Cardiovascular Physiology

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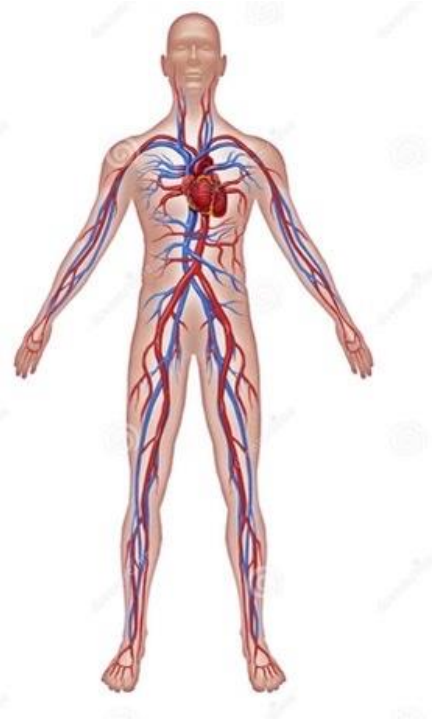

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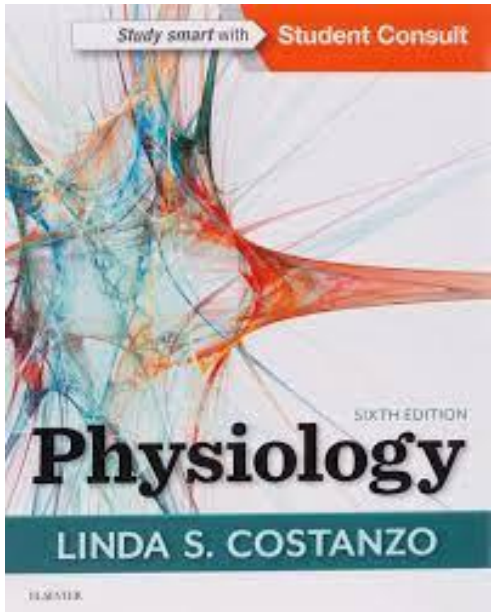
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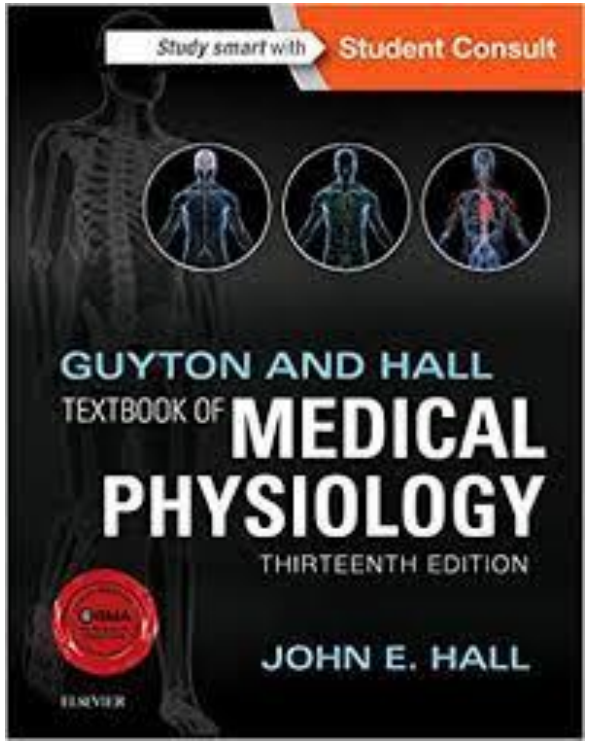
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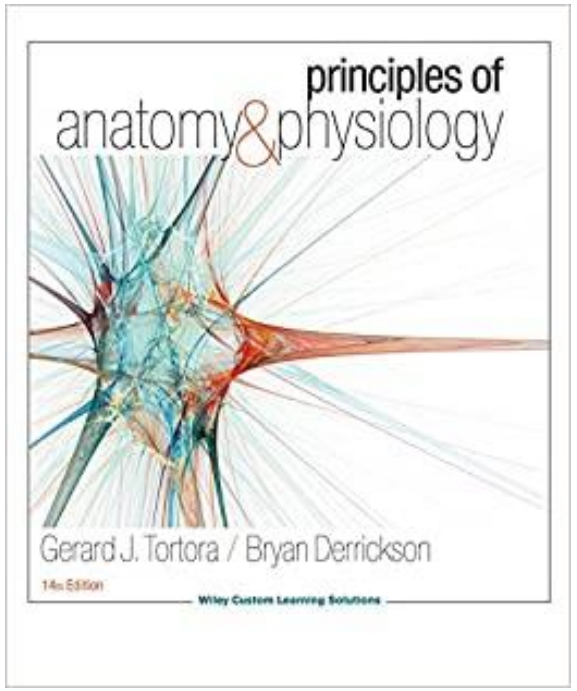
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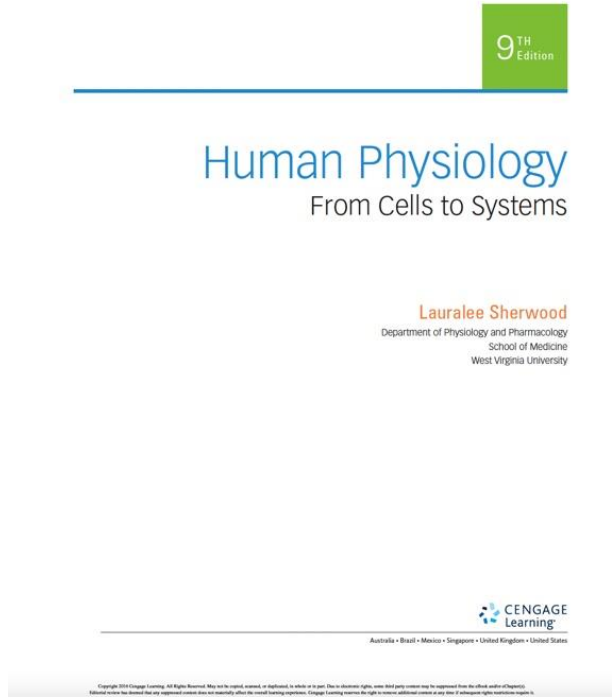
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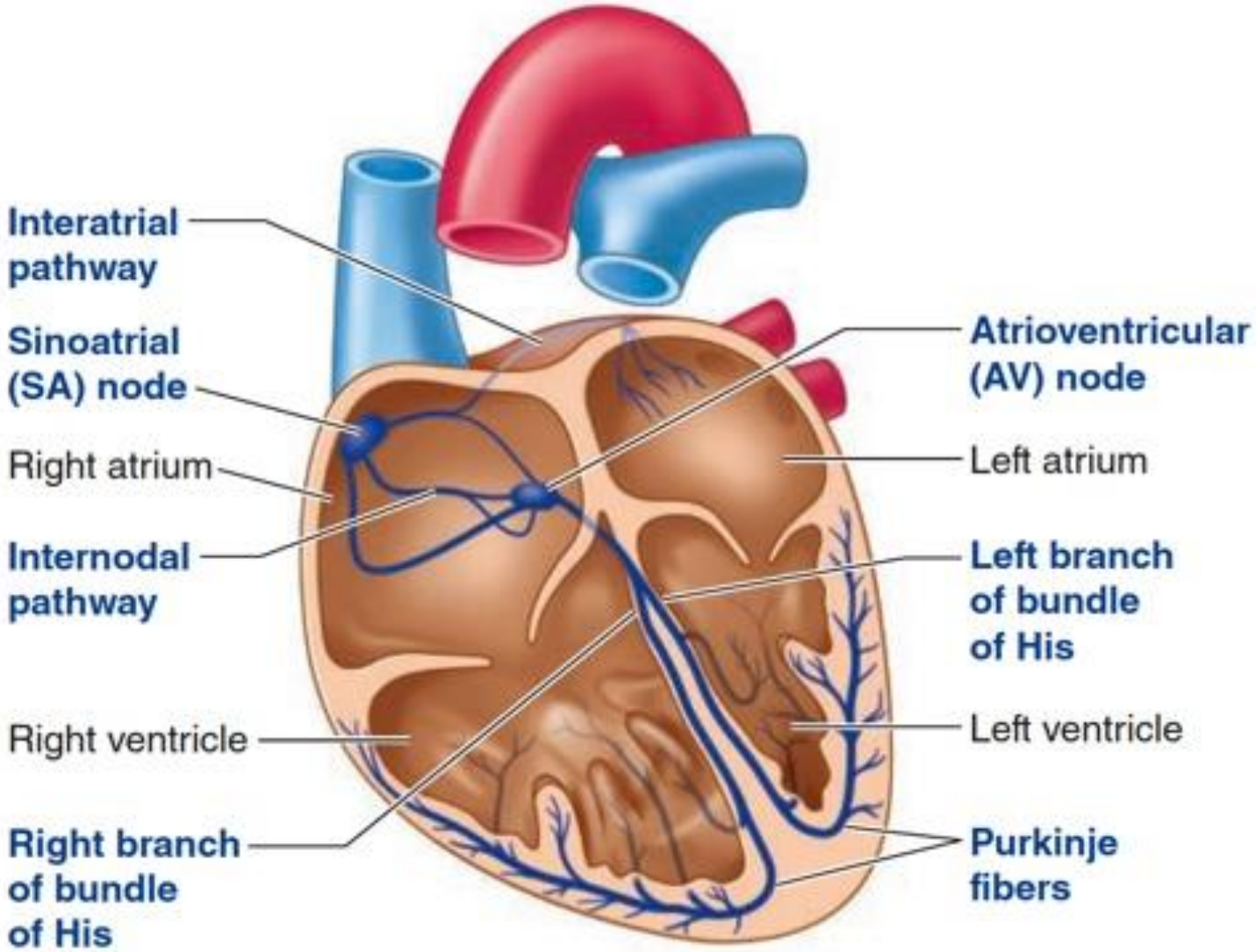
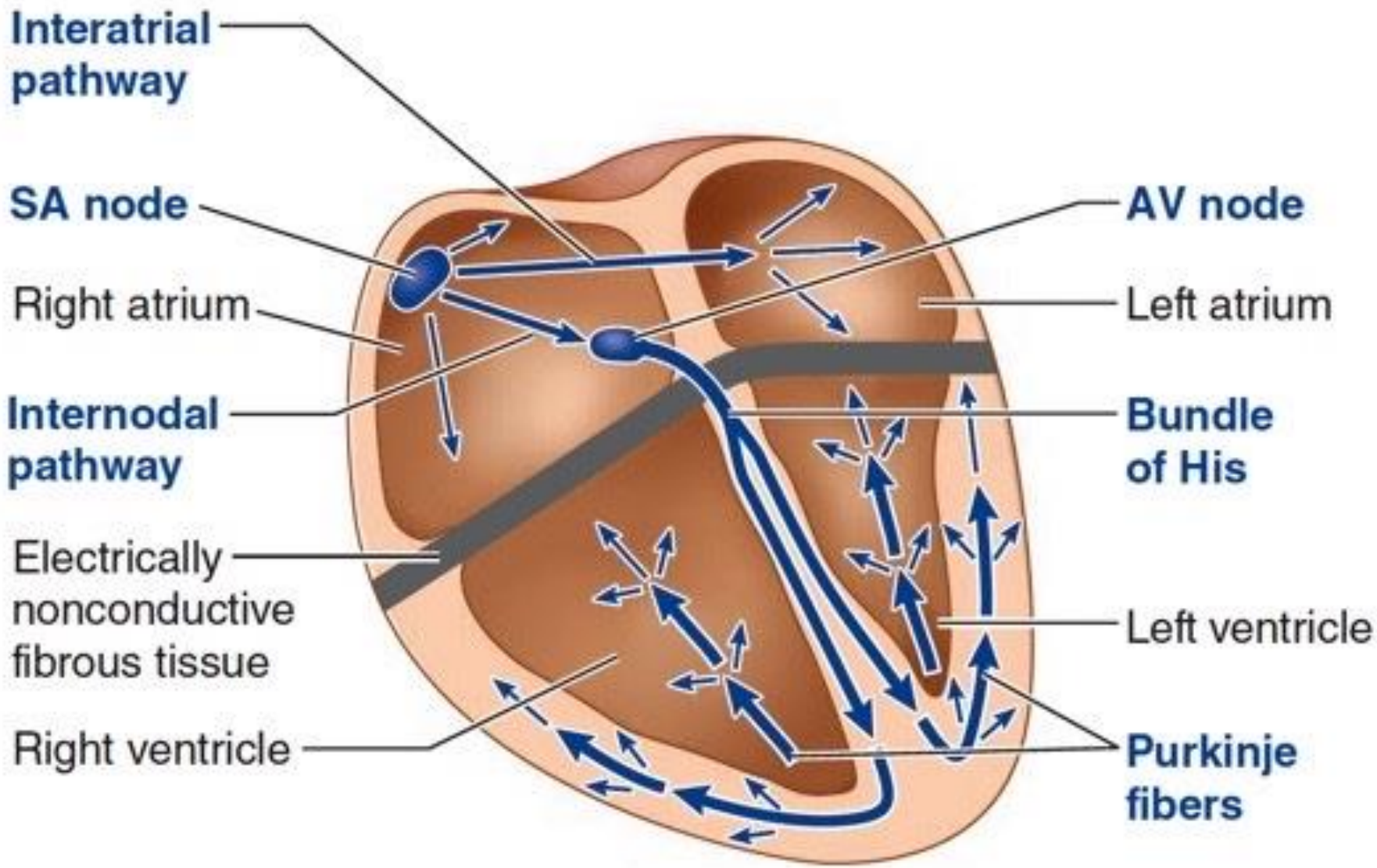
Excitatory and conductive systems of the heart

The conduction system

- a network of specialized cardiac muscle fibers called autorhythmic fibers because they are self-excitable.
- 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.


Interatrial pathway
Sinoatrial (SA) node
Right atrium
Internodal pathway
Right ventricle
Right branch of bundle of His
Atrioventricular (AV) node
Left atrium
Left branch of bundle of His
Left ventricle
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

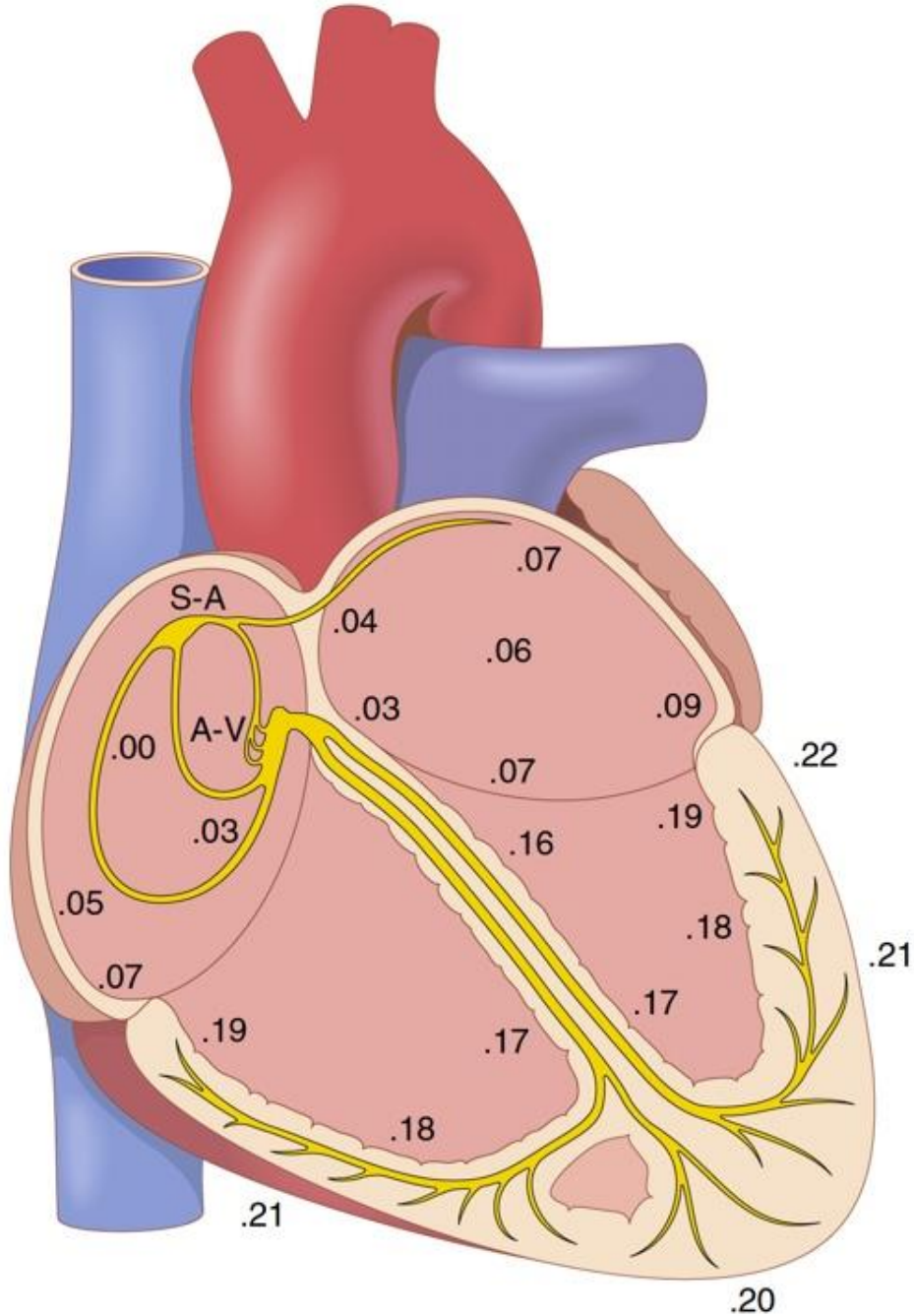
SA node to Atria

- the action potential of SA node spreads through the entire atrial muscle mass and, eventually, to the A-V node.

- 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.

- 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.


S-A
A-V
.07
.04
.06
.03
.09
.00
.07
.22
.19
.03
.16
.05
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.21
.07
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.21
.20

- 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, 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.

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.
- 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.
- 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.
- 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.

- 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 about one-third of the way into the muscle mass and finally become continuous with the cardiac muscle fibers.

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. 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.

- 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?
- 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.

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.

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.

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