AV block and AV dissociation

Dr Mazdak Khalili
Cardiologist
Electrophysiologist
AV dissociation denotes a situation where by ventricular activation is elicited by a nonatrial source, and ventricular activity is uninfluenced by atrial activity.

AV block denotes pathologic conduction of an atrial impulse to the ventricle, this may occur within the atrium, AV node, and/or HIS-purkinje system.
Atrioventricular block

Subclassification:
- first degree
- third degree block
- second degree
  - Type I
  - Type II
  - 2:1 AVB
  - paroxysmal AV block
  - advanced AV block.
First degree AV block

First degree AV block is defined by a PR interval in excess of 200 msec following a normally timed (nonpremature) P wave.

Sites of AV conduction delay that can result in first-degree AVB include atrium (intraatrial delay), AV node, and HIS-purkinje system.

AV node involved in more than 80% of adults.
A very long (e.g. >300 msec) PR interval generally denotes involvement of the AV node, as does significant variability in the duration of the PR interval.

If QRS duration is normal, the sole site of block is highly likely (90%) to be AV node.

If QRS duration is prolonged, then the AV node remains a likely participant but abnormalities of intra-atrial and HIS-purkinje system also become far more likely (particularly in patients with LBBB or RBBB plus left or right axis deviation).
in the presence of the IVCD, the surface P-R interval does not appear to be any value in selecting those with prolonged H-V interval.
Markedly prolonged $H-V$ interval in the presence of a normal overall $P-R$ interval
P–R interval with a normal H–V interval
**IVCD &very short or long P-R interval**

- IVCD associated with (i.e. <160 msec) makes a prolonged H-V interval (i.e. 100 msec) unlikely
- IVCD associated with a PR interval of >300 msec:
  - means some abnormality of A-V conduction.
Prognosis

- There is no evidence to suggest that isolated first-degree AVB carries an attributable mortalities.
  - Risk of AF $\rightarrow$ higher than normal

- Isolated first degree AV block usually is asymptomatic.

- Marked PR prolongation (>300 msec) can cause symptoms (pacemaker like syndrome)
Second degree AV block

- Subcategorized into:
  - type I and type II block
  - high degree block
  - paroxysmal block
Type I block

- Characterized by the Wenckebach phenomenon: progressive PR prolongation prior to and leading up to nonconducted P wave.

- The P wave immediately after the nonconducted P wave is followed by a shorter PR interval.

- Magnitude of the pause between the two QRS complexes encompassing the nonconducted P wave will be less than twice the P-P interval.
Type I block

The increment in the PR interval between the first and second beat of a cycle is the largest, in subsequent beats the increments progressively decrease (a pattern of gradual RR interval shortening prior to the blocked P wave).
Type I AV block
Type I AV block
Type 2 second degree AV block in the AV node
Type I AV block can develop in the AV node, HIS bundle, or the conduction system distal to the HIS bundle.

AV node remains a common site of block in the presence of IVCD although involvement of the HIS-purkinje system is also common.

Compared with AV nodal block, type I block localized to the HIS-bundle or distal to the HIS bundle is associated with a shorter base-line PR interval and smaller PR increments preceding the block.
Similar to first degree AV block, type I AV nodal block is relatively common in otherwise healthy adults particularly aerobic athletes and during sleep.

Type I AV nodal block in patients with structurally normal hearts: benign prognosis, seldom progresses to higher degree block.
Increased vagal tone

- Pain, carotid sinus massage or hypersensitive carotid sinus syndrome can have a strong vagotonic effect that results in slowing of the sinus rate or the development of AV block.

- Vagal tone can be high during sleep and sufficient to produce a Wenckebach type AV block.
Exercise and AVB

In the rare case where sympathetic tone (e.g., exercise) initiates or exacerbates type I block, an infranodal location of the block should be considered, even in patients without apparent IVCD.
**Type II block**

- ECG: abrupt failure of a P wave to be followed by a QRS complex.
- Magnitude of the pause between the two QRS complexes encompassing the nonconducted P wave will be twice the P-P interval.
- It is always associated with IVCD.
- Although cases of intra-HIS bundle block have reported in which a narrow QRS complex was present, apparent type II block in this setting is more likely to be an atypical form of type I block.
Type II AV block
Type II second degree AV block in the HIS-purkinje system
**Type II block**

- Anatomical sites of block: HIS bundle or distal conduction system.
- Site of type II block in patients with BBB: block in the contralateral bundle branch.
Type II block can be mimicked by: atypical type I block, concealed extrasystoles, arising from the HIS-purkinje systems, junctional parasystole.
Pseudo-type II block secondary to a concealed his bundle depolarization
**prognosis**

- Type II block is usually associated with significant symptoms.
- It frequently progress to the third-degree block in which the subsidiary (escape) pacemaker is unreliable.
2:1 AVB
baseline
Before CSM
After atropine injection
After CSM
Advanced block

- Failure of more than 2 consecutive P waves to conduct to the ventricle.
Advanced AV block
**Paroxysmal block**

- Sudden failure of repetitive (nontachycardia) P wave to conduct to the ventricle in the setting of otherwise normal AV conduction.
- Episodes are commonly associated with prolonged periods of ventricular asystole.
- Block is distal to the HIS bundle and that escape pacemaker is unreliable.
- Paroxysmal AV block, in adults can sometimes be due to overdrive suppression.
Third degree AV block

- Dissociated atrioventricular depolarizations.
- QRS morphology and cycle length are dictated by the exact location of the escape focus.
- Site of block: AV node, HIS bundle, Purkinje systems.
- Atrial pacemaker can be sinus or ectopic (AF, AFL, AT).
- Wide QRS complex: location in vast majority of cases is within or distal to the HIS-Purkinje system.
- Narrow QRS complex: location is approximately evenly divided between AV node and HIS bundle.
Etiology of AV block

1. Fibrosis and sclerosis
2. Ischemic heart disease
3. Drugs (B-blockers, digoxin, calcium-blocking drugs, amiodarone, procainamide, class 1c)
4. Increased vagal tone
5. Valvular heart disease
6. Cardiomyopathies and infiltrative disease
7. Myocarditis
8. Infective endocarditis
9. Trauma
10. Congenital heart disease
11. Electrolyte imbalance
The most common cause of CHB is idiopathic and related to aging.

Complete AV nodal block is generally congenital, exhibits normal QRS complexes and rates of 40 to 60 bpm.

HIS-purkinje level block is almost always acquired.

Ventricular rate in acquired CHB is less than 40 bpm.
In patients with AV nodal block, Atropin usually speeds both the atrial and the ventricular rates. Exercise can reduce the extent of AV nodal block.
**Short term therapy of CHB**

- Atropin for AV nodal block
- Isoprotrenol for AV block at any site.
- The use of trascutaneous pacing is preferable for temporary pacing
- TPM is preferable for pacing mor than several hours/days.
Ventricular rate usually is regular.

Ventricular rates can vary in response to: PVC, shift in pacemaker site, an irregularly discharging pacemaker focus, or autonomic influences.
What is the most common cause of CHB with narrow QRS complex in the elderly?

Involvement of mitral ring or the central fibrous body by sclerosis
# Indications for permanent pacing in AV block

## Table 31-2: Indications for Permanent Pacing in Atrioventricular Block

<table>
<thead>
<tr>
<th>Type of AV Block</th>
<th>Pacemaker Necessary</th>
<th>Pacemaker Probably Necessary</th>
<th>Pacemaker not Necessary</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Third</strong></td>
<td>Symptomatic congenital CHB</td>
<td>Asymptomatic, type I, at infra-His or infra-His level*</td>
<td>AV block of any degree that is expected to resolve and unlikely to recur, e.g., drug toxicity, Lyme disease, sleep apnea, without treatment</td>
</tr>
<tr>
<td></td>
<td>Acquired symptomatic AV block</td>
<td>Hemodynamically symptomatic due to loss of AV synchrony</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Atrial fibrillation with CHB</td>
<td>Neuromuscular diseases with AV block, with or without symptoms</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Acquired asymptomatic CHB</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Neuromuscular diseases with AV block, with or without symptoms</td>
<td>Asymptomatic, type I, at supra-His (AV node) level</td>
<td></td>
</tr>
<tr>
<td><strong>Second</strong></td>
<td>Symptomatic second-degree AV block regardless of type</td>
<td>Hemodynamically symptomatic due to loss of AV synchrony</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Asymptomatic advanced second-degree AV block with asystole ≥ 3.0 sec or escape rate &lt; 40 beats/min in awake patients</td>
<td>Neuromuscular diseases with AV block, with or without symptoms</td>
<td></td>
</tr>
<tr>
<td><strong>First</strong></td>
<td></td>
<td>Hemodynamically symptomatic due to loss of AV synchrony with markedly prolonged PR interval, e.g., &gt; 300 msec</td>
<td>Asymptomatic</td>
</tr>
</tbody>
</table>

AV = atrioventricular; CHB = complete heart block.

Intrinsic conduction system of the heart

- Sinoatrial node
- Right atrium
- Atrioventricular node
- Right ventricle
- Left atrium
- Left ventricle

Bundle of His
The three bundle branches comprise the Trifascicular system.
Congenital AV block in children
CCHB with stable Junctional escape
Congenital CHB with Rate < 50 bpm
Congenital CHB
In children, the most common cause of AV block is congenital.

Congenital CHB may be an isolated finding or be associated with other lesion.
**Prevalence**

- Congenital CHB is an uncommon cardiac defect occurring in 1 of every 15000-20000 live births.
- Associated structural heart disease is present in approximately **30%** of these patients.

With the diagnosis of CCHB **prenatally**, associated congenital heart disease is found in up to **53%** of fetuses.

**Fetal bradycardia** (HR<80bpm) is a manifestation of CCHB.
Causes of CHB in children

- Myocarditis
- Congenitally corrected transposition of great vessels
- Polyspelenia with common AV canal defect
- Familial ASD
- Kearns-sayre syndrome
L-transposition of great arteries are the most common anatomical defect associated with CCHB.

In fetus the most common heart defect is left atrial isomerism.
Isolated CCHB detected at or before birth is strongly associated with maternal autoantibodies directed to SS-A/Ro ribonucleoproteins causing neonatal lupus syndrome.

CCHB diagnosed later in life is not known to be associated with anti-SS-A/Ro antibodies.
In the absence of Congenital heart disease, neonatal lupus is responsible for 60% to 90% of cases of congenital CHB.

Among women with anti-RO/SSA and/or anti-La/SSB antibodies, CHB occurs in approximately 2% of pregnancies.
Anatomy of congenital CHB

- Anatomical disruption between atrium musculature and peripheral part of the conduction system and nodoventricular discontinuity are two common histological findings.
Intrinsic conduction system of the heart

- Sinoatrial node
- Right atrium
- Atrioventricular node
- Left atrium
- Left ventricle
- Right ventricle
Clinical course

- Natural history of CCHB is largely determined by the presence of congenital heart disease and the time of diagnosis.
- Children are most often asymptomatic.
- Mortality is highest in neonatal period, much lower in childhood and adolescence, and increase slowly later in life.
Natural history

- Mortality rate in neonatal period: 50%
- Survival rate in patients with isolated CCHB is 85%
- Mortality rate outside of the neonatal period is significantly lower (3-18%), reflecting higher Junctional escape rates and absence of carditis
Despite the absence of symptoms in childhood, 50% of patients will develop symptoms in adulthood and 10% will die prematurely.
Mitral regurgitation can occur in up to 10% of adults with CCHB and can occur anywhere from the teen years to middle adulthood.

Resolution of mitral insufficiency after pacemaker implantation has been reported to occur only rarely.
- **Adams-stokes attacks** in children can occur with congenital CHB at any age especially in those with **HR<50bpm** and frequent syncope
Congenital CHB with syncope
Predictors of development of symptoms in children with CHB

- Prolonged recovery times of escape foci following rapid pacing
- Slow heart rate on 24-hrs holter monitoring
- Occurrence of paroxysmal tachycardias
Overdrive suppression of automaticity in congenital CHB
A subset of patients with CCHB especially those with implanted PPM early in life has been reported to develop cardiomyopathy with reported incidence of 6%.
Class I:

- CCHB with a **wide QRS escape rhythm**
- Complex ventricular ectopy
- Ventricular **dysfunction**
- In infants with ventricular rate **less than 50 to 55 bpm** or with congenital heart disease and ventricular rate less than 70 bpm
- Sustained pause-dependent VT with or without prolonged QT, in which the efficacy of pacing is thoroughly documented
Congenital CHB with torsades de pointes
Class IIa

- CCHB beyond the first year of life with an average rate less than 50 bpm, abrupt pause in ventricular rate that are two or three times the basic CL, or associated with symptoms due to chronotropic incompetence
- Long QT syndrome with 2:1 AV block or third degree AV block
Class II b

- CCHB in the asymptomatic neonate, child or adolescent with an acceptable rate, narrow QRS complex, and normal ventricular function
Tank you for your attention
**Lev’s disease:**

- Known as idiopathic bilateral bundle branch fibrosis
- Progressive replacement of the cardiac skeleton and **proximal** bundle branches by fibrosis as a result of the aging process exaggerated by HTN and atherosclerosis
**Lenegre’s disease**

- Idiopathic conduction disorder involving the peripheral part of the bundle branches
- Some causes of lenegre’s disease may be due to mutation in the SCN5a, causing a hereditary form of AV conduction disease
- Predilection for the right bundle branch AND left anterior fascicle.
**AV dissociation**

Electrophysiologic mechanisms:

1. Default of the primary pacemaker: the intrinsic atrial activation rate falls below that of a subsidiary pacemaker (junctional or supernumerary), allowing the latter to produce an escape complex.

2. Isorhythmic: ventricular rate only minimally exceeds that of the atria.

3. CHB

4. Usurps of latent pacemaker: acceleration of subsidiary pacemaker: VT, junctional tachycardia

5. Combination of the above: digitalis toxicity can produce AVB (generally type I) along with a junctional tachycardia.
Isorhythmic AV dissociation
Type II second degree AV block in the HIS-purkinje system
Pseudo-type II block secondary to a concealed his bundle depolarization
First degree AV block
Type I second degree AV block in the AV node
Type I AV block
2:1 AV block proximal (B) and distal to HIS bundle (A)
Congenital CHB
CHB with retrograde VA conduction
VA conduction during ventricular pacing can lead to atrial contraction during ventricular systole (or in cases of long VA conduction, early diastole).

VA conduction has been found in as many as 90% of patients with SSS and in 15% to 35% of individual with AV block.
AF with CHB
Type II AV block
Type I AV block