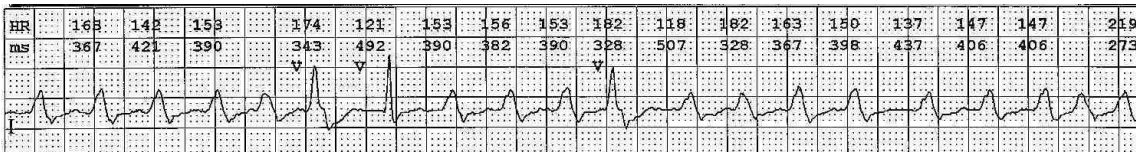
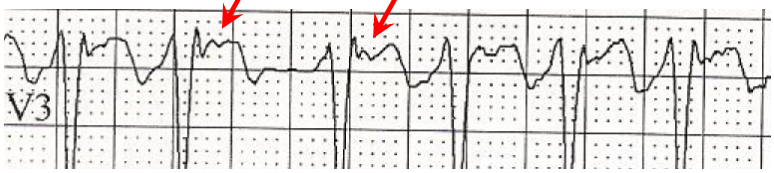
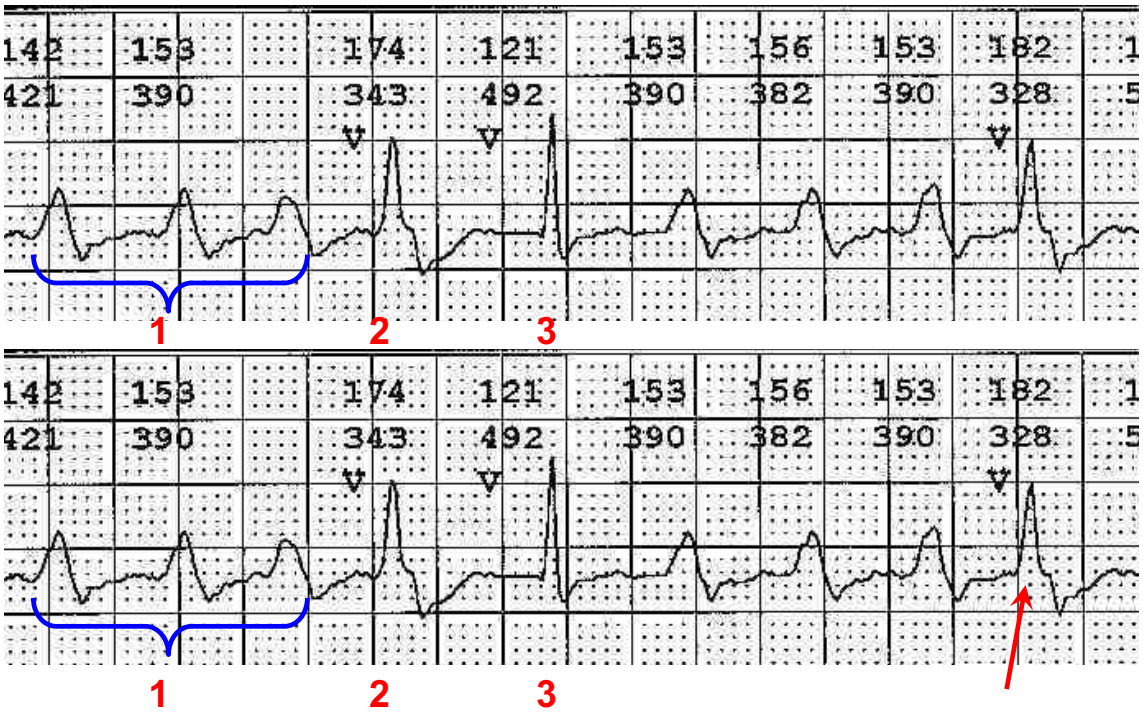
Thank you for this difficult ECG



- This is totally irregular rhythm
- This suggest AF, (DD: Atrial Tachycardia)
- There is no clear P waves, despite there is some atrial activity



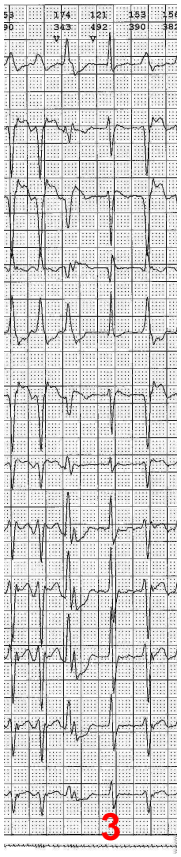
- The QRS is wide, >160 ms, this may be due underlying structural heart disease or tachycardia induced aberrancy.
- The presence of irregularity and no clear AV dissociation excludes VT
- BUT, there is 2 beats with different morphology from 1



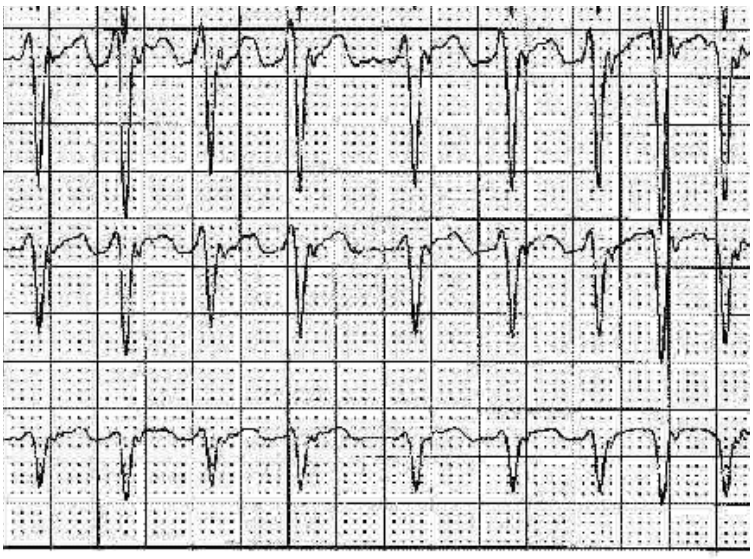
- Number 2 is a VPC and it reoccurred again

- The other is looks here narrow, but when we look in other leads it is wide and changes the axis of RBBB pattern

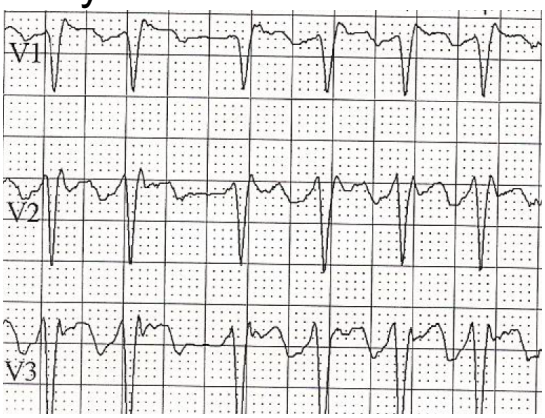
- I think this again is another VPC of different origin



- The patient has also Electrical Alternance which is more clear in the chest leads



- He also has Qs in the inferior leads, and loss of forces in the chest leads which may suggest underlying structural heart disease.
- BUT he also has ST segment elevation in the chest leads in V1- V3 suggestive of Brugada Syndrome type 2
- Despite its appearance is atypical due to the presence of the atrial activity and it is not characteristic of BrS and



Now…….

- Can you give me some clinical hints.

- Thank you very much

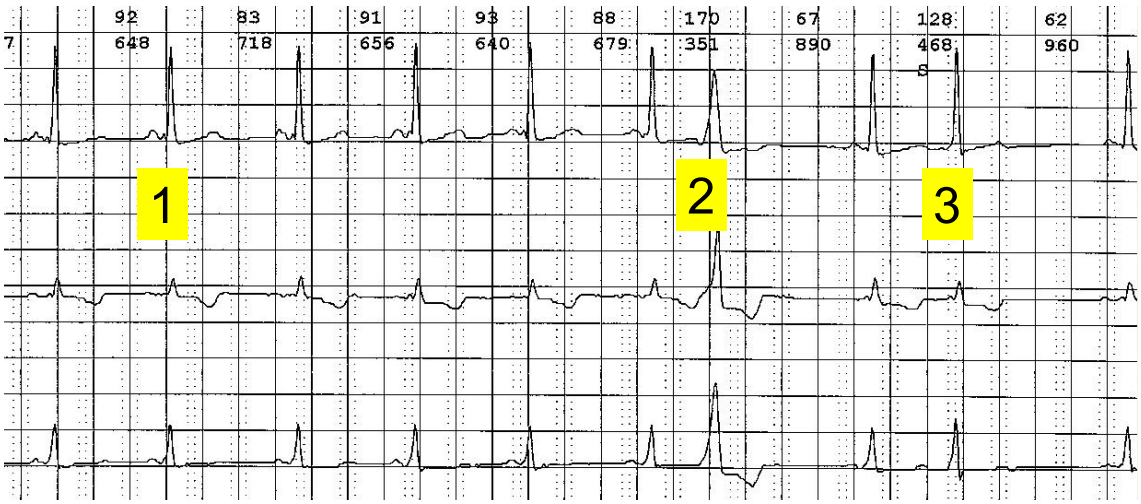
Raed

Thank you for this challenge

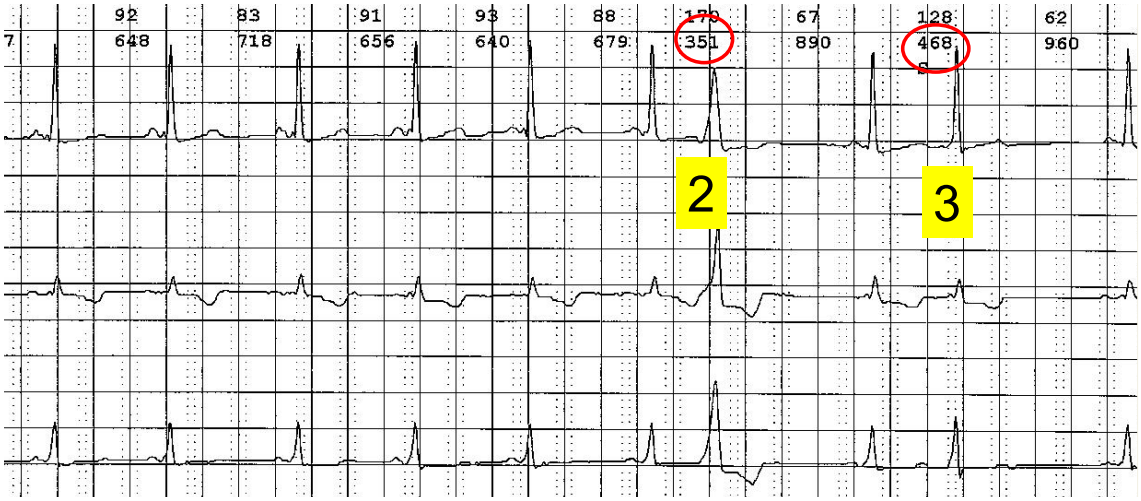
Raed Abu Sham'a

- Sinus Rhythm
- Frequent APCs
- Three different QRS morphology:

1. **regular sinus**
2. **narrow pre-excited**
3. **wide pre-excited**

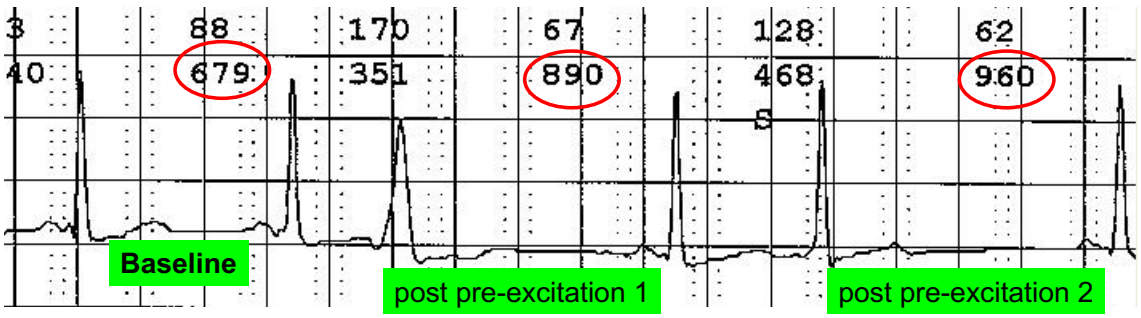


- The narrow pre-excited beat (3)always has a longer cycle length before the APC
- While the wide pre-excited (2) has a shorter CL
- The wider QRS with shorter CL because the normal pathway is still refractory, while the other one may be fusion due to partial refractory period

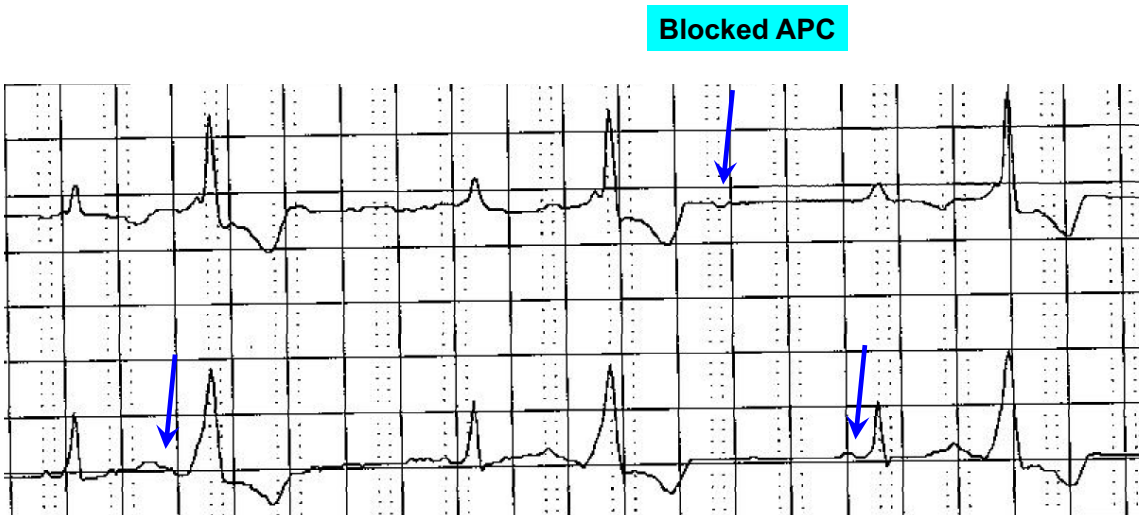


Now……..

- Why post the pre-excited there is a long CL (R - R or P- P) and longer pause than the baseline cycle length?



- I think there is some non conducted APCs after the pre-excited beats
- So… we have 3 different P wave morphologies.



In Conclusion

- A case of WPW syndrome
- ? Multi-focal Atrial Tachycardia
- 12 lead ECG would be more helpful
- I would recommend Exercise Stress Test for more evaluation
- RF ablation is the treatment of choice
- If not available or the patient refused, then class Ia drugs may be a reasonable option

Thank you very much

Dear Raed excellent analysis! You are really amazing.
About the tracings shown, I have to say: when we find irregular RR, as in this case, we should ask ourselves the following question: Is there a P wave or not?

1) If you do not have a P wave = AF
2) If you do have P wave, there are three possibilities:
    a. Flutter with variable AV block. In such a case, it would be easier to see "F" waves of flutter in the moment of the least AV block degree;
    b. Multifocal Atrial Tachycardia (MAT): In this case, there should be 3 or more morphologies of P in the same lead;
    c. Junctional tachycardia with variable AV block: In this case there would be a negative, retrograde P wave after QRS, of negative polarity in the inferior side (retrograde P waves). Its electrophysiological basis is caused by ectopic runs of hyperautomatism.

I think that there is no P wave, although a suggestive apiculate event (P waves) makes me doubt, which is located near the J point in the inferior side leads, with positive polarity, and negative in DI and aVL, which may correspond to antidromic tachycardia, as a consequence of a WPW syndrome circuit, since a delta wave is doubtfully hinted in QRS of aVL.
When we find broad QRS as in this case, it could be:

1) Aberration depending on heart rate;
2) Ashman phenomenon in AF;
3) Antidromic tachycardia by WPW syndrome reentry. This defines an antidromic tachycardia as reentry with anterograde conduction by accessory pathway and retrograde conduction by the AV node.

In this case I think ***it is AF with a high rate of ventricular response and Ashman phenomenon***.
This electrophysiological phenomenon is caused by aberration of supraventricular impulse, secondary to change in QRS cycle length. In 1947 (exactly 61 years ago!), Gouaux and Ashman (***Gouaux JL, Ashman R. aberración con fibrilación auricular paroxística taquicardia ventricular simulación. Am Heart J 1947; 34:366***.) reported about AF, when appearing with a long cycle followed by a short cycle, often with CRBBB morphology. This phenomenon is usually confused with premature contractions. If a sudden increase in QRS cycle duration occurs, a posterior impulse happens, that will have a normal or short duration, that may conduct with less aberration (seventh complex of ECG, conducted on Dec. 30th, at 17:30 and 50 sec.) Studies highlight the significance of heart rate in the genesis of ventricular arrhythmias.
The introduction of premature atrial stimuli by catheter electrode may originate the following aberration patterns:
1) CRBBB: 24%; 2) CRBBB + LAFB: 18%; 3) LAFB: 15%; 4) CRBBB/LPFB: 10%; 5) CLBBB: 5%; 6) ILBBB: 6%; 7) minimal changes in outline: 6%; 8) Prominent Anterior Forces (PAF): 4%. The way we see it, these imply a block in the middle division of the left branch.
But I think that the rare macroreentry of WPW cannot be completely ruled out.
We need he clinical dates I'm waiting for it.
All the best
Andrés R. Pérez Riera