How to distinguish between paroxysmal intrinsic and extrinsic AVB

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AV block may present as:

- Congenital heart block
- Acquired high-degree or complete heart block
- Vagally mediated AV block (extrinsic)
- Paroxysmal atrioventricular block (intrinsic)

Paroxysmal AV Block (Intrinsic)

- understanding and recognition of paroxysmal AVB is essential, **asystole with SCD,** preventable with **PPM**.
- First recognized 1993 as paroxysmal AVB **precipitated by premature atrial beats**.
- unique disorder of **diseased HPS**,
- Secondary to **local phase 4 block** in the His bundle or in the bundle branches after a critical change in the H-H interval.

Paroxysmal AV Block (Intrinsic)

- The hallmark is a **sudden** CHB, **initiated by a pause**.
- Defined as a sudden, **pause-dependent phase 4 AV block** occurring in diseased conduction system.

Epidemiology:

No established prevalence, underreported:

- Poor recognition.
- No marker for AV conduction disease between episode(s).
- Not clearly defined in the guidelines.

Epidemiology

ILR results of 52 **(RBBB) patients with syncope and a negative (EPS)**:

- Sudden **CHB** in 13 of 52 (25%).
- Of the 13 patients, 5 (38%) CHB events were **triggered by APB or VPB**

Epidemiology:

- More frequent in older adults, 26 - 99 years (72% age> 60 years).
- Documented in few pediatric cases
- Equally in women and men

Predictors:

- No established predictors exist.
- **Evidence of distal conduction disease** at baseline is often present,
- with **RBBB** being the most common finding.

Predictors:

- Published series of 68 patients from US & Netherlands:
  - (45%) had **RBBB**,
  - (15%) had **LBBB**,
  - (12%) had **IVCD**,
  - (28%) had **normal QRS**

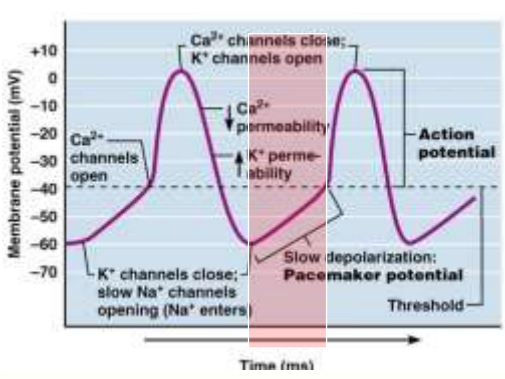
Triggers of paroxysmal AVB:

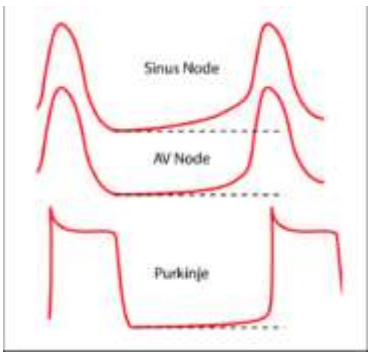
- In a series of 30 patients with PAVB, initiation of PAVB:
  - **PAC** (30%),
  - **PVC** (23%),
  - **His extrasystole** (10%).
  - The remaining (37%) **SVT, CSM, Valsalva maneuver, and spontaneous sinus rate slowing.**

Mechanism of PAVB:

- unique disorder of **diseased HPS**,
- **local phase 4 block** in the His bundle or in the bundle branches after a critical change in the H-H interval.

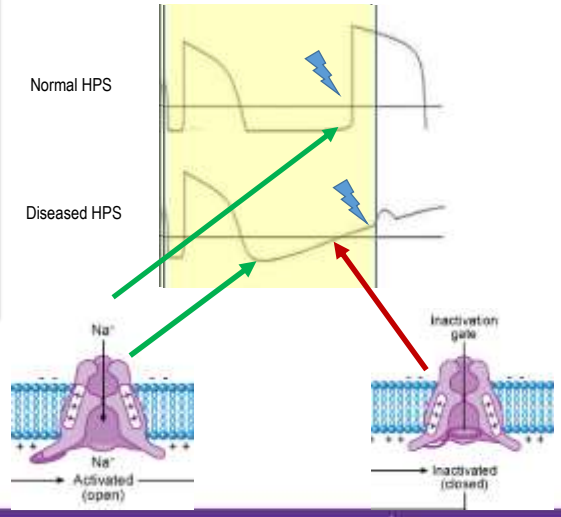
Phase 4 depolarization is a normal property of (SAN) & HPS responsible for automaticity





- **Phase 4 block**:

Impulse reaches diseased(HPS) during phase 4 of AP.
sodium channels are inactive.
impulses can not depolarize the diseased tissue causing asystole.



Mechanism

Response to premature atrial beat in normal HPS versus diseased HPS.

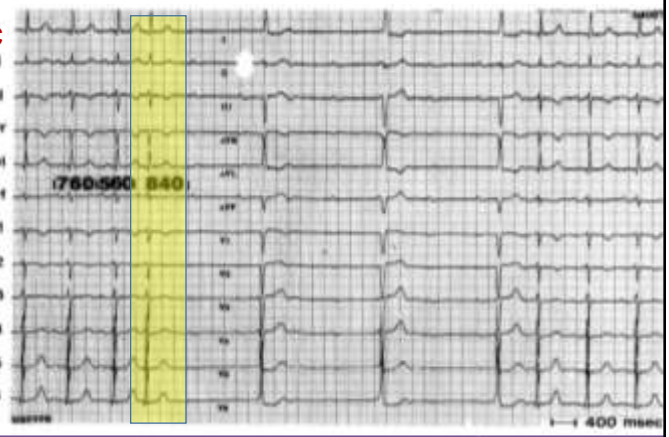
- Conducted **PAC**
- Compensatory **pause** after (longer pp, HH)
- Ventricular **asystole**

Summary of the mechanism:

- **Triggers** for the block (PVC, PAC, termination of SVT) are followed by **pause** (compensatory pause in case of PVC or PAC)
- This pause (long diastolic period) (long HH) initiates a **phase 4 depolarization in the HPS** (HPS cells spontaneously depolarize; Na channel activate and elevate the membrane potential to a less negative value)
- When the **next sinus beat arrives to the HPS, there will not be enough Na channel available** for activation to initiate action potential and **ventricular asystole** occurs.[PHASE 4 BLOCK]
- **Appropriately timed escape beat or premature beat** (sinus or ectopic) can reset the transmembrane potential and terminates the block.

Initiation with a Conducted PAC:

- initiation by a **conducted PAC**
- **Prolonged P-P interval** after an atrial premature beat compared with preceding sinus P-P interval.
- **His bundle escape rhythm**
- **1:1 conduction resumes** near the end of the recording.



Initiation with His extra-systole or non conducted PAC

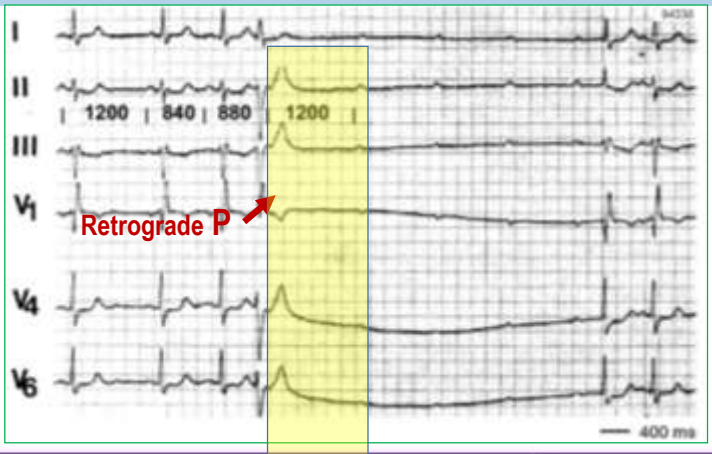
- Initiated by a premature beat (**negative p wave**):
  - likely His extrasystole,
  - Nonconducted PAC
  - Sinus acceleration (shortening P-P) during asystole does not affect the block
  - until an **appropriately timed escape abolishes asystole**

Initiation with Atrial tachycardia termination

- Atrial tachycardia
- terminated with a pause.
- Pause initiated the PAVB
- Asystole is terminated by pacing.

Initiation with PVC

- Baseline RBBB
- VPB with V-A conduction. retrograde conduction (negative P wave)
- PAVB phase 4 block in the left bundle.



Diagnosis of PAVB

- No optimal specific test,
- Normal ECHO and baseline ECG can not rule out the diagnosis

- Holter and loop recorder.
- TT test: low specificity and reproducibility thus not clinically useful

EPS:

- Ajmaline and procainamide test: HV lengthening in all causes of infrahissian block (**not specific for PAVB**)
- Critically times PAC or PVC or rapid Vpacing can reproduce the PAVB (**specific but not sensitive)**

Management of PAVB

- During acute episode, **delivery of PVC (precordial thump)** may be life saving.
- **PPM implantation**, except in the setting of acute reversible cause eg: ACS

PAVB (intrinsic) vs Vagal (extrinsic) AVB

- PAVB precipitate by **slowing of a HR** → a possible mistaken as vagally mediated.
- **Abrupt complete AVB** from high vagal tone → mistaken for PAVB.
- Heigh vagal tone may accentuate the critical pauses needed for phase 4 block. However, it is **not an essential component** of PAVB.

PAVB (intrinsic) vs Vagal (extrinsic) AVB

- Differentiation is important.
- Vagal AVB often benign, **no previous studies showned benefit of PPM**.
- Clear distinction may not always be possible, especially if the AV block episode is short.

Evidence supporting PAVB:

1- initiation with PAC, PVC, His extrasystole.

2- initiation with tachycardia

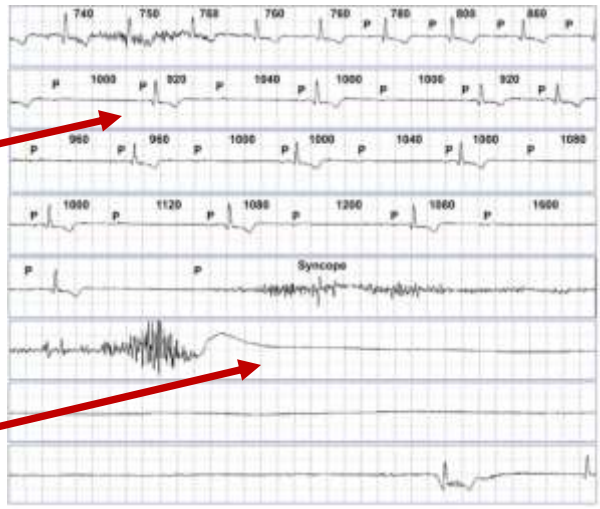
3- sinus acceleration (shortening of P-P interval) during ventricular asystole
without affecting the block.

Evidences supporting Vagally mediated AVB:

History of heightened vagal tone

- during micturition,
- phlebotomy,
- Visceral pain etc

gradual slowing of the sinus rate (P-P lengthening) and AV conduction (prolonging PR),



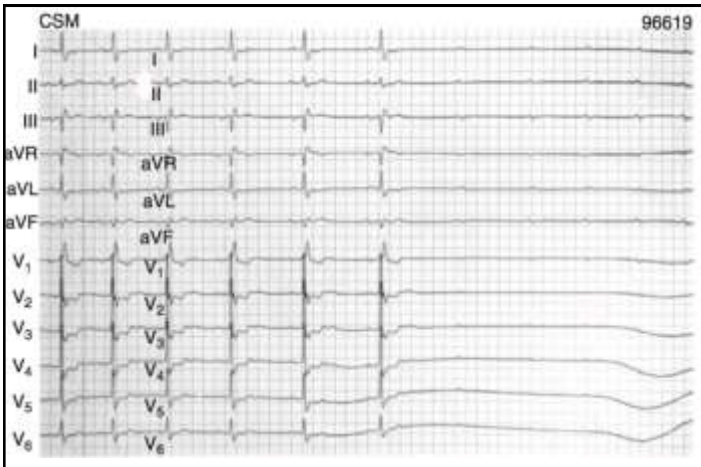
followed by sinus arrest and complete AV block.

However, a **more prominent AV response with sudden block** may also occur with high vagal tone .

As a result, slowing of a heart rate before initiation of AVB and/or sudden development of AVB alone are **neither diagnostic nor specific** for paroxysmal AVB

Findings highly suggestive of vagal AVB:

1. significant PR prolongation or Wenckebach before initiation of AVB
2. Prolonging P-P interval during ventricular asystole
3. resumption of AV conduction on sinus acceleration (shortening of P-P interval)
4. Significant PR prolongation on resumption of AV conduction



CSM in PAVB :

P-P lengthening without changes in preceding PR intervals before Heart block.

Vagal AVB: PR interval prolongs before the AVB.

Summary of differentiation:

	Vagal AVB	Paroxysmal AVB
Level of block	AV nodal	Infra-nodal
Initiated by APB, VPB, HES	No	Yes
Tachycardia before initiation	No	May be seen
Resumption of conduction	Sinus acceleration (P-P shortening) or withdrawal of vagal input	Appropriately timed escape beat, or premature beats (sinus or ectopic APB, VPB, HES)
Initiation		
P-P lengthening	Present	Present but not mandatory
PR prolongation	Present	Typically not present

Thank you

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