IV. BRADYCARDIAS (slow heartbeats)

A. Abnormal impulse formation

As noted, the normal heartbeat has its beginnings in the sinus node; the maximum rate of discharge of the sinus node (i.e., maximum heart rate) decreases with age following this relation:

[Maximum sinus heart rate = 225 - (age in years)].

Thus, we do slow down as we age; some slow down more than others, and *sinus* node *dysfunction* manifested as inappropriate *sinus* bradycardia is a major cause of fatigue, weakness, and syncope (transient loss of consciousness) in the elderly. Other manifestations of sinus node dysfunction include the *brady-tachy syndrome*, in which tachycardias, such as atrial fibrillation or flutter, bombard the sinus node, suppressing its function; when the atrial fibrillation ceases, the sinus node may be abnormally sluggish in recovering from this suppression and take several seconds to resume functioning. If there is no His bundle ("junctional") escape beat to support the circulation, syncope can result. Medications (ß-adrenergic blocking agents, Ca^{++} channel blocking agents, digitalis, antiarrhythmic medications) can slow the sinus rate dramatically.

B. Abnormal impulse transmission (AV block)

Once impulses are successfully formed in the sinus node, they practically always make it through the atria to the AV node, but don't always reach the ventricles from this point. Difficulties in transmitting between atrium and ventricle are called *heart block*; several types of heart block have been described.

1) First degree (1°) AV block (AVB) - *delayed* conduction

This is a misnomer since there is really no "block"; all impulses do get to the ventricles, but with a longer-than-normal PR interval (>200 ms). The most common location for 1° AVB to occur is in the AV node, associated with an increased AH interval; in some cases the HPS is responsible (yielding a long HV interval). Medications (particularly ß- and Ca^{++} blockers) can slow AV nodal conduction enough to produce 1° AVB.

2) Second degree (2°) AVB - *intermittent* conduction

The hallmark of 2° AVB is the occasional lack of AV conduction. This takes two forms, named after Mobitz (an early descriptor):

a) Mobitz Type 1 2° AVB - gradual increase PR interval--> non-conducted P wave--resumption of conduction for another cycle, most often with a 3:2 or 4:3 ratio of P's to QRS's. Usually known as *Wenckebach* block, this type of AVB is

relatively common and is almost always localized to the AVN. Accordingly, it is less serious than block at a lower level, since if conduction through the AVN fails entirely, the discharge rate of the subsidiary pacemaker (His bundle) is usually sufficient to provide adequate cardiac output.

b) Mobitz Type II 2° AVB - constant PR interval until a P wave abruptly fails to conduct; then resumes conducting until "dropping" another QRS. This type of AVB is less common than Type I, but more serious; the block is in the HPS and if conduction fails entirely, the rate of the escape focus (lower HPS) is less reliable and may not support the circulation

3) Third degree (3°) AVB - no conduction

There is no communication between atria and ventricles, each beating at their own rate (ventricles always slower, driven by an escape focus in the HPS).

C. Treatment strategies

Bradycardia can be effectively treated in 1 of 2 ways: removing the cause of the bradycardia (sinus or heart block) by altering medications, or by implanting a permanent electronic pacemaker. These devices have been around since the late 1950s and are quite sophisticated now; they can not only pace the ventricle but also the atrium, respond to intrinsic cardiac electrical activity by following each P wave with a paced QRS, and even record and telemeter electrical signals from the heart, tell how much of the time the heart is paced, and modulate the pacing rate to adjust for metabolic needs. With all these advantages, why doesn't everyone have one? They are relatively costly, have their own set of "side effects", and have a limited (5-12 years) life span. Specific guidelines exist for determining whether a particular patient needs a pacemaker.