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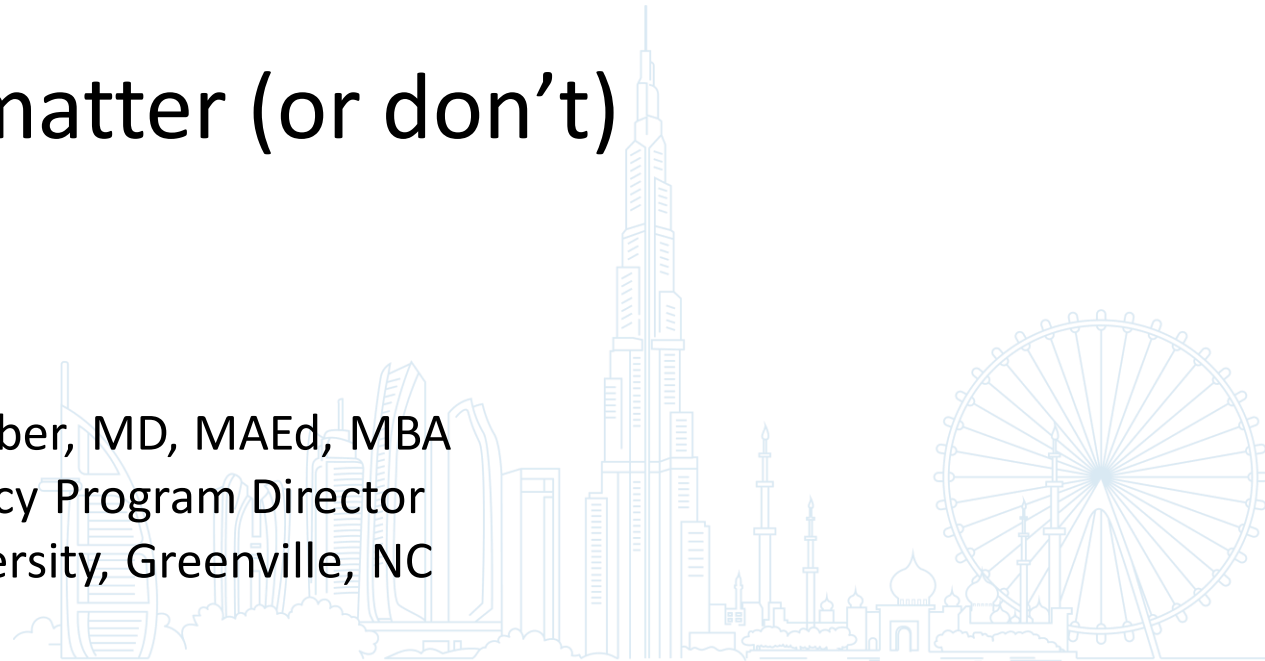
WirediV



Commonly missed things on EKGs

And why they matter (or don't)

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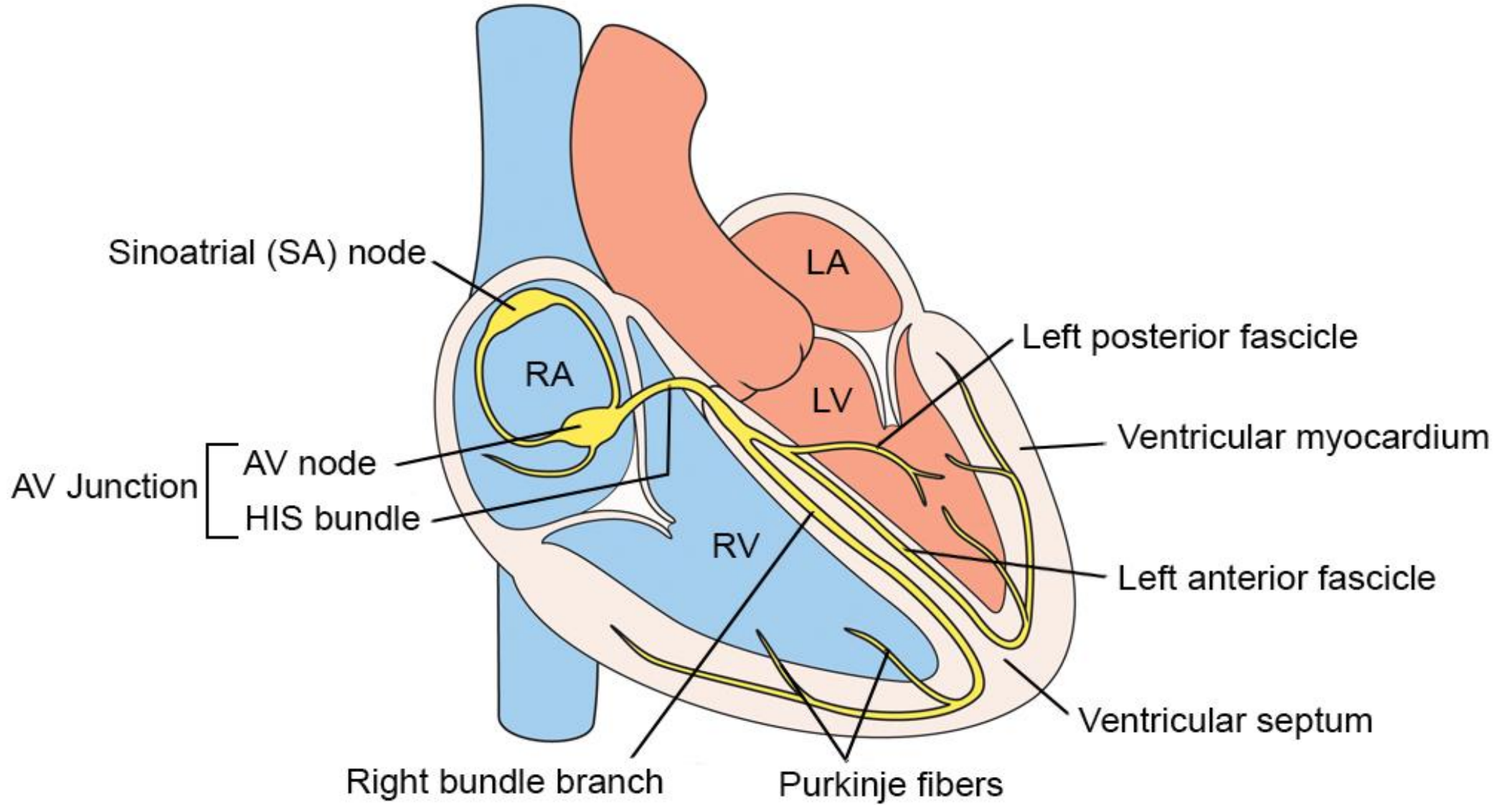




Reading EKGs

- The 10-second proficiency rule.
- Train yourself to cover up (or truly ignore) the machine interpretation
- Seek a mentor.
- Practice, practice, practice.





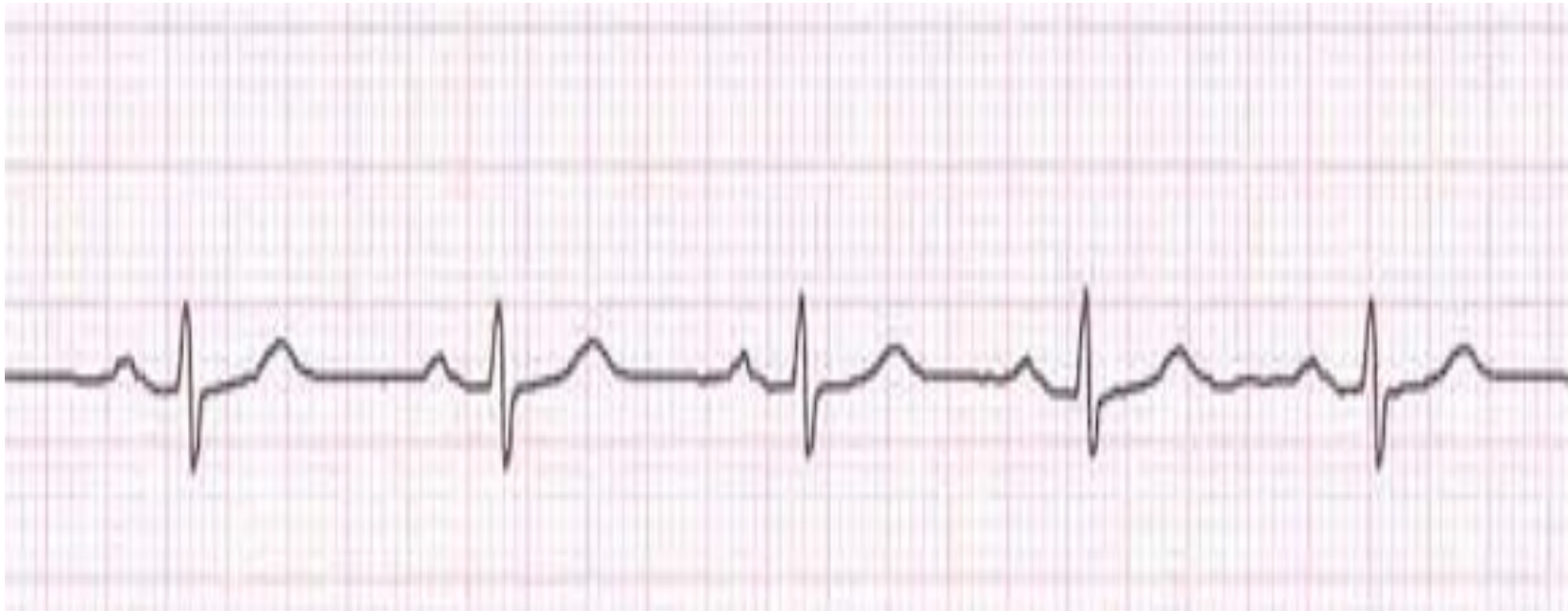


First-degree AV block





First degree AV block



- Diagnosis requires only that the PR interval is longer than 0.20 seconds.
- Conduction delay at AV node or bundle of His.



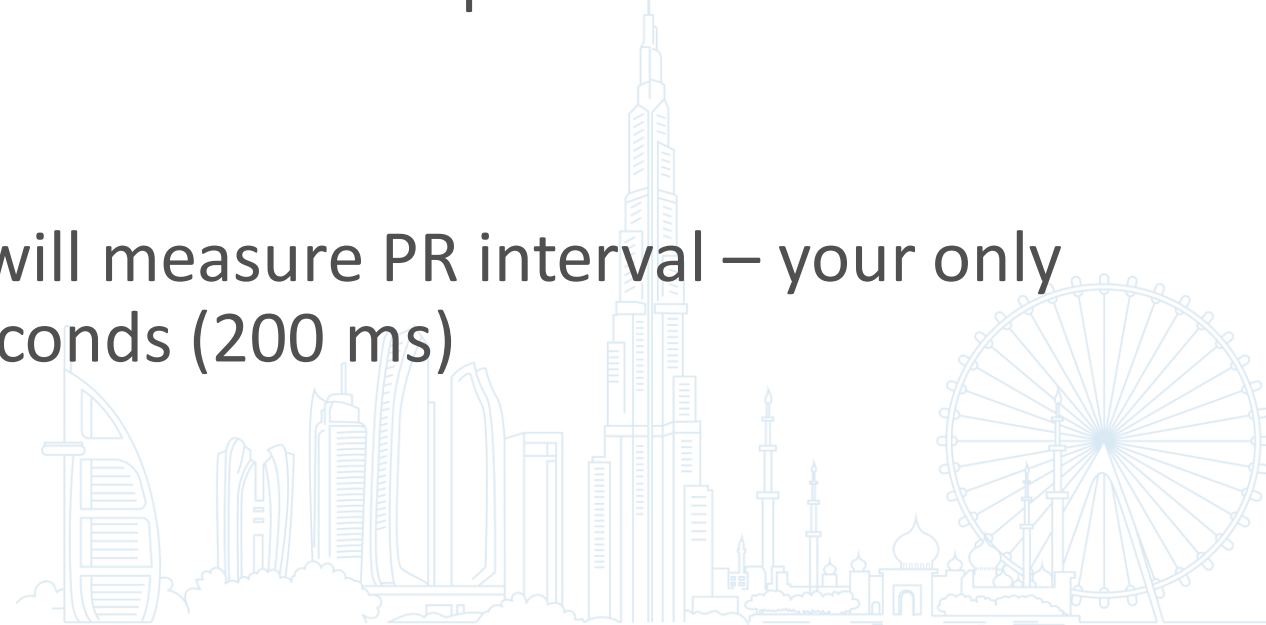


Does it matter?

First degree AV block

- Not much. Avoid unnecessary use of drugs that slow AV conduction (e.g., verapamil, diltiazem, beta-blockers, digoxin, amiodarone) and avoid combinations of these agents. No direct impairment of cardiac function.

Difficulty rating: 1 – the computer will measure PR interval – your only job is to recognize that it's >0.20 seconds (200 ms)





Left atrial abnormality



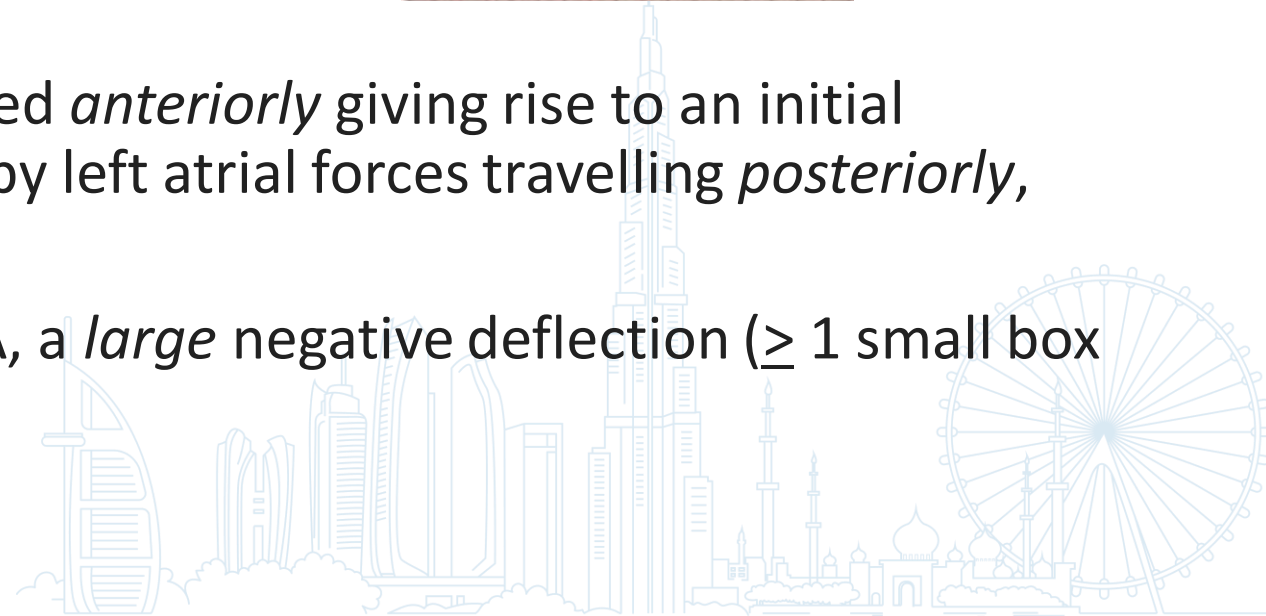
Left atrial abnormality



Lead II:



- In V1: Early right atrial forces are directed *anteriorly* giving rise to an initial *positive* deflection; these are followed by left atrial forces travelling *posteriorly*, producing a later *negative* deflection.
- A biphasic P is common in V1. With LAA, a *large* negative deflection (≥ 1 small box in area) is suggestive of LAA.
- Also may see an M-shaped P in lead II.



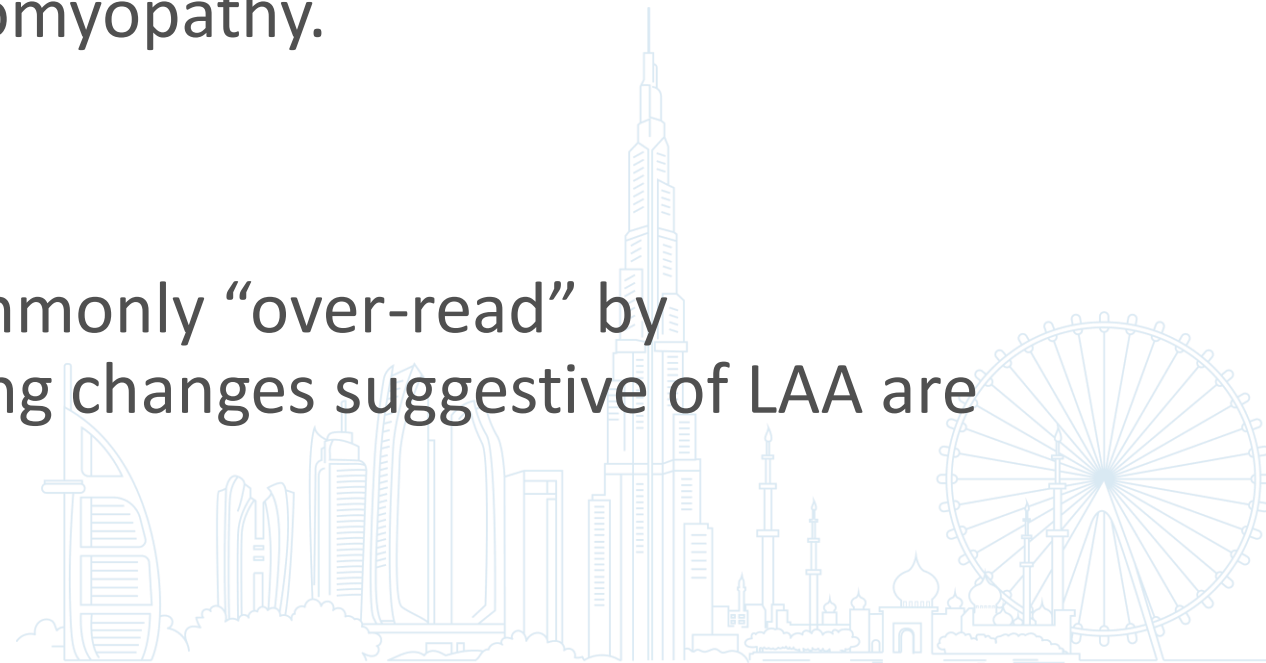


Does it matter?

Left atrial abnormality

- Maybe. Typically goes along with left ventricular hypertrophy, so think of those causes: aortic stenosis, mitral incompetence, systemic hypertension, hypertrophic cardiomyopathy.

Difficulty rating: 3 – this is very commonly “over-read” by interpretation algorithms; convincing changes suggestive of LAA are less common.





Mobitz I Second-degree AV block

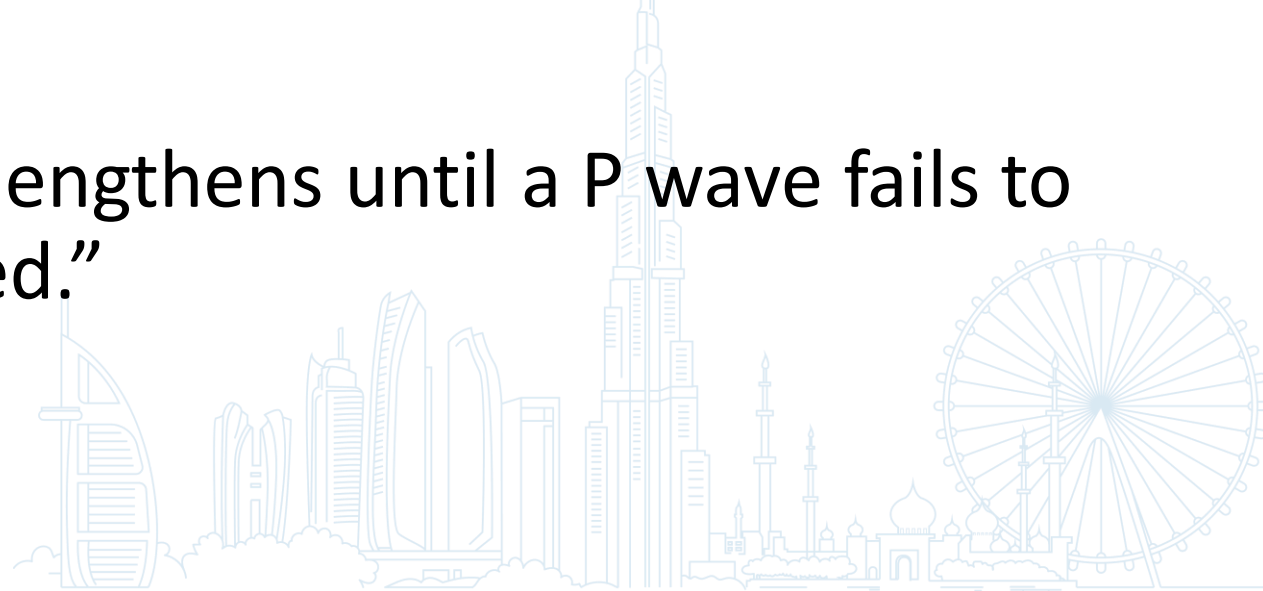




Mobitz type I second-degree AV block (Wenckebach)



- The PR interval progressively lengthens until a P wave fails to conduct and a beat is “dropped.”





Mobitz type I second-degree AV block (Wenckebach)

- Almost always represents disease of the *AV node*.
 - May be seen in athletically fit individuals, especially during sleep.
- In the acute setting, inferior wall ischemia is likely.
 - Inferior wall is supplied by RCA, which also supplies the AV node.
- Treatment: the rhythm itself generally does not require treatment; the underlying cause may.





Does it matter?

Mobitz type I second-degree AV block

- It depends on the context. In most cases, reflects high vagal tone, which is not a worrisome situation.

Difficulty rating: 3 (averaging 1 and 5); some are very straightforward, while others can be really tricky to spot.

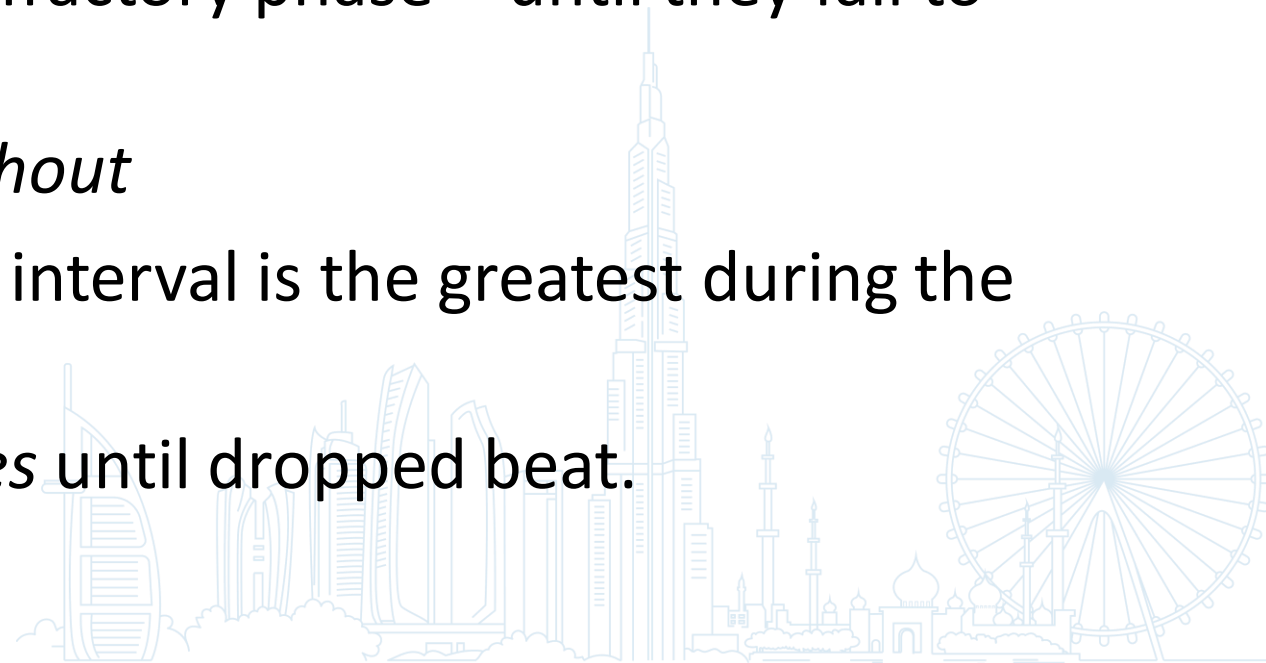




Mobitz type I second-degree AV block

Here is more, if you're curious:

- Malfunctioning AV nodal cells progressively fatigue – basically increasing the duration of their refractory phase – until they fail to conduct an impulse.
- PP interval *stays the same throughout*
- The incremental change in the PR interval is the greatest during the second PR segment.
- RR interval progressively *decreases* until dropped beat.

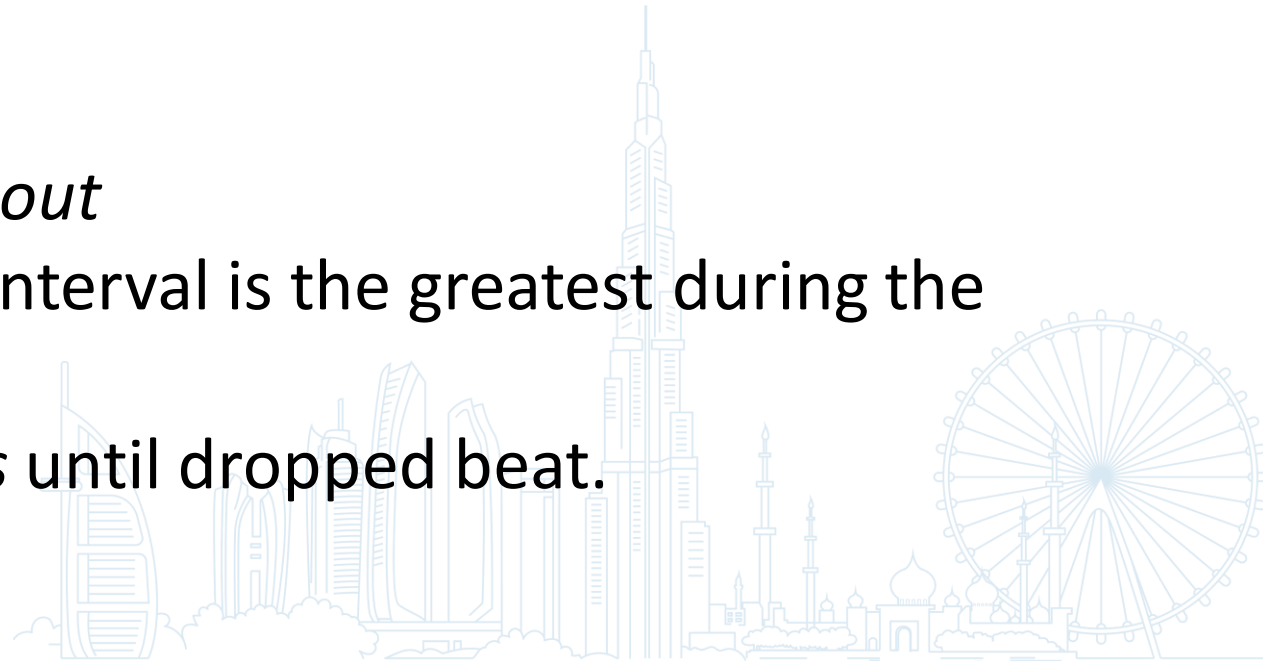




Mobitz type I second-degree AV block



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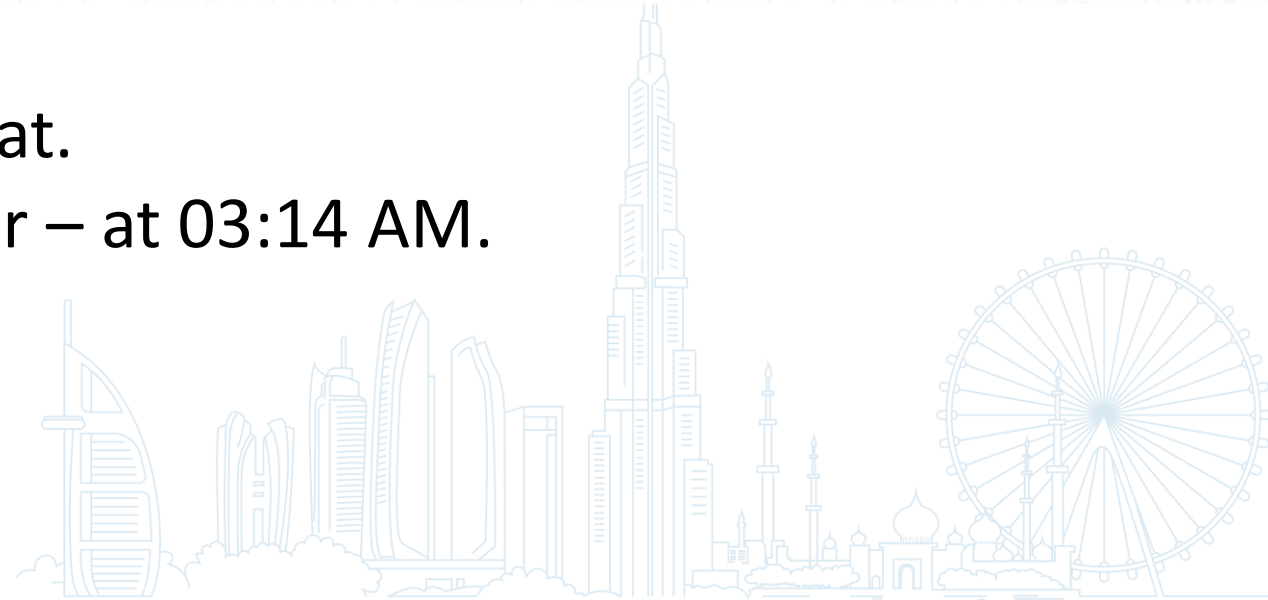




Mobitz type I second-degree AV block



- One more, just because they're neat.
- This was from an extended monitor – at 03:14 AM.

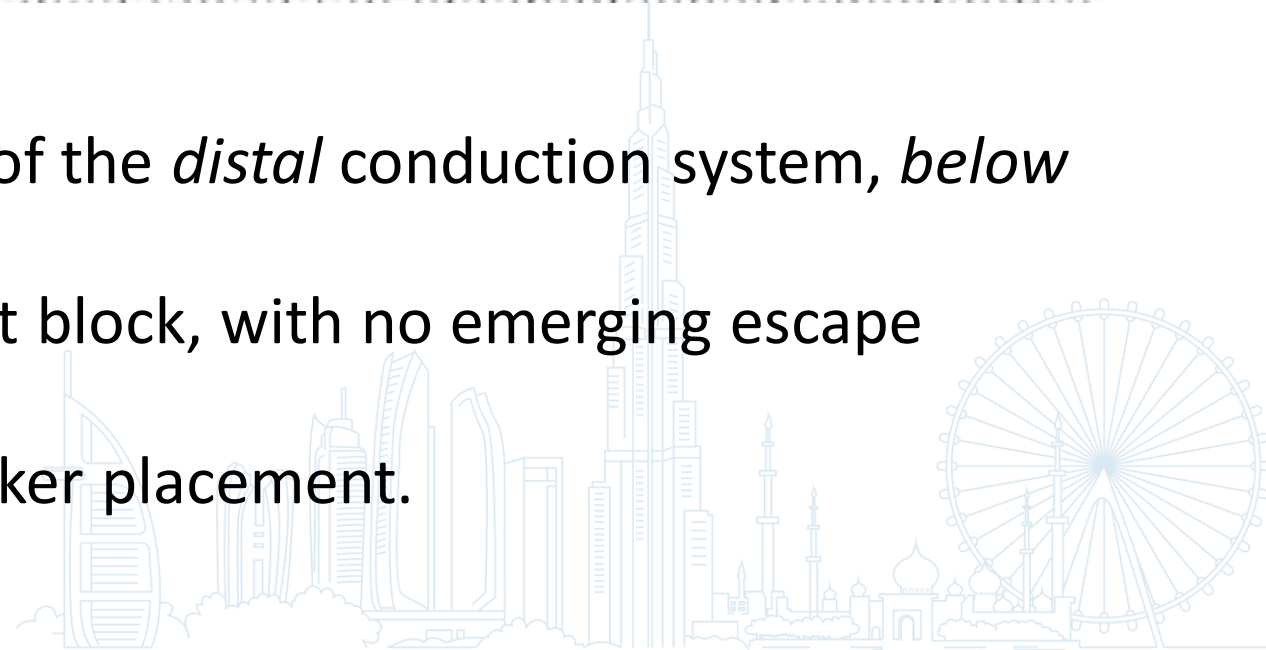




Contrast: Mobitz II second-degree AV block

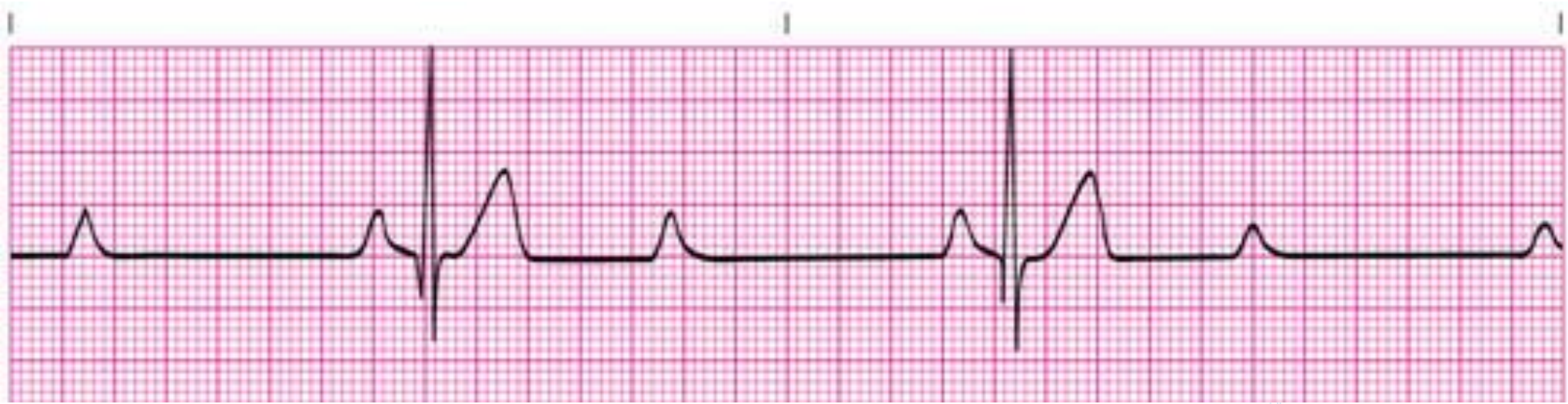


- Almost always represents disease of the *distal* conduction system, *below* the AV node: His-Purkinje system
- May progress to third-degree heart block, with no emerging escape rhythm
- Most commonly results in pacemaker placement.

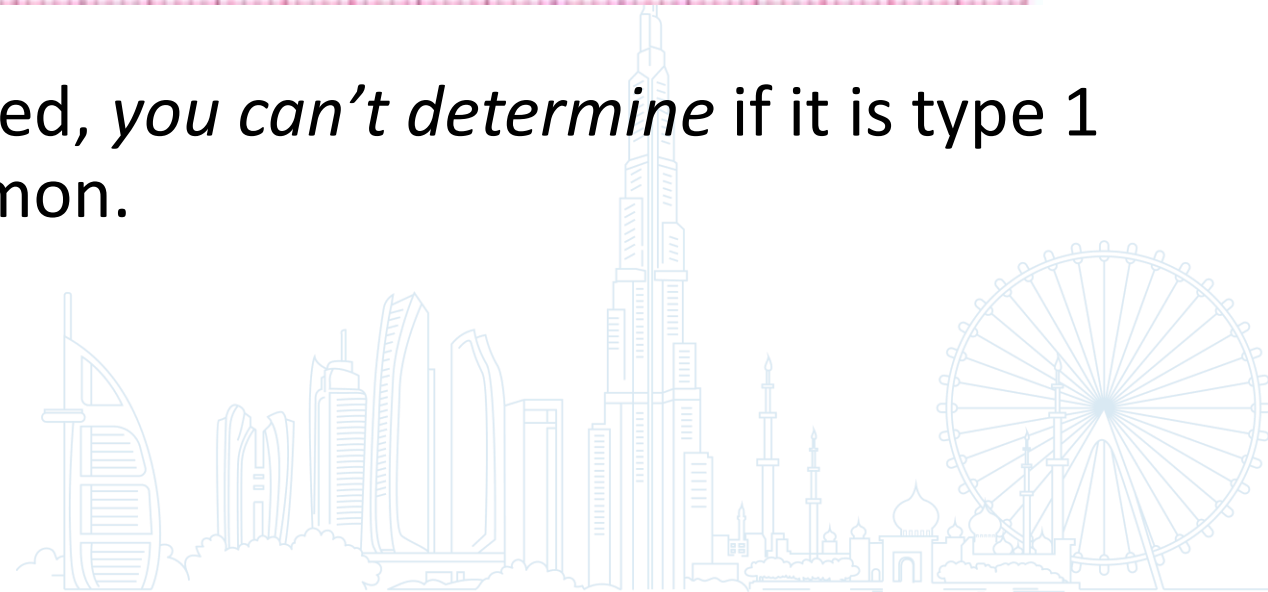




This is 2:1 second-degree block



- When every other P wave is blocked, *you can't determine* if it is type 1 or type 2. Type 1 is far more common.



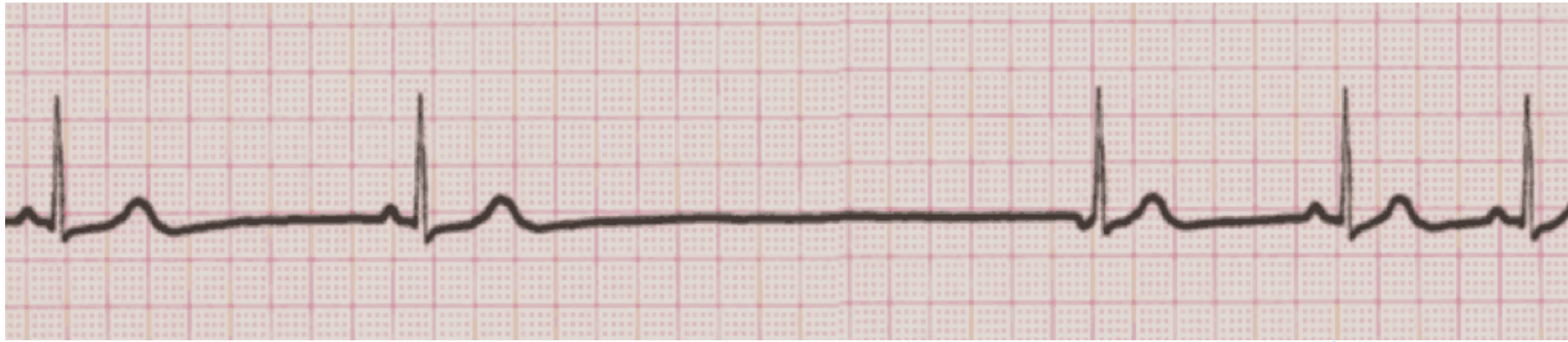


Sinus pause

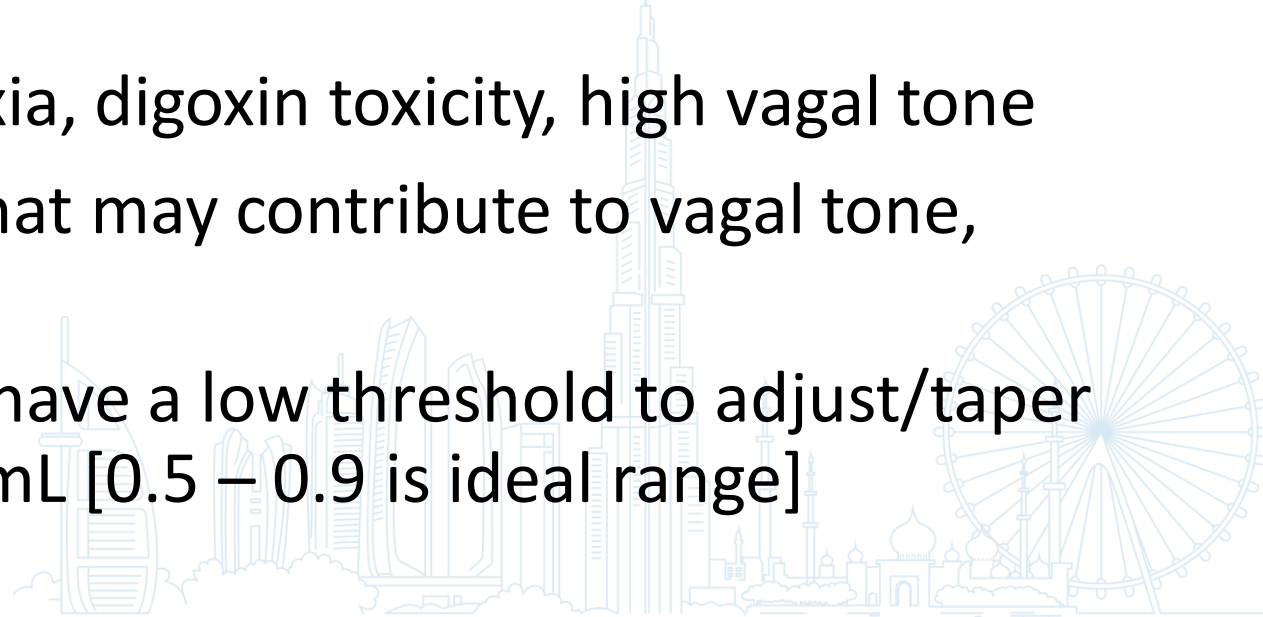




Sinus pause

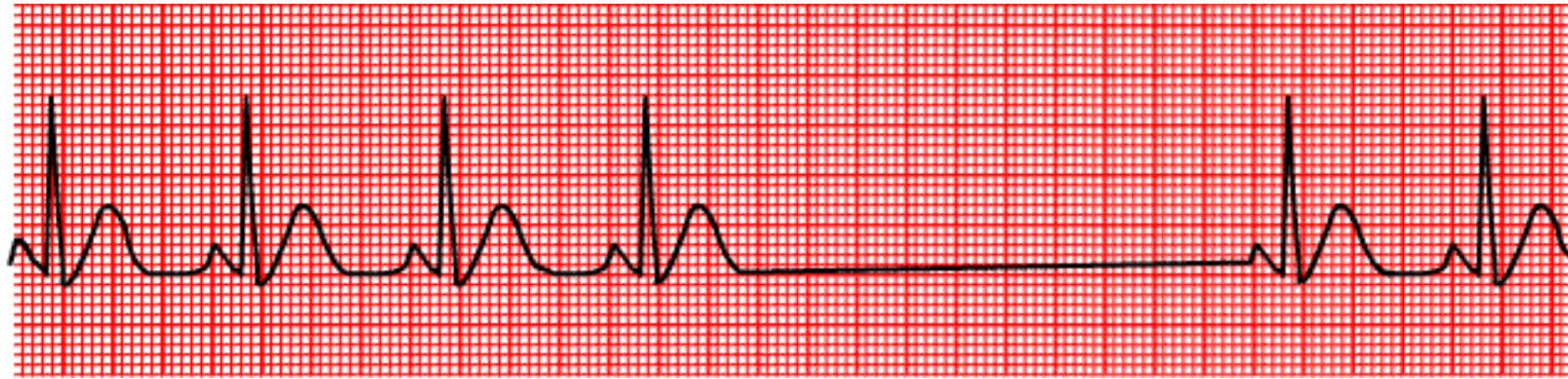


- Think: sinus node ischemia, hypoxia, digoxin toxicity, high vagal tone
- Consider removing medications that may contribute to vagal tone, especially digoxin
- If using digoxin, follow levels and have a low threshold to adjust/taper dose – especially if level >1.0 ng/mL [0.5 – 0.9 is ideal range]

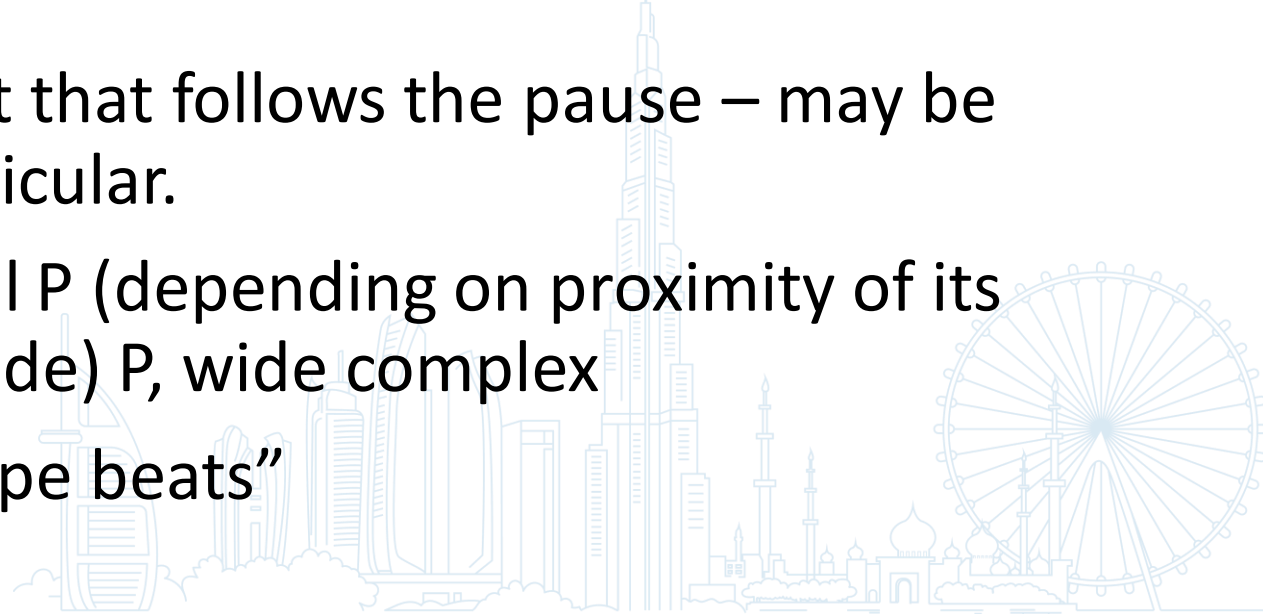




Sinus pause



- Also pay attention to the first beat that follows the pause – may be sinus or atrial, junctional, or ventricular.
- So: normal P, potentially abnormal P (depending on proximity of its origin to SA node), no (or retrograde) P, wide complex
- Non-sinus beats are termed “escape beats”





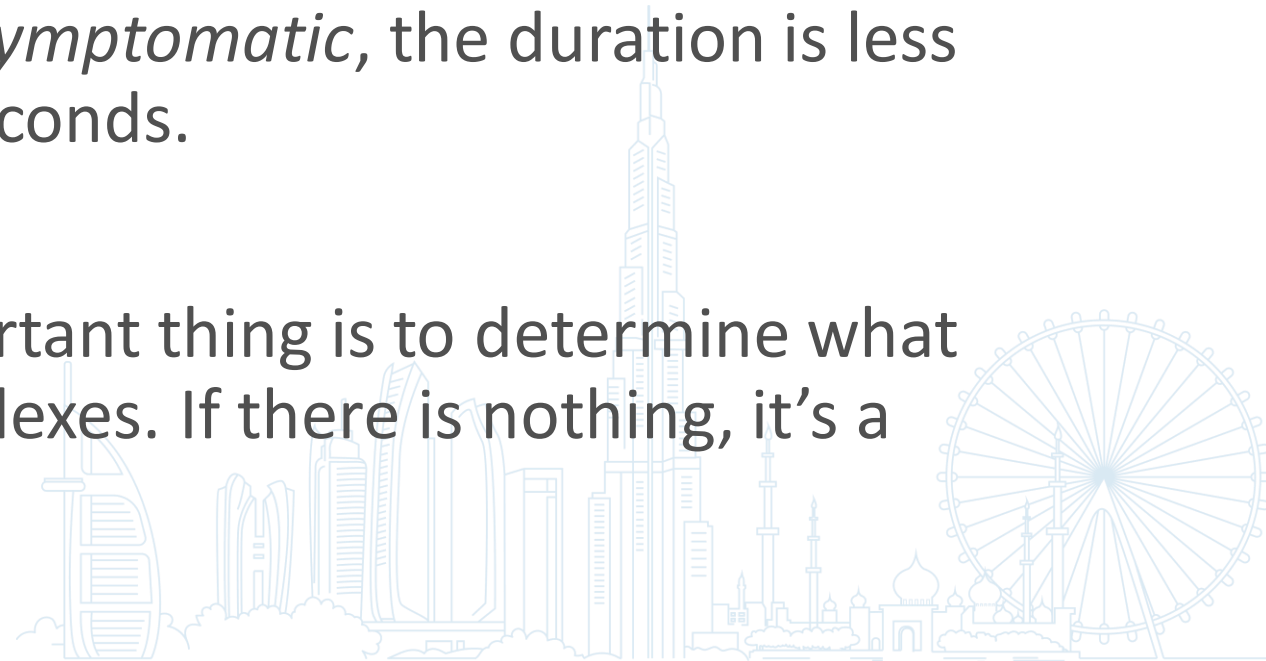
Does it matter?

Sinus pause

- It depends. IF *symptomatic*, may pursue treatment of sinus node dysfunction – i.e., electrophysiology evaluation.

IF *completely asymptomatic*, the duration is less important, even if exceeding 3-5 seconds.

Difficulty rating: 2 – the most important thing is to determine what happens in between the QRS complexes. If there is nothing, it's a sinus pause.





Left anterior fascicular block (or hemiblock)

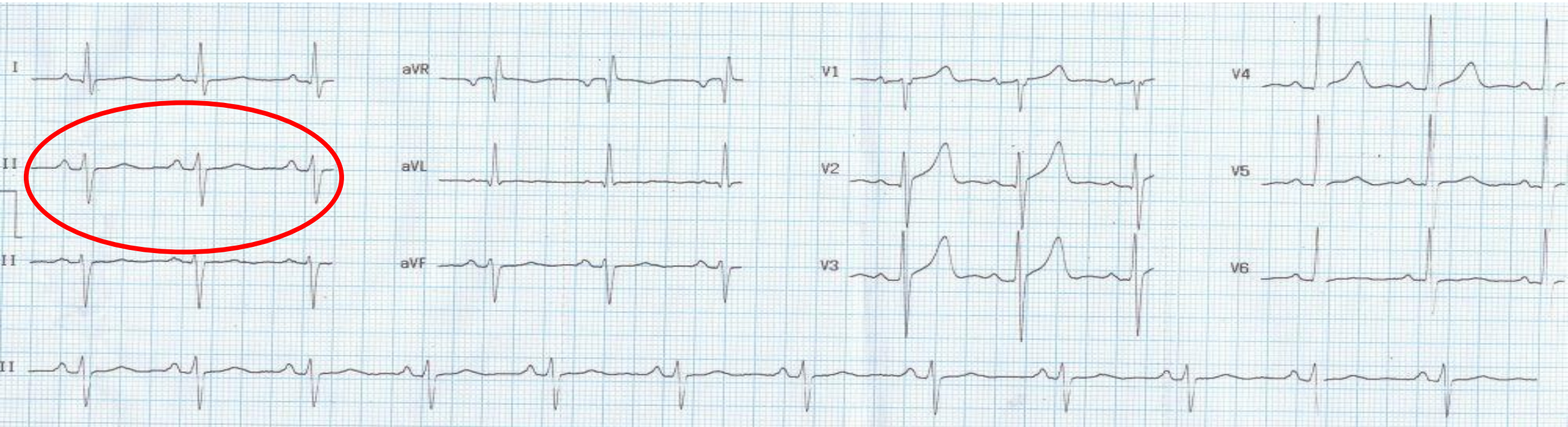




Left anterior fascicular block

- Conduction initially travels down L posterior fascicle, which supplies the inferior LV
 - Depolarization progresses inferior to superior and right to left
 - Resulting L axis deviation – *at least* -30°
- Quick identification on EKG: lead II is negative





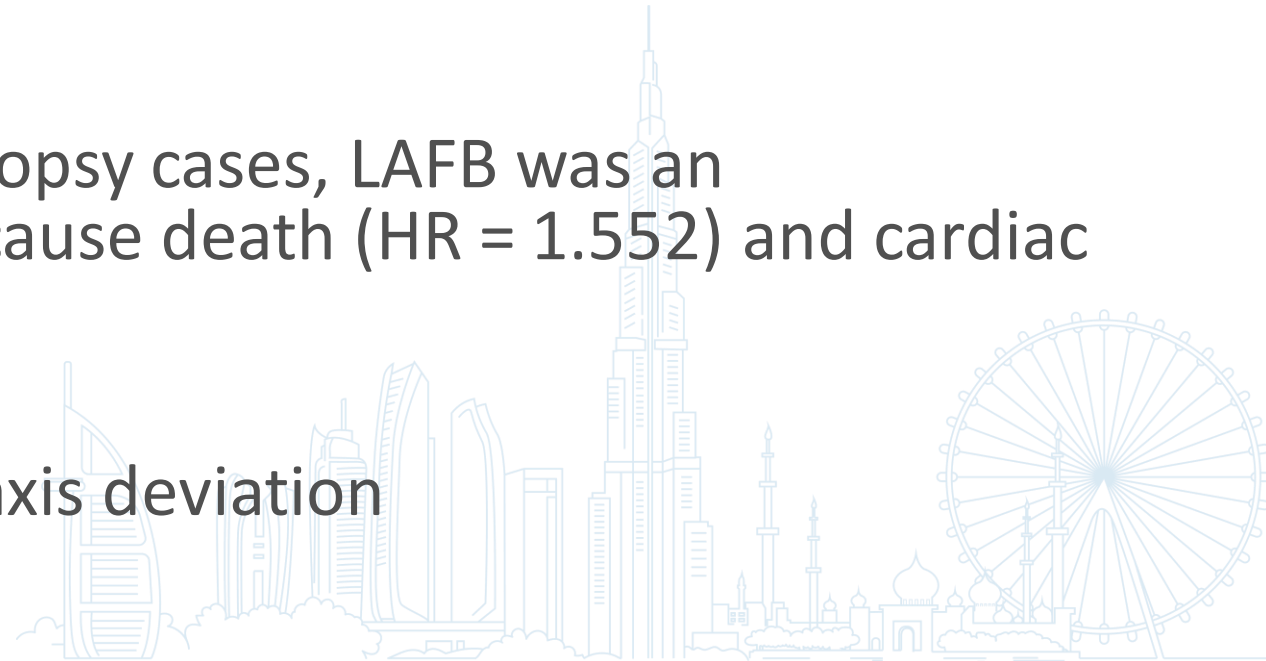


Does it matter?

Left anterior fascicular block

- Short term:
 - Not really. QRS duration is very slightly prolonged but no impact on cardiac function
- Longer term:
 - Probably. In a study of 570 autopsy cases, LAFB was an independent risk factor of all-cause death (HR = 1.552) and cardiac death (HR = 2.287).

Difficulty rating: 2 – recognize left axis deviation





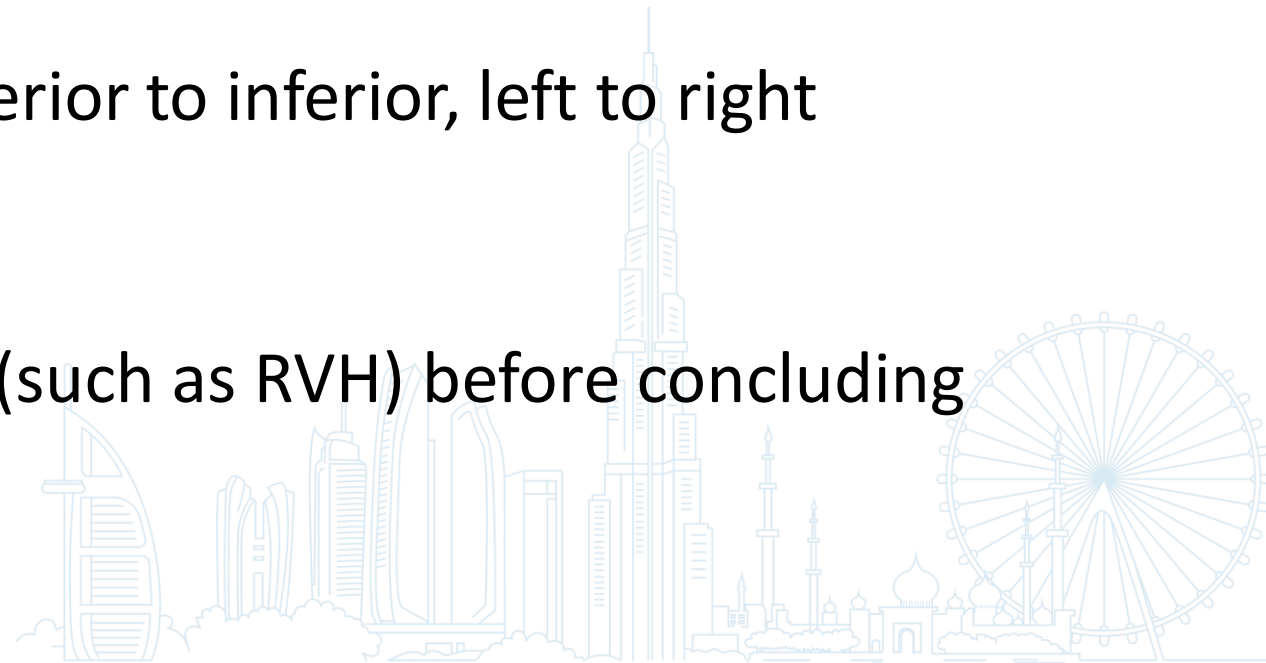
Contrast: Left *posterior* fascicular block





Left posterior fascicular block

- *Much* less common than LAFB
 - Blood supply to L anterior fascicle is more tenuous
- Conduction initially travels down L anterior fascicle, which supplies the anterior LV
 - Depolarization progresses superior to inferior, left to right
 - Resulting R axis deviation
- EXCLUDE other causes of RAD (such as RVH) before concluding that LPFB is present



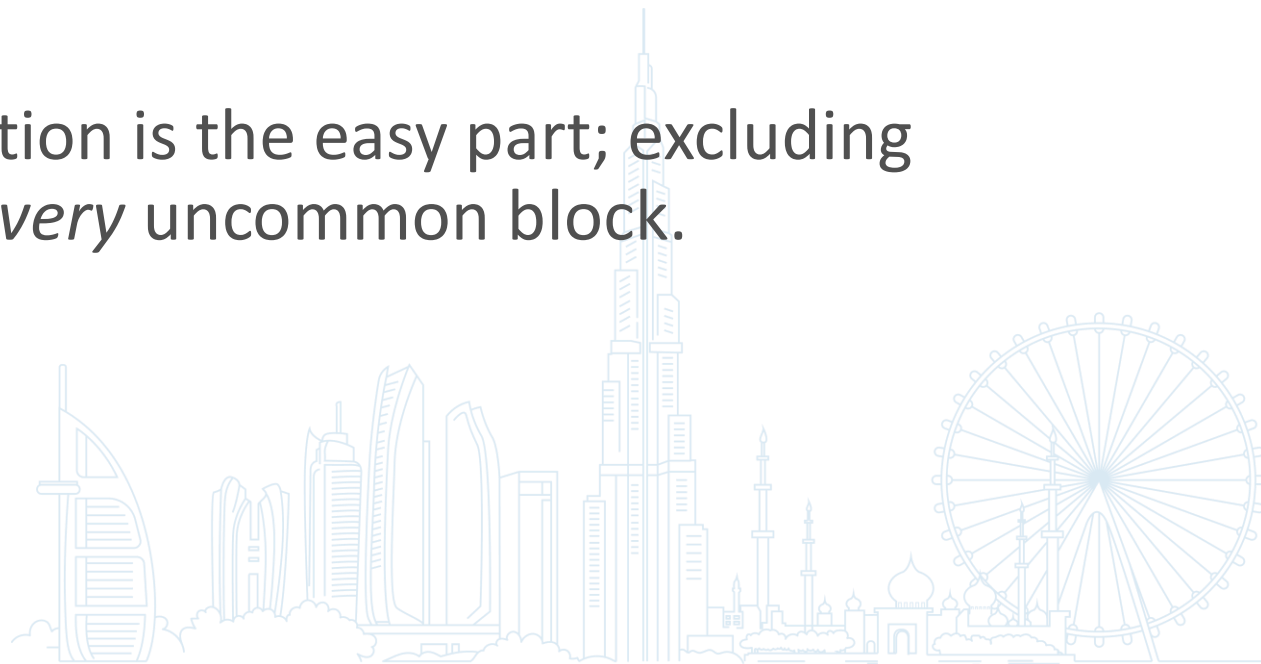


Does it matter?

Left posterior fascicular block

- Yes. LPFB is reliably connected with inferior wall MI and generally reflects severe two- or three-vessel disease.

Difficulty rating: 4 – right axis deviation is the easy part; excluding other causes is more difficult. It's a *very* uncommon block.





Preexcitation

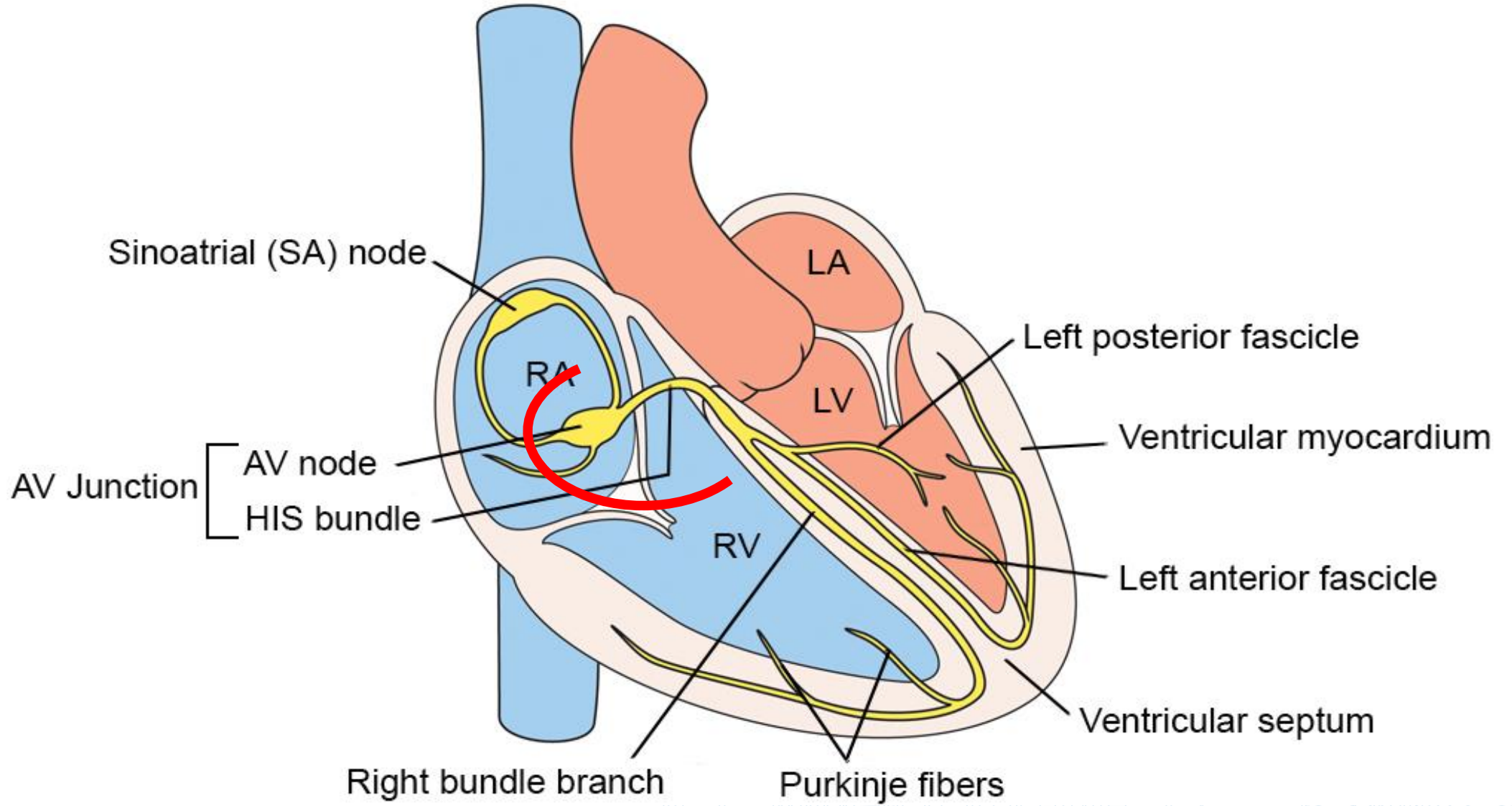




Preexcitation

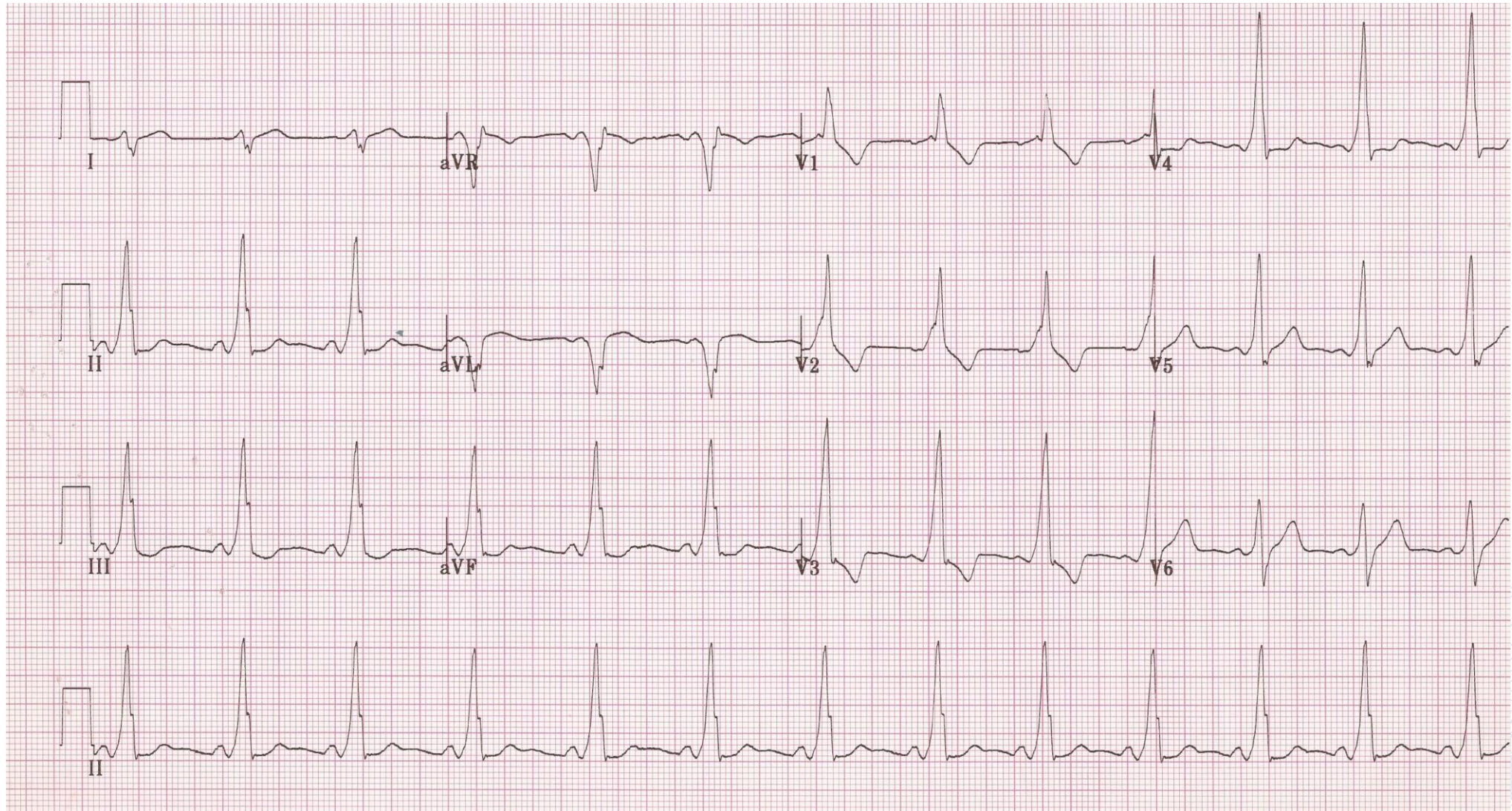
- Wolff-Parkinson White (WPW) Syndrome is the prototype
 - Bundle of Kent
- *Very simplistically*, an accessory pathway exists between the atria and ventricles that bypasses the AV node
- Characteristics:
 - Short PR interval ($<0.12s$)
 - Delta wave with widened QRS complex ($>0.12s$)
- Associated with tachyarrhythmias





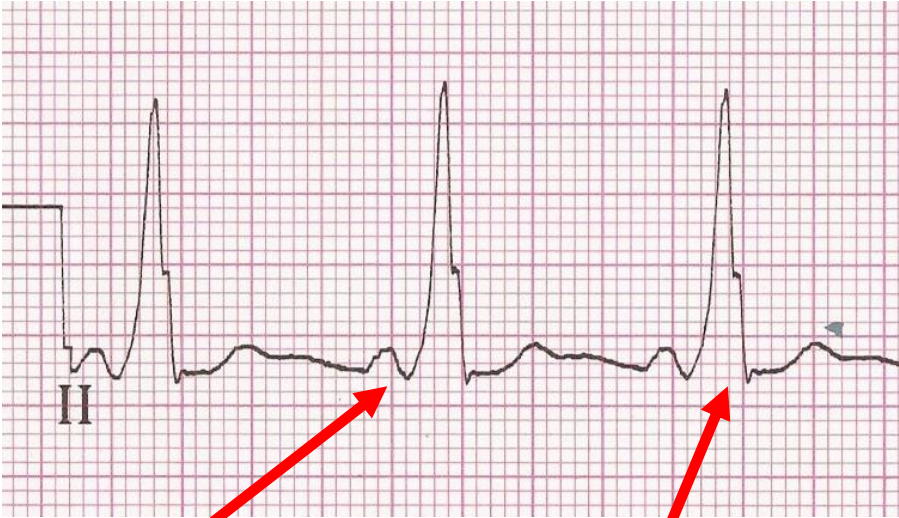


Preexcitation





Preexcitation



Short PR interval

Widened QRS

Delta wave



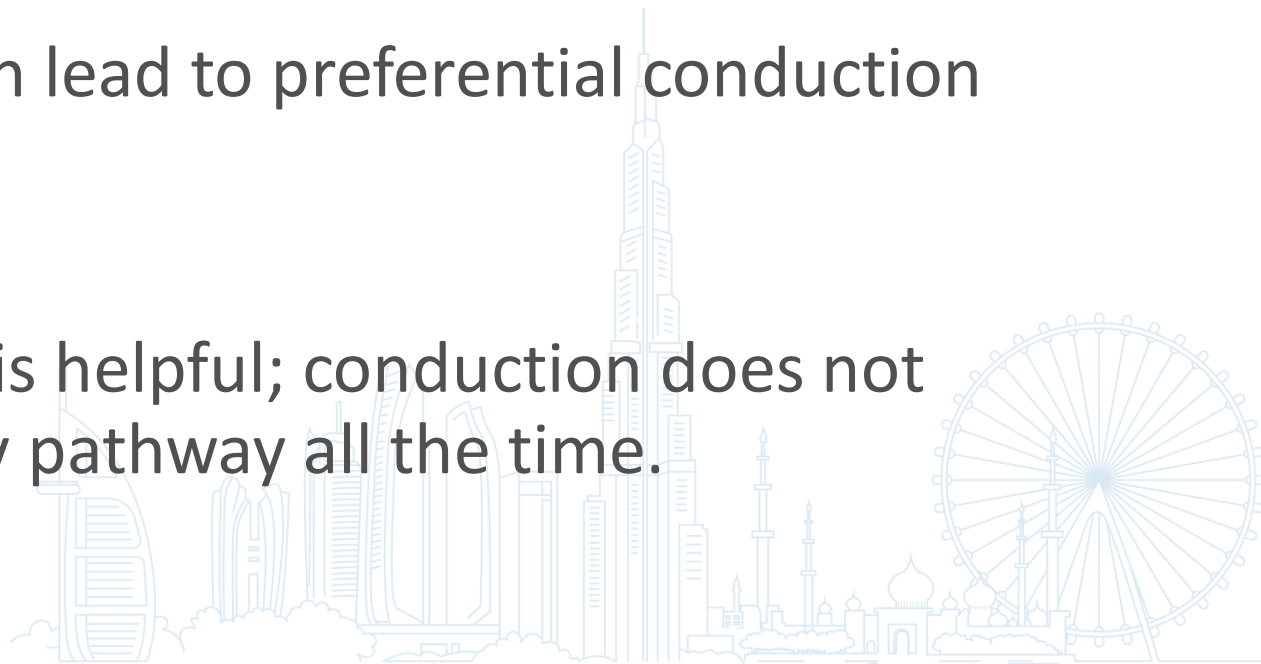


Does it matter?

Preexcitation

- Yes, it absolutely does. Accessory pathways are associated with tachyarrhythmias; most end up in the EP lab for ablation.
- Drugs that slow AV conduction can lead to preferential conduction down the accessory pathway.

Difficulty rating: 5 – clinical history is helpful; conduction does not always proceed down the accessory pathway all the time.



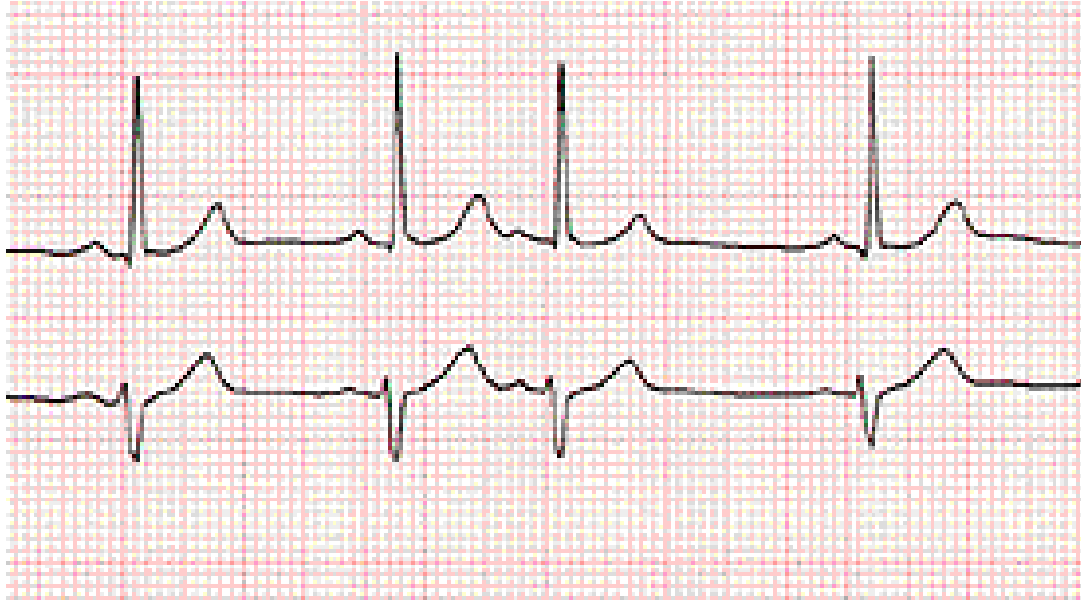


PACs and PVCs





PACs and PVCs



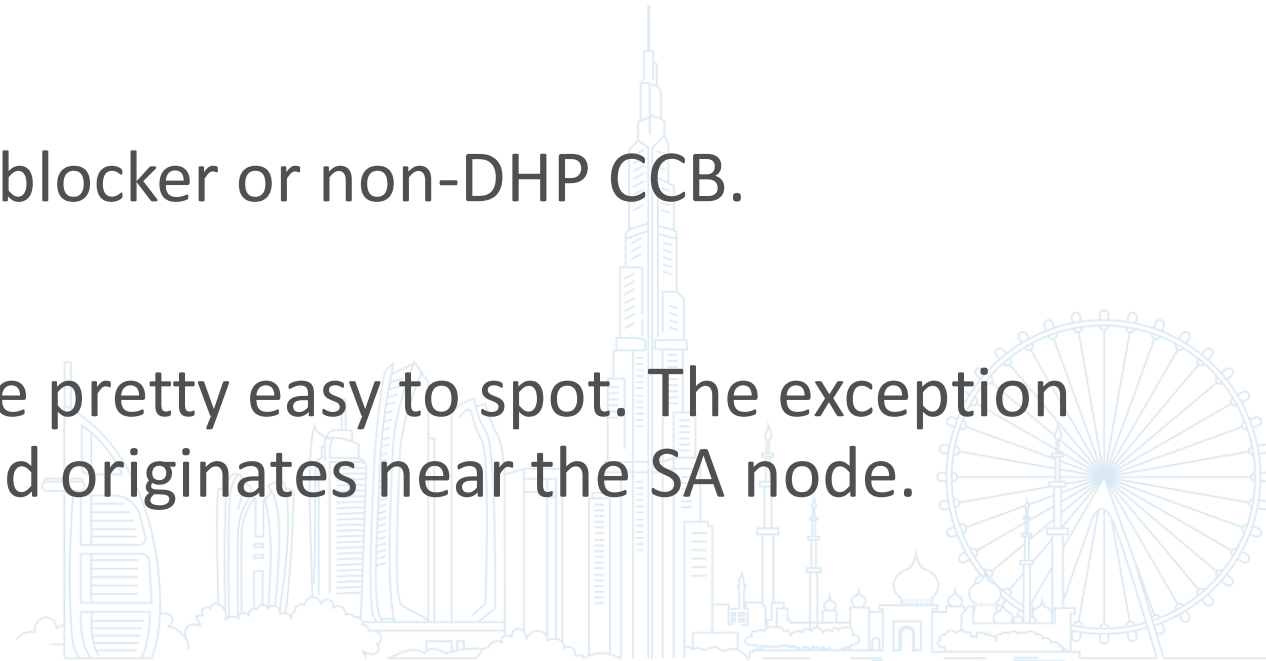


Does it matter?

PACs and PVCs

- PVCs: Not really, unless plentiful. 5% (?) PVC burden is (arbitrarily) “high.” >20% PVC burden should be referred to cardiology.
- PACs: Not really, unless plentiful.
- Both can be decreased with beta-blocker or non-DHP CCB.

Difficulty rating: 1 – these should be pretty easy to spot. The exception is a PAC that is barely premature and originates near the SA node.





Reading EKGs

- The 10-second proficiency rule.
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- Seek a mentor.
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Thank you for your kind attention.

