THE ELECTROCARDIOGRAPHY (ECG) III

CARDIAC ARRHYTHMIAS

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LECTURE OBJECTIVES:

1. Explain the changes in the function of the sinus node.
2. Understand the disturbances in the conduction of impulses.
3. Describe the ectopic foci

ARRHYTHMIAS

1. Cardiac arrhythmia is the *abnormal rhythm of the heart*. This abnormality is either due to disturbances in impulse initiation or impulse propagation.
2. Disturbances in impulse initiation include those that arise from the SA node and those that originate from various ectopic foci.
3. The principal disturbances in impulse propagation are re-entrant rhythms and conduction blocks.

CAUSES OF CARDIAC ARRHYTHMIAS

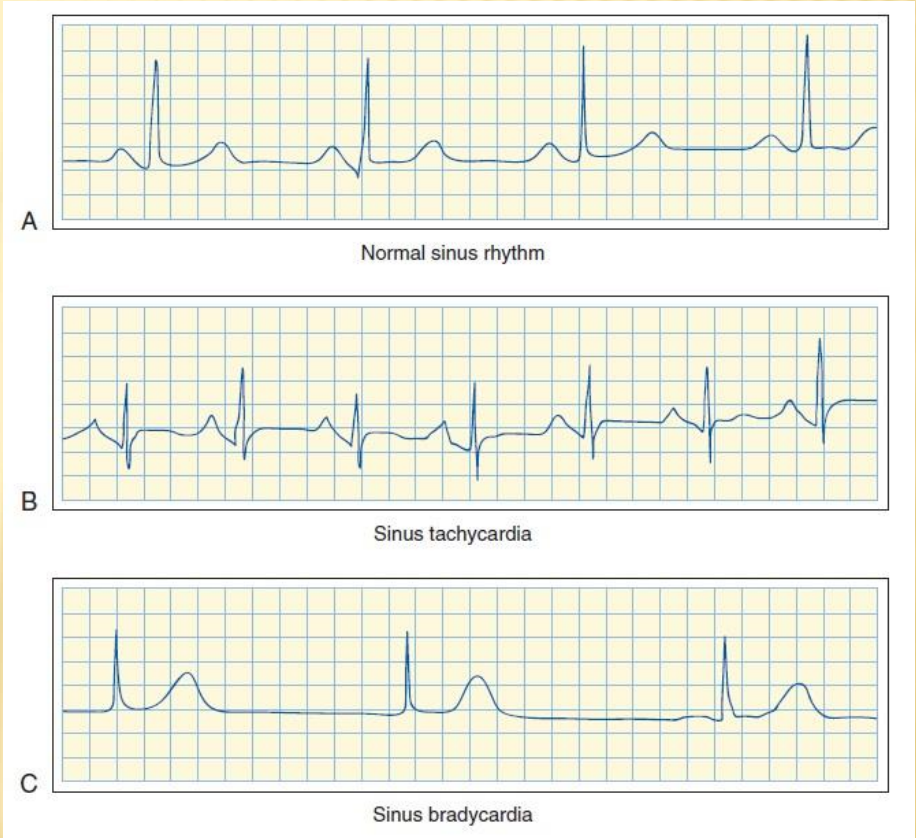
The causes of the cardiac arrhythmias are usually one or a combination of any of the following abnormalities

- Abnormal rhythmicity of the pacemaker
- Shift of the pacemaker from the sinus node to another place in the heart
- Blocks at different points in the spread of the impulse through the heart
- Abnormal pathways of impulse transmission through the heart
- Spontaneous generation of false (unauthentic) impulses in almost any part of the heart

ALTERED SINOATRIAL RHYTHMS

Abnormal sinus rhythms:

1. Sinus tachycardia: when heart rate **exceeds 100** beats/min in an adult. (example; fever, sympathetic stimulation, toxic conditions of the heart, etc.)
2. Sinus bradycardia: the heart slows **below 60** beats/min. This can be a normal physiological finding in **well-trained athletes**. Abnormal bradycardia can be due to over stimulation of the vagus in patients with ***carotid sinus syndrome***.



RESPIRATORY SINUS ARRHYTHMIA (RSA)

Respiratory sinus arrhythmia (RSA) is typically a benign, normal variation in heart rate that occurs during each breathing cycle. *The heart rate increases during inspiration and decreases during expiration*. Normally the heart rate increases and decreases no more than 5 percent during quiet respiration.

There are TWO physiological explanations for the RSA;

1. During inspiration blood flow to the heart increases, this in turn triggers atrial baroreceptors which act to diminish vagal tone. This causes an increase in heart rate.

   During expiration the diaphragm relaxes, moving upward, causing an increase in intrathoracic pressure. This increase in pressure inhibits venous return to the heart resulting in both reduced atrial expansion and reduced activation of baroreceptors. This relieves the suppression of vagal tone and leads to a decreased heart rate.

2. Respiratory sinus arrhythmia may result from “spillover” of signals from the medullary respiratory center into the adjacent vasomotor center during inspiratory and expiratory cycles of respiration. The spillover signals cause an alternate increase and decrease in the number of impulses transmitted through the sympathetic and vagus nerves to the heart.

ATRIOVENTRICULAR CONDUCTION BLOCKS

First-Degree Heart Block:

The PR interval increases to greater than 0.20 second.

Second-Degree Heart Block:

There are two types of second-degree AV block:

1. Type I (also known as *Wenckebach periodicity*) is characterized by progressive prolongation of the PR interval until a ventricular beat is dropped. In most cases, this type of block is benign and no specific treatment is needed.
2. Type II. There is usually a fixed number of nonconducted P waves for every QRS complex. For example a 2 : 1 block. Patient may require implantation of an *artificial pacemaker.*

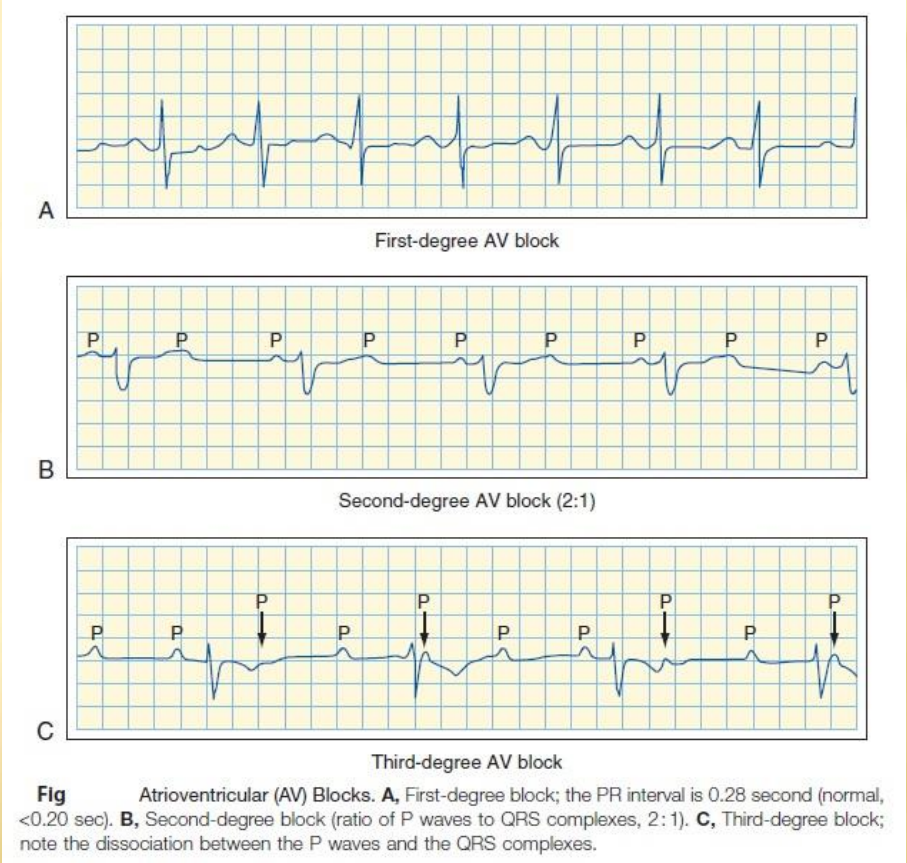


Fig Atrioventricular (AV) Blocks. **A,** First-degree block; the PR interval is 0.28 second (normal, <0.20 sec). **B,** Second-degree block (ratio of P waves to QRS complexes, 2:1). **C,** Third-degree block; note the dissociation between the P waves and the QRS complexes.

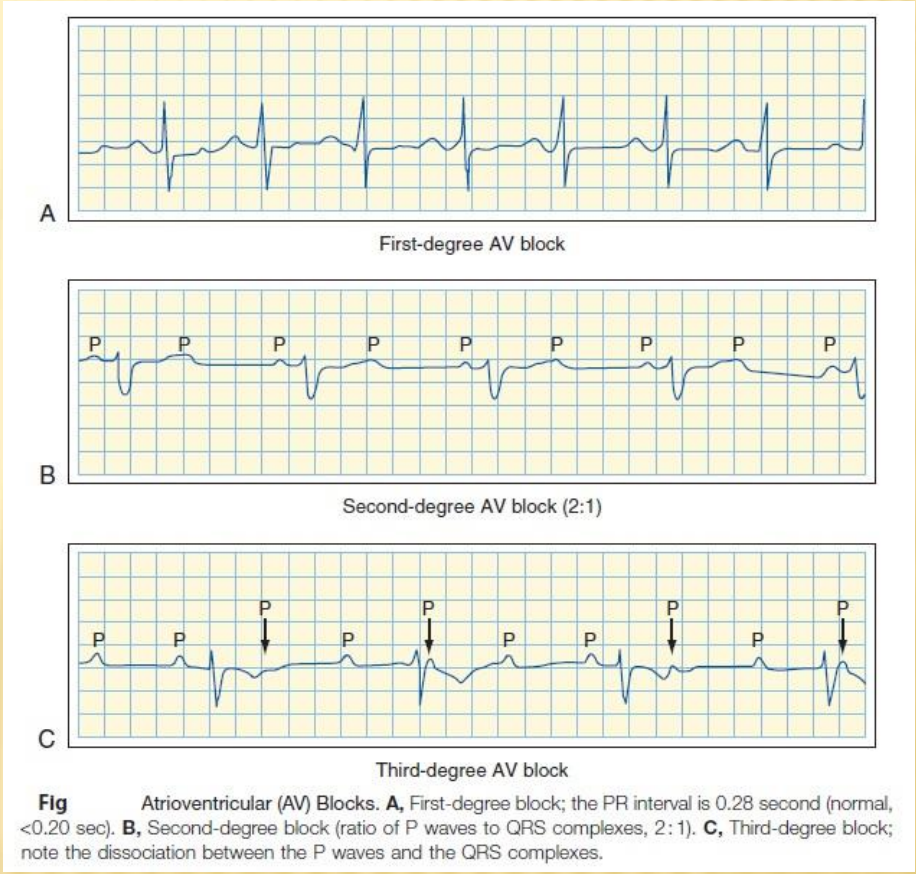
ATRIOVENTRICULAR CONDUCTION BLOCKS (CONT.)

Complete AV Block (Third-Degree Heart Block):

There is a complete block of the impulse from the atria into the ventricles. In this case, the ventricles spontaneously establish their own signal, usually originating in the AV node or AV bundle distal to the block.

There is no relation between the rhythm of the P waves and that of the QRS-T complexes because the ventricles have “escaped” from control by the atria and are beating at their own natural rate.

Most of these patients need to implant an *artificial pacemaker*. The pacemaker provides continued rhythmical impulses to the ventricles.



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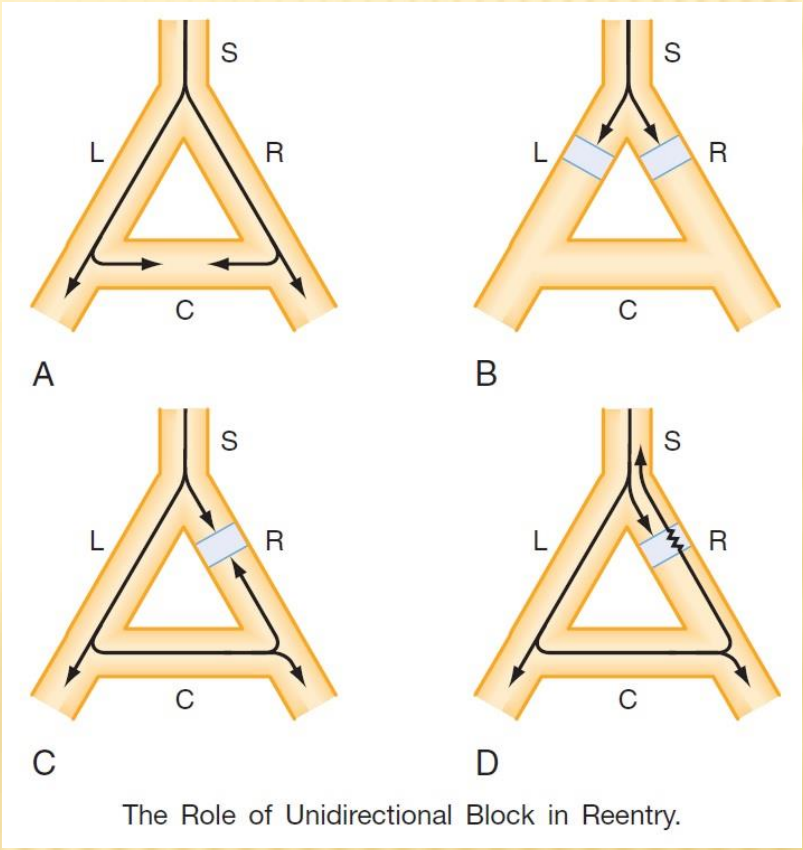
REENTRY MECHANISM

A, An excitation wave traveling down a single bundle (S) of fibers continues down the left (L) and right (R) branches. The depolarization wave enters the connecting branch (C) from both ends and is extinguished at the zone of collision.

B, The wave is blocked in the L and R branches.

C, A bidirectional block exists in the R branch.

D, A unidirectional block exists in the R branch. The antegrade impulse is blocked, but the retrograde impulse is conducted through and reenters the S bundle.



The Role of Unidirectional Block in Reentry.

Note:

For reentry to occur, the effective refractory period of the reentered region must be shorter than the conduction time around the loop. Therefore, the conditions that promote reentry are those that prolong the conduction time or shorten the effective refractory period.

PREMATURE DEPOLARIZATIONS

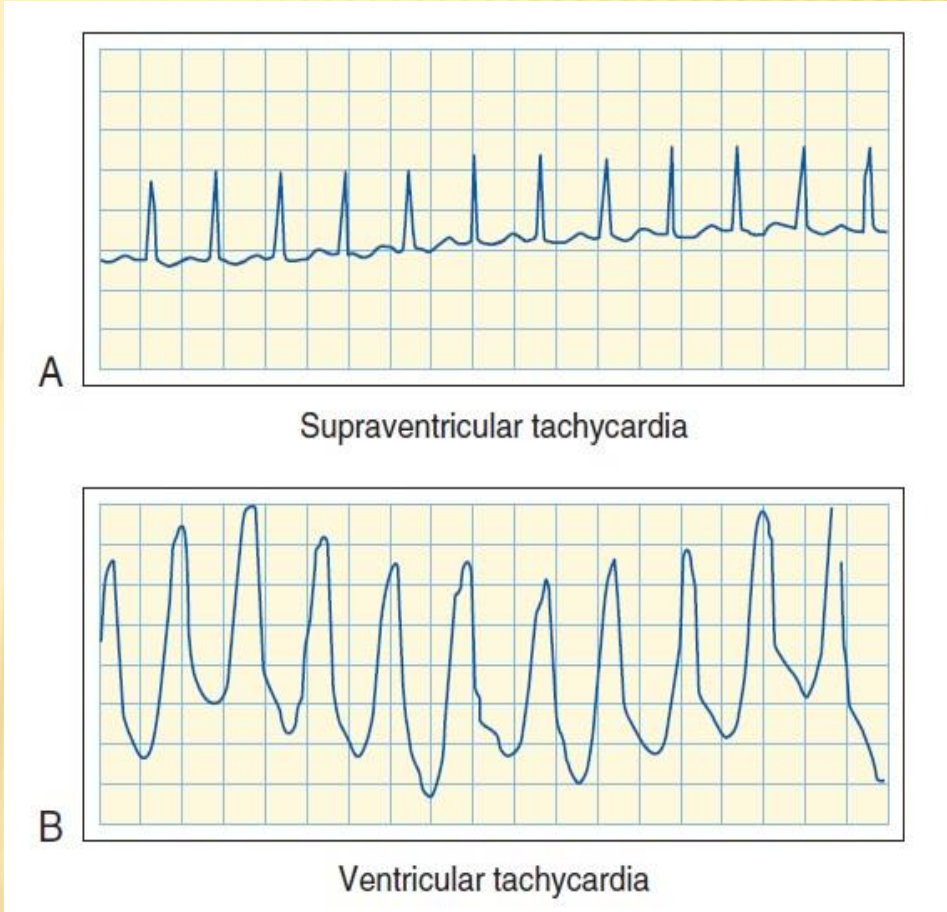
(**A**) Premature Atrial Depolarization. The premature atrial depolarization (second beat) is characterized by an inverted P wave (just below the second “P”) and normal QRS complexes and T waves. The interval after the premature atrial depolarization is not much longer than the usual interval between beats. The brief rectangular deflection just before the last atrial depolarization is a standardization signal.

(**B**) The premature ventricular depolarization is characterized by bizarre, inverted QRS complexes and elevated T waves and is followed by a compensatory pause.

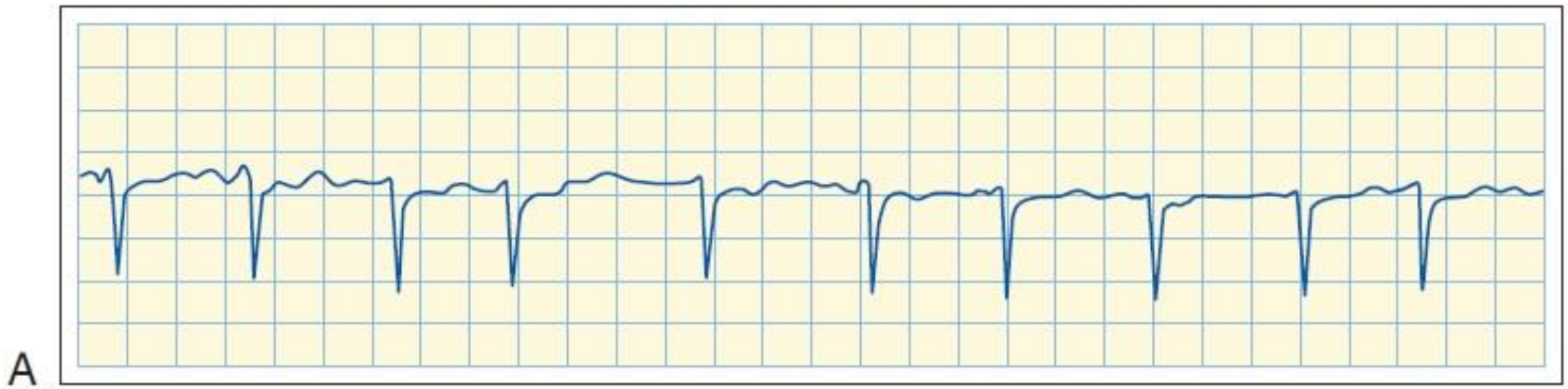
ECTOPIC TACHYCARDIA:

This phenomenon is believed to be caused most frequently by re-entrant "**circus movement**". Because of the rapid rhythm in the irritable focus, *this focus becomes the pacemaker of the heart*.

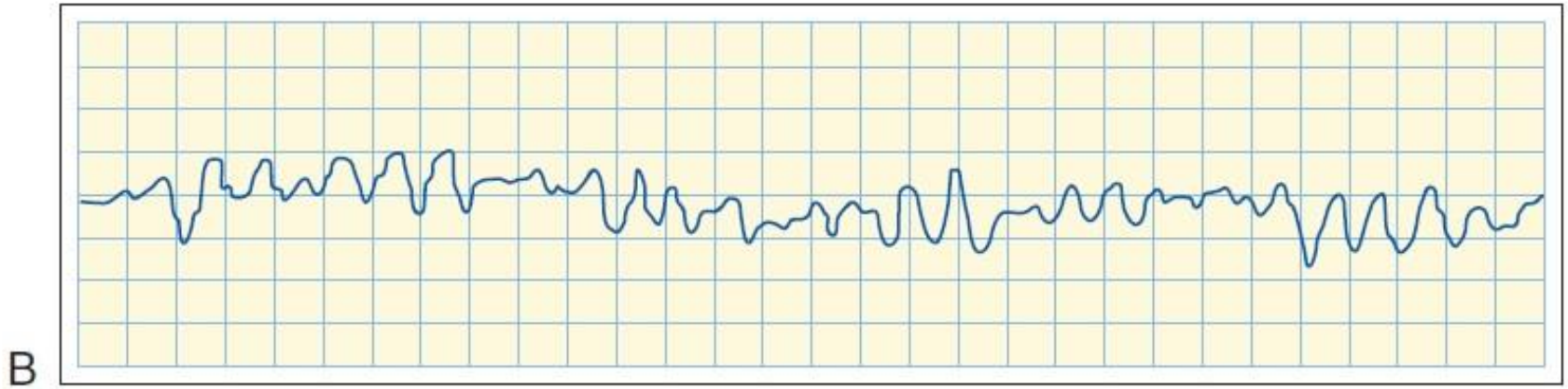
The attack of tachycardia comes in the form of **paroxysm** (i.e. sudden attack). The paroxysm usually ends as suddenly as it began, with the pacemaker of the heart instantly shifting back to the sinus node. The paroxysm may last for a few seconds, a few minutes, a few hours, or even much longer.



FIBRILLATION:



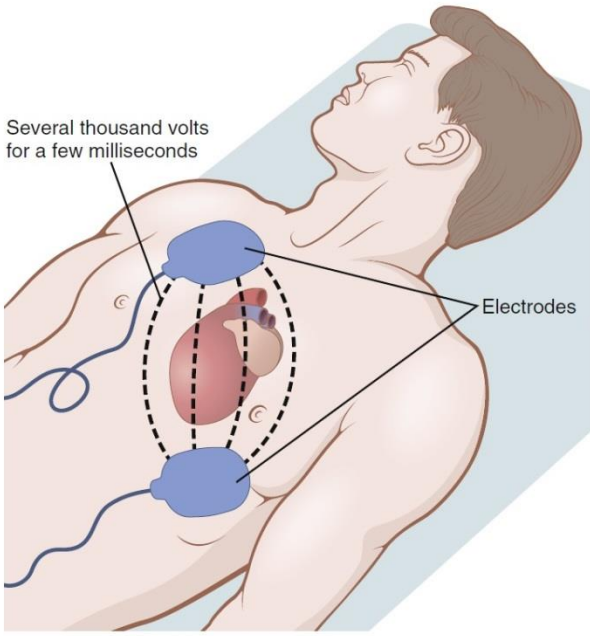
Atrial fibrillation



Ventricular fibrillation

VENTRICULAR DEFIBRILLATION:

- A procedure in which a strong high-voltage electrical current is passed through the ventricles.
- A **direct current** with 1000 volts is applied for a few thousandths of a second.
- In most cases, defibrillation current is delivered to the heart in **biphasic waveforms** (alternating the direction of the current pulse through the heart).
- Such current can stop fibrillation by throwing all the ventricular muscle into simultaneous refractoriness.
- The biphasic waveforms current reduces the threshold needed for successful defibrillation, thereby decreasing the risk for burns and cardiac damage.
- All action potentials stop, and the heart remains quiescent for 3 to 5 seconds, after which it begins to beat again, usually with the SA node or some other part of the heart becoming the pacemaker.



Application of electrical current to the chest to stop ventricular fibrillation.

TEST QUESTION

Q. P wave of ECG is absent in?

A. Atrial hypertrophy.

B. Ventricular extrasystole.

C. Heart failure.

D. Supraventricular tachycardia.

E. First degree heart block.