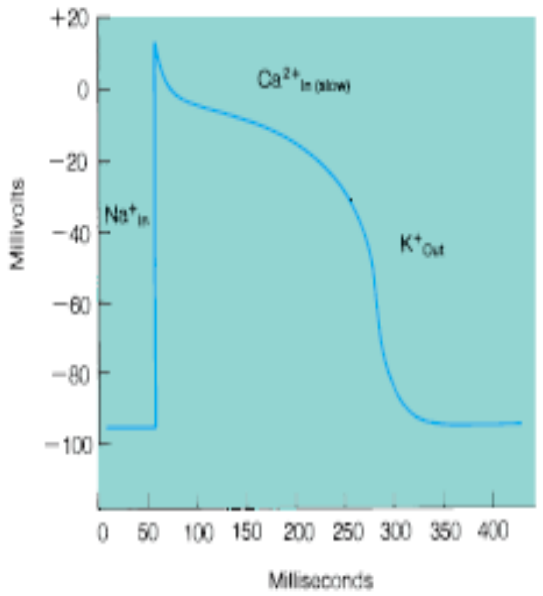
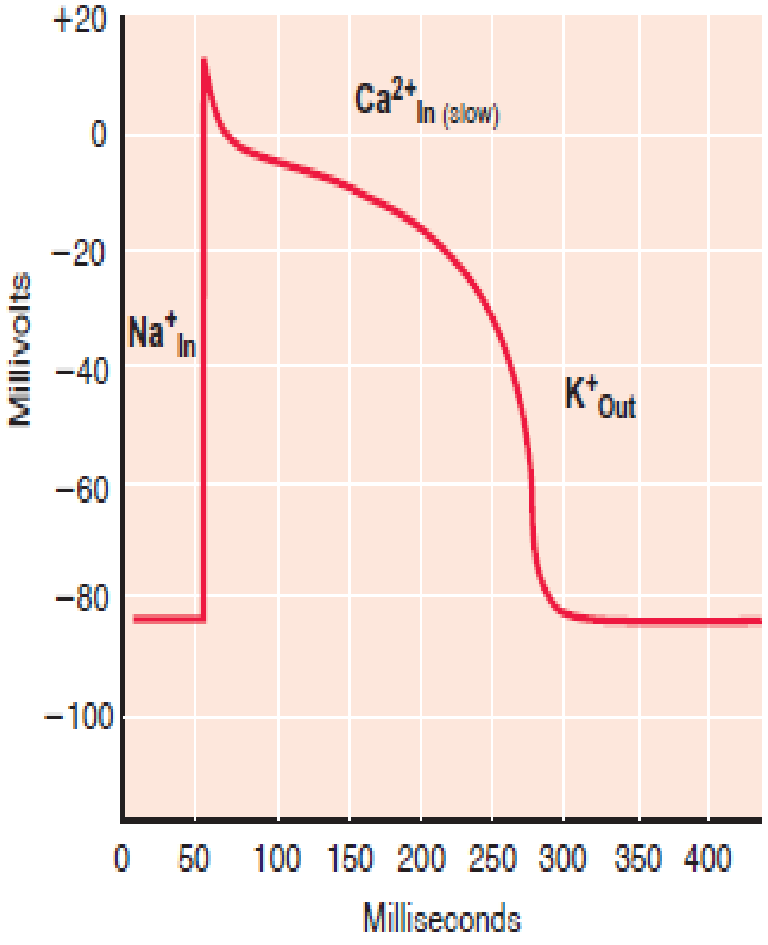
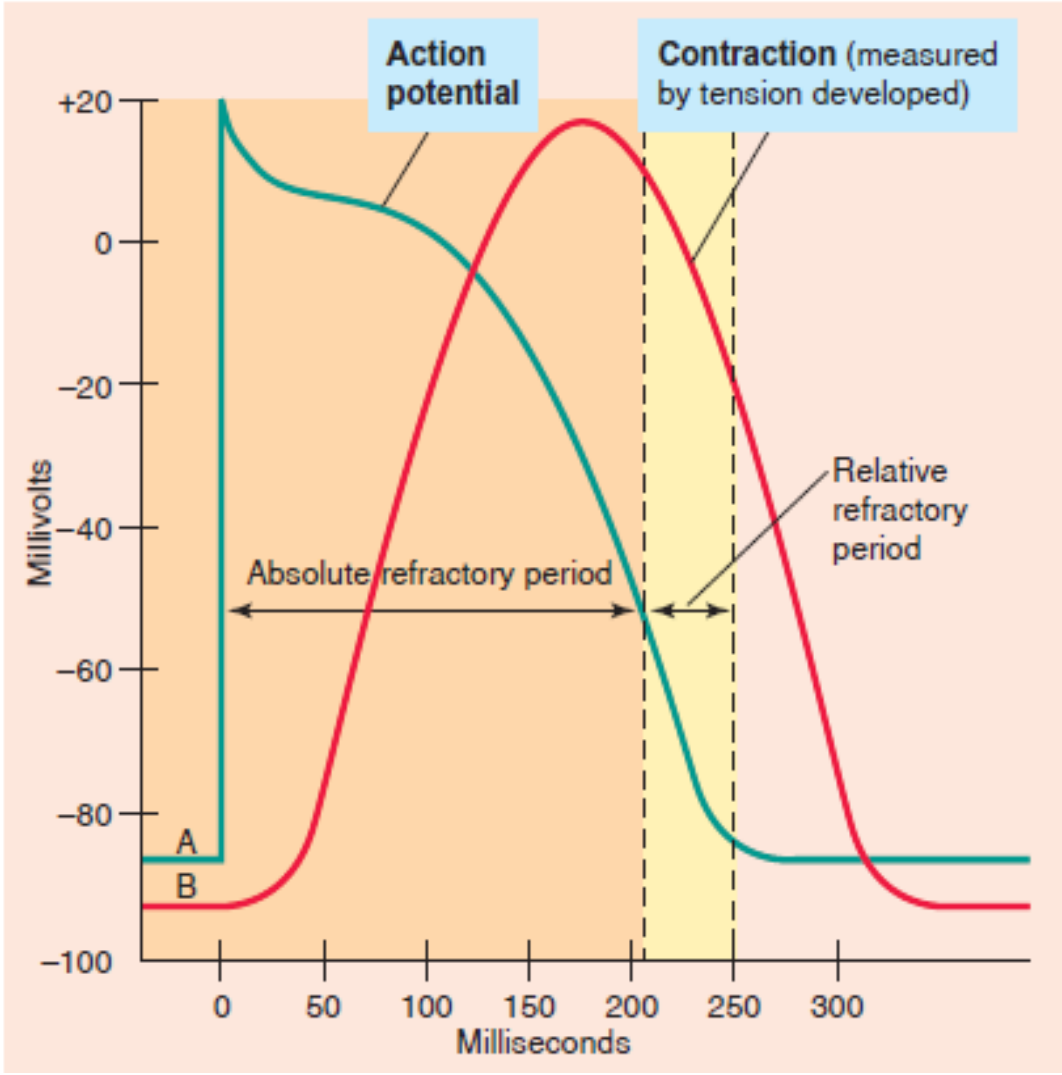


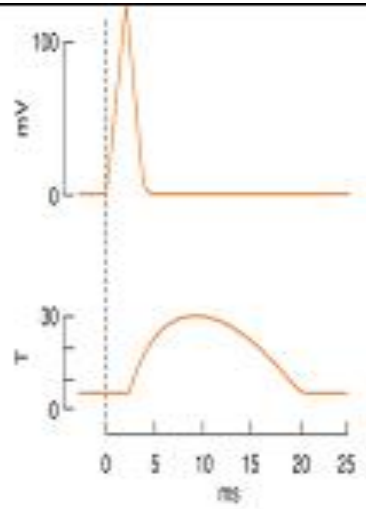
Figure An action potential in a myocardial cell from the ventricles.

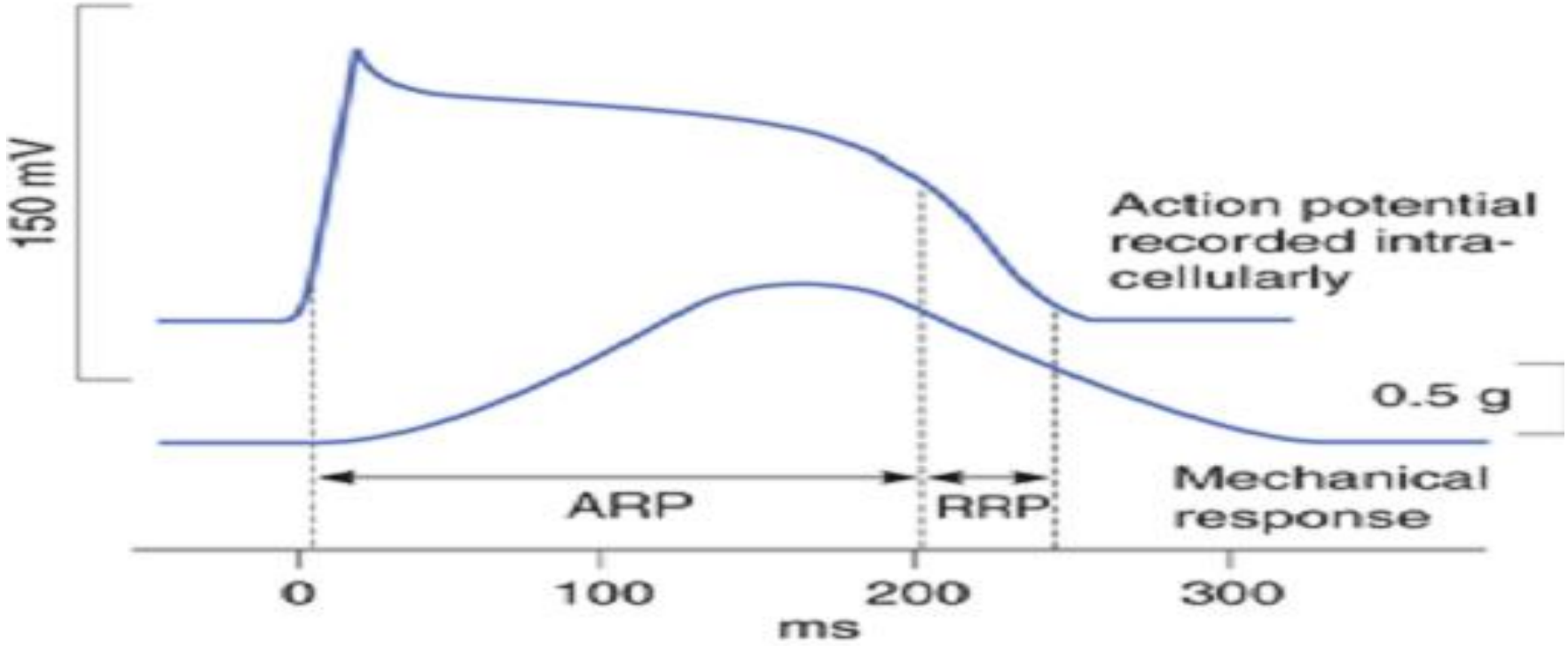
Cardiac Muscle Action potential

:






100
mV
0
30
T
0
0 5 10 15 20 25
ms


150 mV
Action potential recorded intra-cellularly
0.5 g
ARP
RRP
Mechanical response
0
100
200
300
ms

Ionic basis of the AP of the cardiac ventricular muscle fiber cell

Phase 0 (upstroke):
initial rapid depolarization with an overshoot due to rapid Na^+ influx.

Phase 1 (partial repolarization):
following closure of Na^+ channels, initial rapid repolarization is due to K^+ efflux (outflow).

Phase 2 (plateau):
prolonged depolarization due to slower opening of the voltage-gated Ca^{+2} channels with Ca^{+2} influx.

Phase 3 (rapid repolarization):
due to (K^+ efflux) followed the closure of Ca^{+2} channels and, this restores the membrane potential.

Phase 4 (complete repolarization):
Restoration of the resting potential (- 90 mV) . This is achieved by the Na^+-K^+ pump and Na^+-Ca^{+2} exchanger .

Major differences between the membrane properties of cardiac and skeletal muscle

I. With the fast Na^{+} channels, AP is caused by an entirely different population of channels. The slow Ca^{+2}**-** also called **slow Ca^{+2} channels.**

II. Furthermore, Ca^{+2} ions that enter during plateau phase activate the muscle contractile process.

III. Immediately after the onset of AP, the permeability of the muscle membrane for K^{+} ions decreases about five folds.

IV. Membrane permeability for K^{+} increases rapidly when the slow **Ca^{+2} - Na^{+} channels** close at the end of 0.2 to 0.3 sec ; thus ending the AP.