



# CVS PHYSIOLOGY



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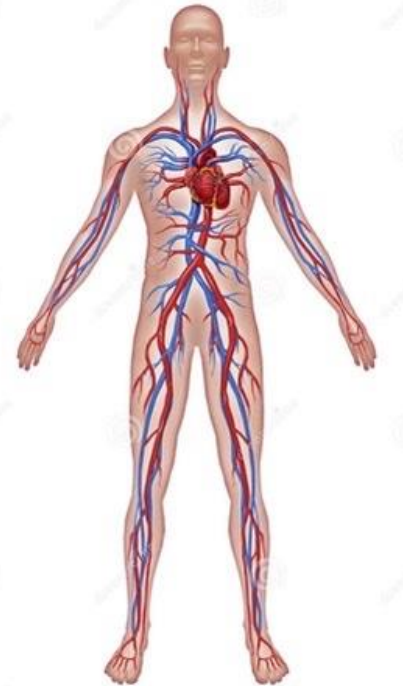
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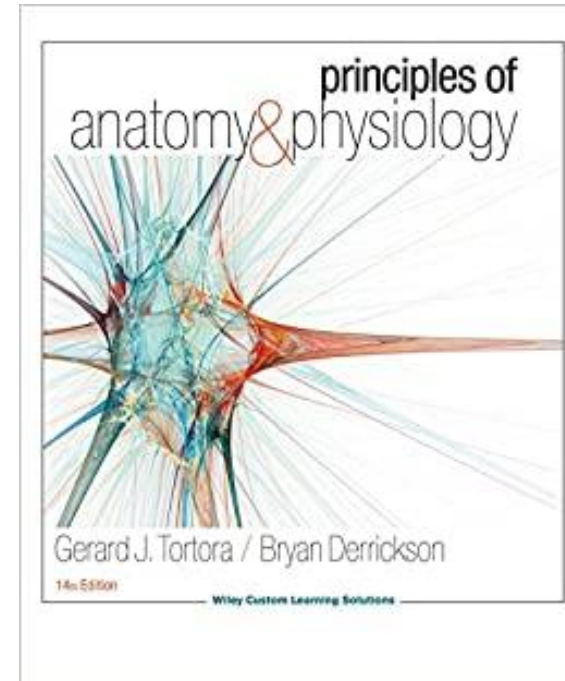
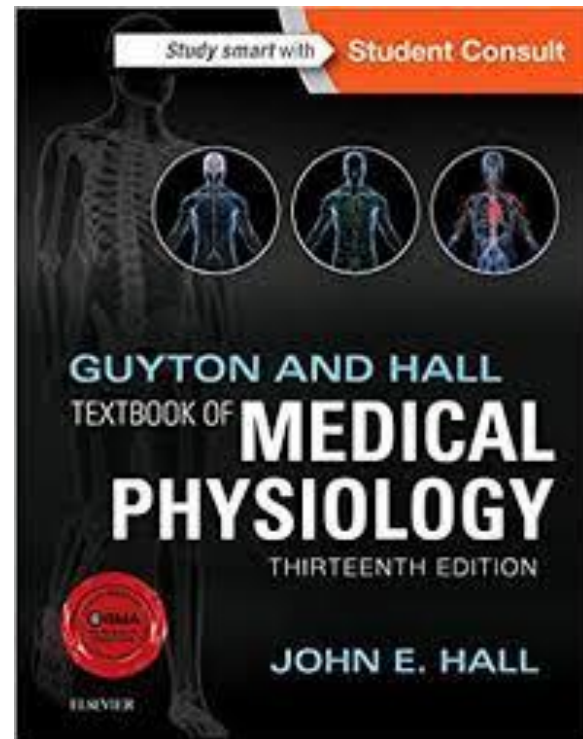
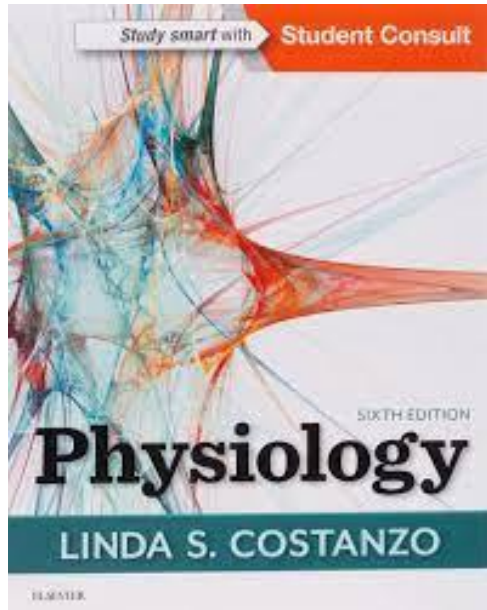
# Cardiovascular Physiology

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# References



9<sup>TH</sup>  
Edition

## Human Physiology From Cells to Systems

Lauralee Sherwood  
Department of Physiology and Pharmacology  
School of Medicine  
West Virginia University

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# Excitatory and conductive systems of the heart

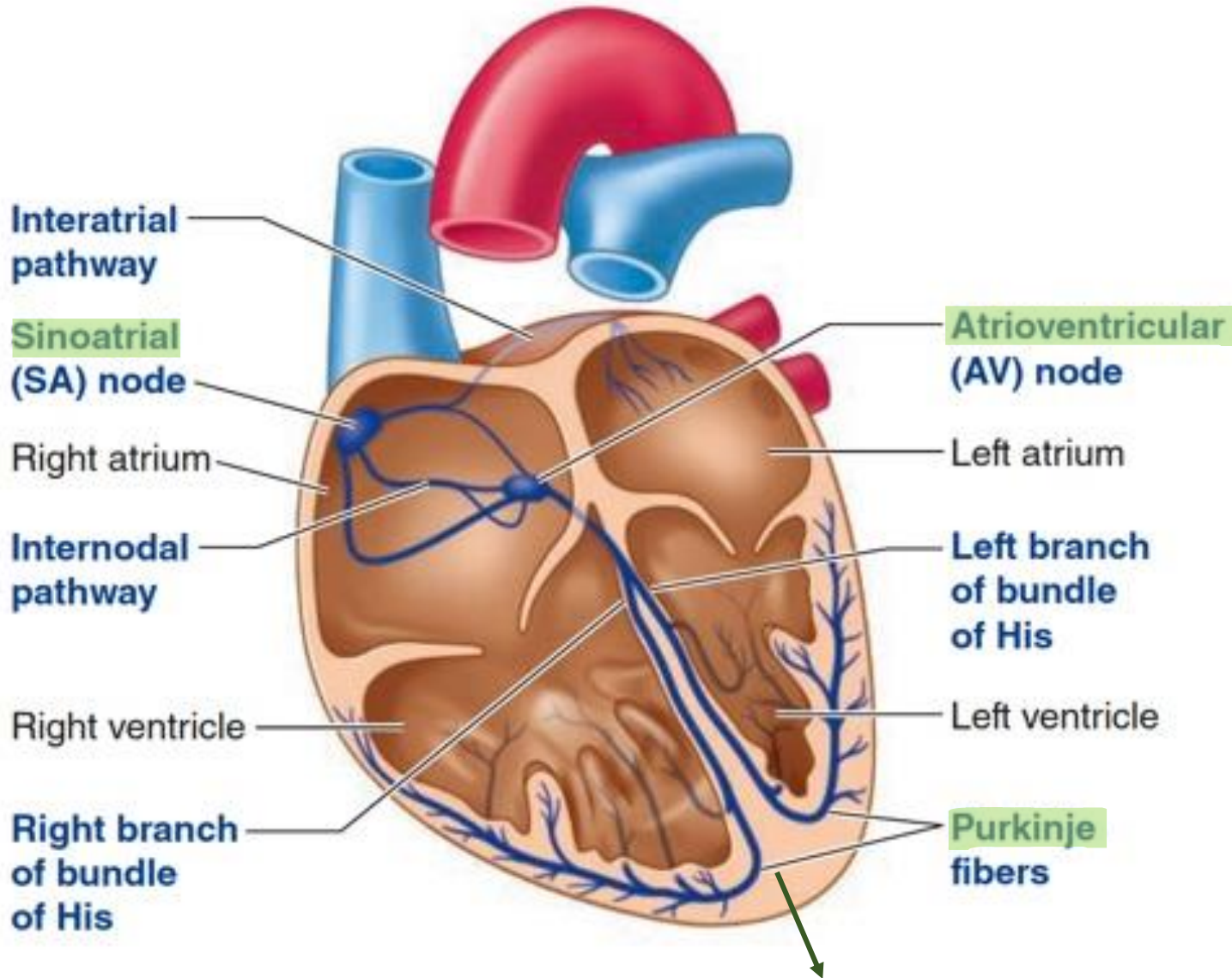
As we mentioned In the previous lecture, there are three types of muscles in the heart:

1.ventricular 2.atrial 3.specialized muscle fibers

these specialized muscle cells almost have no contractile elements but have the ability to generate action potential and induce autorythmicity, and they make up the Conductive System of the heart to ensure this action potential reaches all muscle cells of the heart and promotes their contraction.

# The conduction system

- a network of specialized cardiac muscle fibers called autorhythmic fibers because they are self-excitabile.
- Autorhythmic fibers repeatedly generate action potentials that trigger heart contractions.
- They are about 1% of the cardiac muscle fibers.
- provide a path for each cycle of cardiac excitation to progress through the heart.
- The conduction system ensures that cardiac chambers become stimulated to contract in a coordinated manner, which makes the heart an effective pump.



The branching only begins after reaching the apex of the heart

This conduction system is composed of several main structures that generate and transport action potential in a one-way manner, to muscles that cannot self-generate it so they can contract.

The parts that can autogenerate AP:

- 1- SA nodes, found in the right atrium posterolateral-superiorly just below the superior vena cava opening  
Also called "the pacemaker of the heart" because they determine the frequency of heart contractility by driving the generation of action potential to be transmitted to all cardiac myocytes at the same rate
- 2- AV nodes
- 3- Purkinje fibers

**Interatrial pathway**

**SA node**

Right atrium

**Internodal pathway**

Electrically nonconductive fibrous tissue

Right ventricle

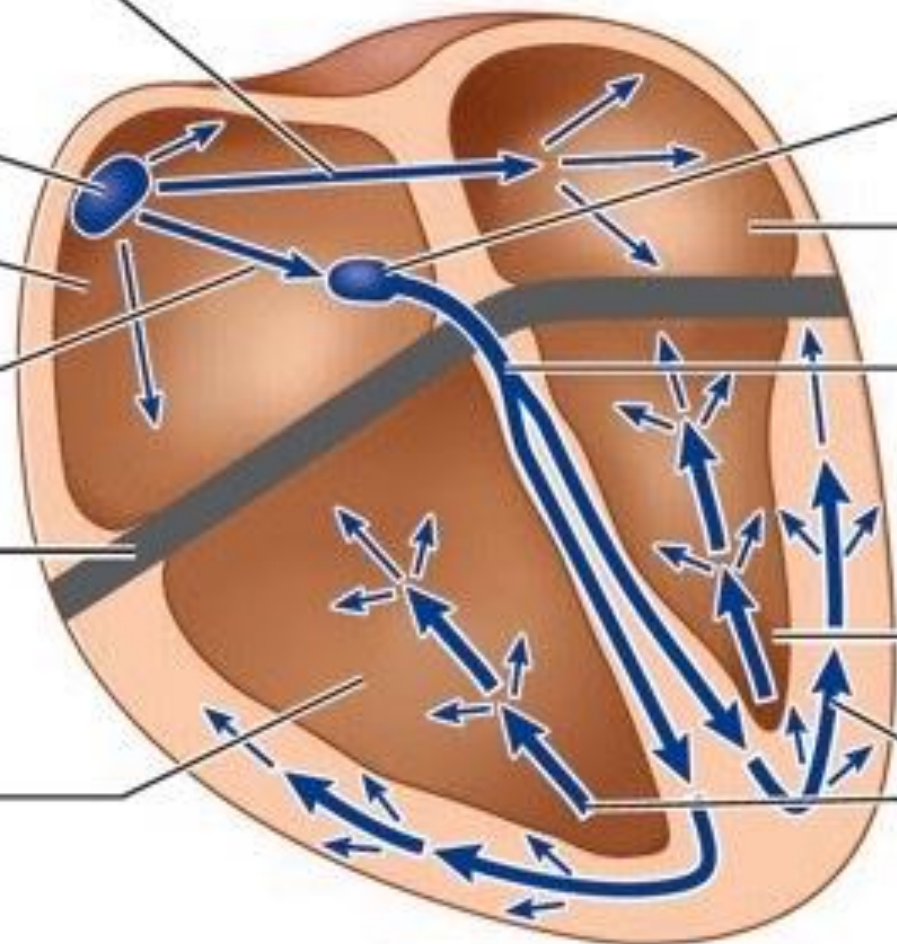
**AV node**

Left atrium

**Bundle of His**

Left ventricle

**Purkinje fibers**



# SA node to Atria

- the action potential of SA node spreads through the entire atrial muscle mass (directly to the right atrium and through Inter-Atrial pathway to the left atrium) and, eventually, to the A-V node.

**NOTE:** types of communication between excitable cells include:

1.chemical synapse: neurotransmitters are exocytosed and bind to receptors on the next structure→ indirect and takes more time (ex. between a motor neuron and skeletal muscle through the motor end plate)

2.**electrical synapse:** gap junctions allow ions to move faster from one cell to another and ,therefore, facilitate rapid propagation of action potential between the great mass of muscle cells in the heart so it can contract as one unit.

Different parts of this conduction system have different **conduction velocities** (the speed at which they transport action potential to the next structure) according to the number of gap junctions between their cells ,hence purkinji fibers, for example, have the highest conduction velocity, because they have the highest no. of gap junctions.

- The velocity of conduction in most atrial muscle is about 0.3 m/sec, but conduction is more rapid, about 1 m/sec , in several small bands of atrial fibers, such as the anterior interatrial band (also called Bachman's bundle), passes through the anterior walls of the atria to the left atrium

so both Atria can contract at the same time, since the left atrium is further away.



- In addition, three other small bands curve through the anterior, lateral, and posterior atrial walls and terminate in the A-V node. These are called, respectively, the anterior, middle, and posterior internodal pathways.
- The cause of more rapid velocity of conduction in these bands is the presence of specialized conduction fibers. These fibers are similar to even more rapidly conducting Purkinje fibers of the ventricles.

These numbers represent the time from the excitation of the SA node:

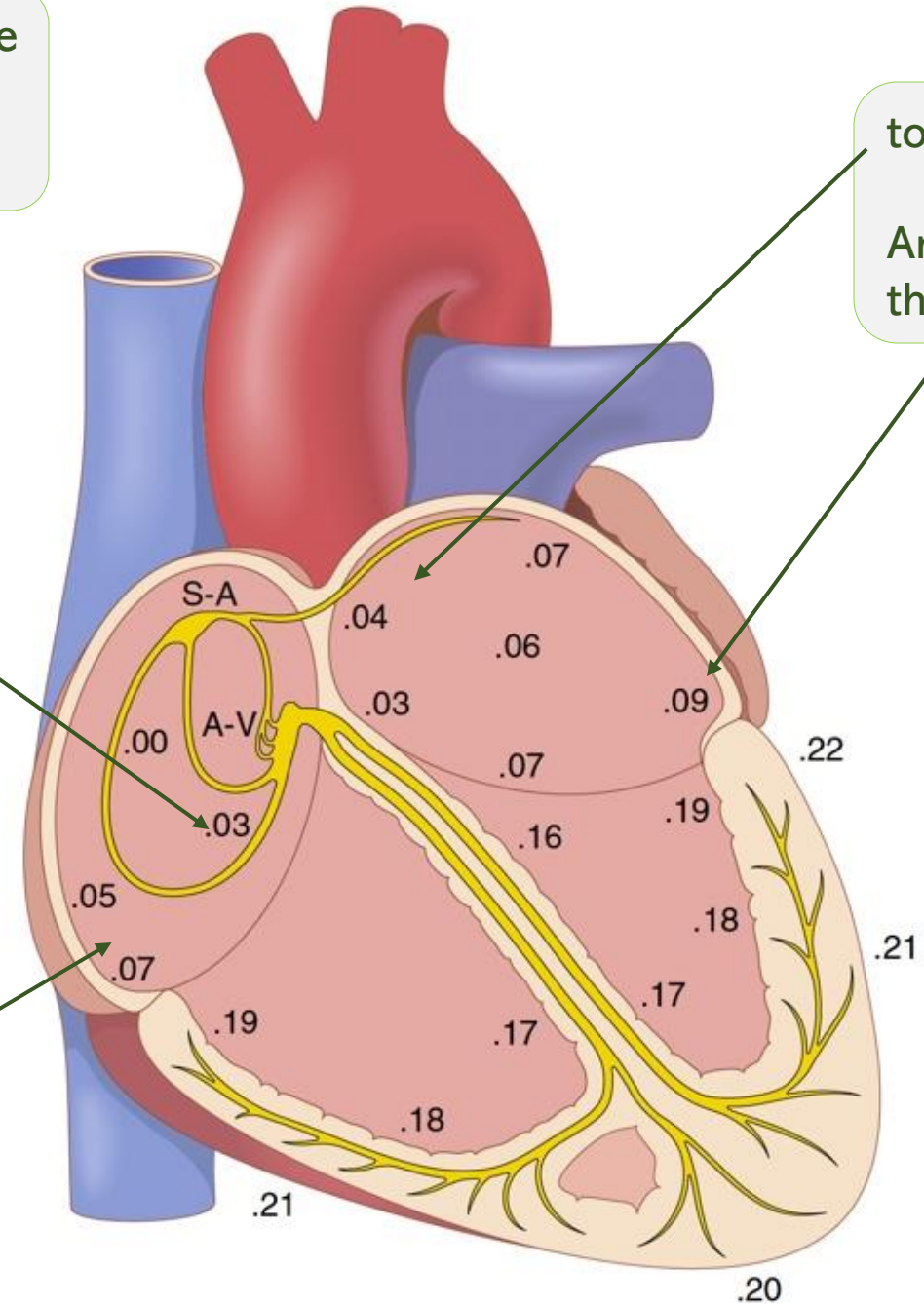
to the left atrium in .04s

And to the point furthest away in the left atrium .09s

Internodal fibers will transmit the discharge signal from the SA nodes to the AV nodes in .03s

directly from SA nodes to the right atrium takes about .05 - .07s

So, the action potential reaches these points with slight differences in time so they can contract as one unit to empty their blood before the ventricles contract



Do not memorize conduction velocities and numbers but understand the relations/sequence and which are faster/slower

- The A-V node is located in the posterior wall of the right atrium, immediately behind the tricuspid valve.
-

# SA node to AV node

- the impulse, after traveling through the internodal pathways, reaches the A-V node about 0.03 second after its origin in the sinus node.
- Then, there is a delay of another 0.09 second in the A-V node itself before the impulse enters the penetrating portion of the A-V bundle (bundle of His), where it passes into the ventricles.
- A final delay of another 0.04 second occurs mainly in this penetrating A-V bundle, which is composed of multiple small fascicles passing through the fibrous tissue separating the atria from the ventricles.
- Thus, the total delay in the A-V nodal and A-V bundle system is about 0.13 second. This delay, in addition to the initial conduction delay of 0.03 second from the SA node to the A-V node, makes a total delay of 0.16 second before the excitatory signal finally reaches the contracting muscle of the ventricles. (0.16 is the time required for the signals to be delivered from the SA node to the bundle branches)

# AV delay

- The atrial conductive system is organized so that the cardiac impulse does not travel from the atria into the ventricles too rapidly; this delay allows time for the atria to empty their blood into the ventricles before ventricular contraction begins. (we want to make sure that the atria did contract and relax before the turn of the ventricles comes) → so the AV delay is so important in maintaining a one-way direction of the signals.
- It is primarily the A-V node and its adjacent conductive fibers that delay this transmission into the ventricles.

# AV bundle as a one way conduction path

- A special characteristic of the A-V bundle is the inability, except in abnormal states, of action potentials to travel backward from the ventricles to the atria. This characteristic prevents re-entry of cardiac impulses by this route from the ventricles to the atria, allowing only forward conduction from the atria to the ventricles.

- everywhere, except at the A-V bundle, the atrial muscle is separated from the ventricular muscle by a continuous fibrous barrier. (electrical current cannot travel through fibrous tissue; so it acts as an insulator)
- This barrier normally acts as an insulator to prevent passage of the cardiac impulse between atrial and ventricular muscle through any other route besides forward conduction through the A-V bundle (bundle of Hiss).
- In rare cases, an abnormal muscle bridge, or accessory pathway, does penetrate the fibrous barrier elsewhere besides at the A-V bundle.
- Under such conditions, the cardiac impulse can re-enter the atria from the ventricles and cause serious cardiac arrhythmias.

**Remember** that the ventricular wall is composed of 3 main different layers:

1- Endocardium; the innermost layer.

2- Myocardium; the muscle layer.

3- **Epicardium** ; the outermost layer.

And the myocardium itself is not a one layer; it's composed of several layers (that are connected together by gap junctions), and it's not just linear; its layers go spirally in different directions from the apex to the base, some of them will go clockwise and others will go counter clockwise —> **why?**

Because this kind of motion helps the base to empty the ventricles from the fluid to get the most efficient contraction possible during pumping ( زي لما )

(نعصر الغسيل نلفه باتجاهين مختلفين

. Myocardium is a thick layer and it's stimulated by the purkinje fibers



- After penetrating the fibrous tissue between the atrial and ventricular muscle, the distal portion of the A-V bundle passes downward in the ventricular septum for 5 to 15 mm toward the apex of the heart.
- Then, the bundle divides into left and right bundle branches on the two respective sides of the ventricular septum.
- Each branch spreads downward toward the apex of the ventricle, progressively dividing into smaller branches.
- These branches, in turn, course sidewise around each ventricular chamber and back toward the base of the heart. The ends of the Purkinje fibers penetrate (from the side of endocardium) about one-third of the way into the muscle mass and finally become continuous with the cardiac muscle fibers. (And the remaining two-thirds will be stimulated through the gap junctions between the ventricular myocytes)

Purkinje fibers kind of penetrate the ventricular cardio myocytes but they don't cover the whole thickness of this muscle layer

# Slow conduction

- The slow conduction in the transitional, nodal, and penetrating A-V bundle fibers is caused mainly by diminished numbers of gap junctions between successive cells in the conducting pathways, so there is great resistance to conduction of excitatory ions from one conducting fiber to the next. Therefore, it is easy to see why each succeeding cell is slow to be excited.

## AV bundle to the ends of Purkinje fibers

- The total elapsed time averages only 0.03 second from the time the cardiac impulse enters the bundle branches in the ventricular septum until it reaches the terminations of the Purkinje fibers. Therefore, once the cardiac impulse enters the ventricular Purkinje conductive system, it spreads almost immediately to the entire ventricular muscle mass.

# Purkinje fibers

- The rapid transmission of action potentials by Purkinje fibers is believed to be caused by a very high level of permeability of the gap junctions at the intercalated discs between the successive cells that make up the Purkinje fibers (it's also said to be due to the very large size of the purkinje fibers which is even larger than the ventricular myocytes). Therefore, ions are transmitted easily from one cell to the next, thus enhancing the velocity of transmission.

# Purkinje fibers

- They are very large fibers, even larger than the normal ventricular muscle fibers, and they transmit action potentials at a velocity of 1.5 to 4.0 m/sec, a velocity about six times that in the usual ventricular muscle and 150 times that in some of the A-V nodal fibers.
- This velocity allows almost instantaneous transmission of the cardiac impulse throughout the entire remainder of the ventricular muscle.

**Purkinje fibers** have the **fastest conduction velocity** in the heart (because they are the ones responsible for delivering the action potential to all the ventricular muscles that they can reach—> why do we need this? Because the contraction should be syncytial at the same time and as a one unit for both ventricles)

- Once the impulse reaches the ends of the Purkinje fibers, it is transmitted through the ventricular muscle mass by the ventricular muscle fibers themselves. The velocity of transmission is now only 0.3 to 0.5 m/sec, one-sixth that in the Purkinje fibers.

- Because of the myocardial angulation, transmission from the endocardial surface to the epicardial surface of the ventricle requires as much as another 0.03 second, approximately equal to the time required for transmission through the entire ventricular portion of the Purkinje system.
- Thus, the total time for transmission of the cardiac impulse from the initial bundle branches to the last of the ventricular muscle fibers in the normal heart is about 0.06 second.

# Role of Purkinji in synchronus ventricular contraction

- The rapid conduction of the Purkinje system normally permits the cardiac impulse to arrive at almost all portions of the ventricles within a narrow span of time, exciting the first ventricular muscle fiber only 0.03 to 0.06 second ahead of excitation of the last ventricular muscle fiber. This timing causes all portions of the ventricular muscle in both ventricles to begin contracting at almost the same time and then to continue contracting for about another 0.3 second.



- Effective pumping by the two ventricular chambers requires this synchronous type of contraction. If the cardiac impulse should travel through the ventricles slowly, much of the ventricular mass would contract before contraction of the remainder, in which case the overall pumping effect would be greatly depressed. Indeed, in some types of cardiac dysfunction, several of which are slow transmission does occur, and the pumping effectiveness of the ventricles is decreased as much as 20% to 30%.

- Why then does the sinus node rather than the A-V node or the Purkinje fibers control the heart's rhythmicity? (In other words: what makes the SA node the pacemaker of the heart?)
- The answer derives from the fact that the discharge rate of the sinus node is considerably faster than the natural self-excitatory discharge rate of either the A-V node or the Purkinje fibers. Each time the sinus node discharges, its impulse is conducted into both the A-V node and Purkinje fibers, also discharging their excitable membranes. However, the sinus node discharges again before either the A-V node or Purkinje fibers can reach their own thresholds for self-excitation.
- Therefore, the new impulse from the sinus node discharges both the A-V node and Purkinje fibers before self-excitation can occur in either of these sites.

**Pay attention:** the conduction velocity is different than the discharge rate

- . The fastest conduction velocity—> purkinje fibers
- . The fastest discharge rate—> SA node

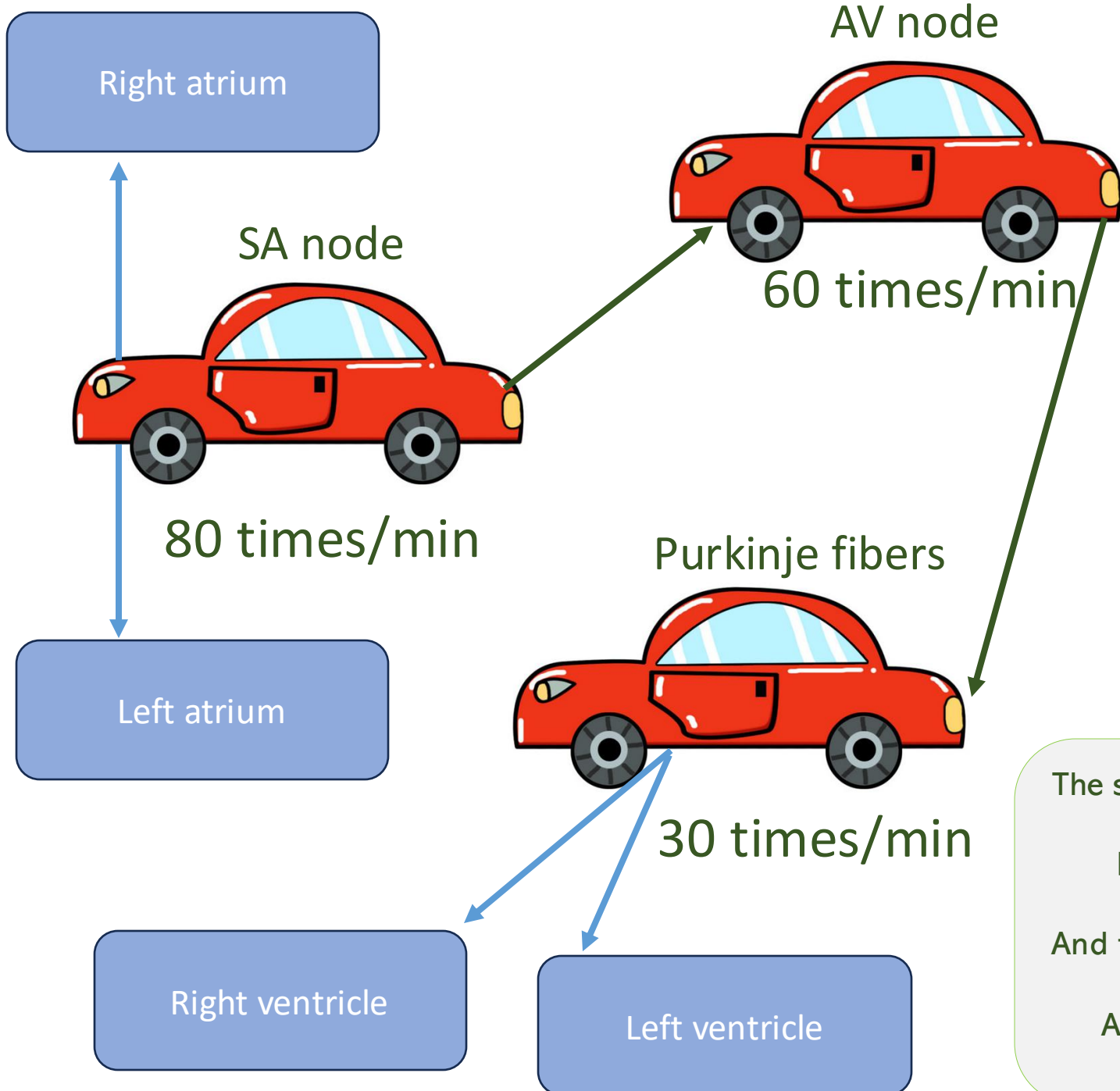
# Discharge rates

- The A-V nodal fibers, when not stimulated from some outside source, discharge at an intrinsic rhythmical rate of 40 to 60 times per minute, and the Purkinje fibers discharge at a rate somewhere between 15 and 40 times per minute. These rates are in contrast to the normal rate of the sinus node of 70 to 80 times per minute.

Discharge rate of the SA node = the heart rate (the pumping rate of the heart/ the usually calculated pulse)  
Hence the name: pacemaker of the heart

# SA node as a pacemaker

- the sinus node controls the beat of the heart because its rate of rhythmical discharge is faster than that of any other part of the heart.
- Therefore, the sinus node is almost always the pacemaker of the normal heart.



**Use your imagination! 💣**

A good example from the doctor for better understanding:

- 1- The written **numbers** under each car represent estimated normal discharge rates for Purkinje fibers, SA and AV nodes (the structures that are able to self-generate action potential)
- 2- The **arrows** represent the transmission of that action potential between different parts of the heart
- 3- The **blue boxes** represent the other parts of the heart that are not able to self-generate AP (don't drive, not cars) and they're gonna wait for the self-generating structures to drive them (the cars)

The system is **connected** and has 3 cars driving in 3 different velocities,  
**In which velocity you think will the system be going?**  
 In **80** of course!

And the 2 other structures will be pulled up with this highest velocity

And that's the principle behind the SA node being the pacemaker of the heart ❤️ (**highest discharge rate**)

**When SA node is affected by a certain pathology**, it becomes non-functional and the AV node (2<sup>nd</sup> fastest discharge rate) will take over, and the heart rate becomes almost 60 times/min  
It's not the best since we didn't reach the full capacity of the heart but it's still OK!

But if the **abnormality is in the connection between the atria and ventricles (A-V block)**—> There will be a disconnection between the atria and the ventricles (1- the **ventricles** will be driven by the purkinje fibers with a very low discharge rate which will be just enough to save the patient life until he gets treated

2- the **atria** would be under the SA node control so they will be normally functioning) (when we conduct an ECG to this patient we will notice normal waves of the atria with much slower waves in the ventricles)

# Ectopic pacemaker

- A pacemaker elsewhere than the sinus node is called an ectopic pacemaker.
- An ectopic pacemaker causes an abnormal sequence of contraction of the different parts of the heart and can cause significant weakening of heart pumping.

- Occasionally, some other part of the heart develops a rhythmical discharge rate that is more rapid than that of the sinus node.
- For example, this development sometimes occurs in the A-V node or in the Purkinje fibers when one of these becomes abnormal.
- In either case, the pacemaker of the heart shifts from the sinus node to the A-V node or to the excited Purkinje fibers. Under rarer conditions, a place in the atrial or ventricular muscle develops excessive excitability and becomes the pacemaker.



- Another cause of shift of the pacemaker is blockage of transmission of the cardiac impulse from the sinus node to the other parts of the heart. The new pacemaker then usually occurs at the A-V node or in the penetrating portion of the A-V bundle on the way to the ventricles.
- When A-V block occurs—that is, when the cardiac impulse fails to pass from the atria into the ventricles through the A-V nodal and bundle system—the atria continue to beat at the normal rate of rhythm of the sinus node while a new pacemaker usually develops in the Purkinje system of the ventricles and drives the ventricular muscle at a new rate, somewhere between 15 and 40 beats per minute.

Thank you

## Additional sources

1. Book pages
2. Youtube videos
3. Webpages...etc
4. .

لا يألّفنّ أحدكم مصاب أخيه وإن طال!  
٦-١١-٢٠٢٤ عام وشهر على أكبر مجزرة شهدها  
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VERSIONS	SLIDE #	BEFORE CORRECTION	AFTER CORRECTION
V1→V2	16	Pericardium	Epicardium
V2→V3			



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