ECG #8

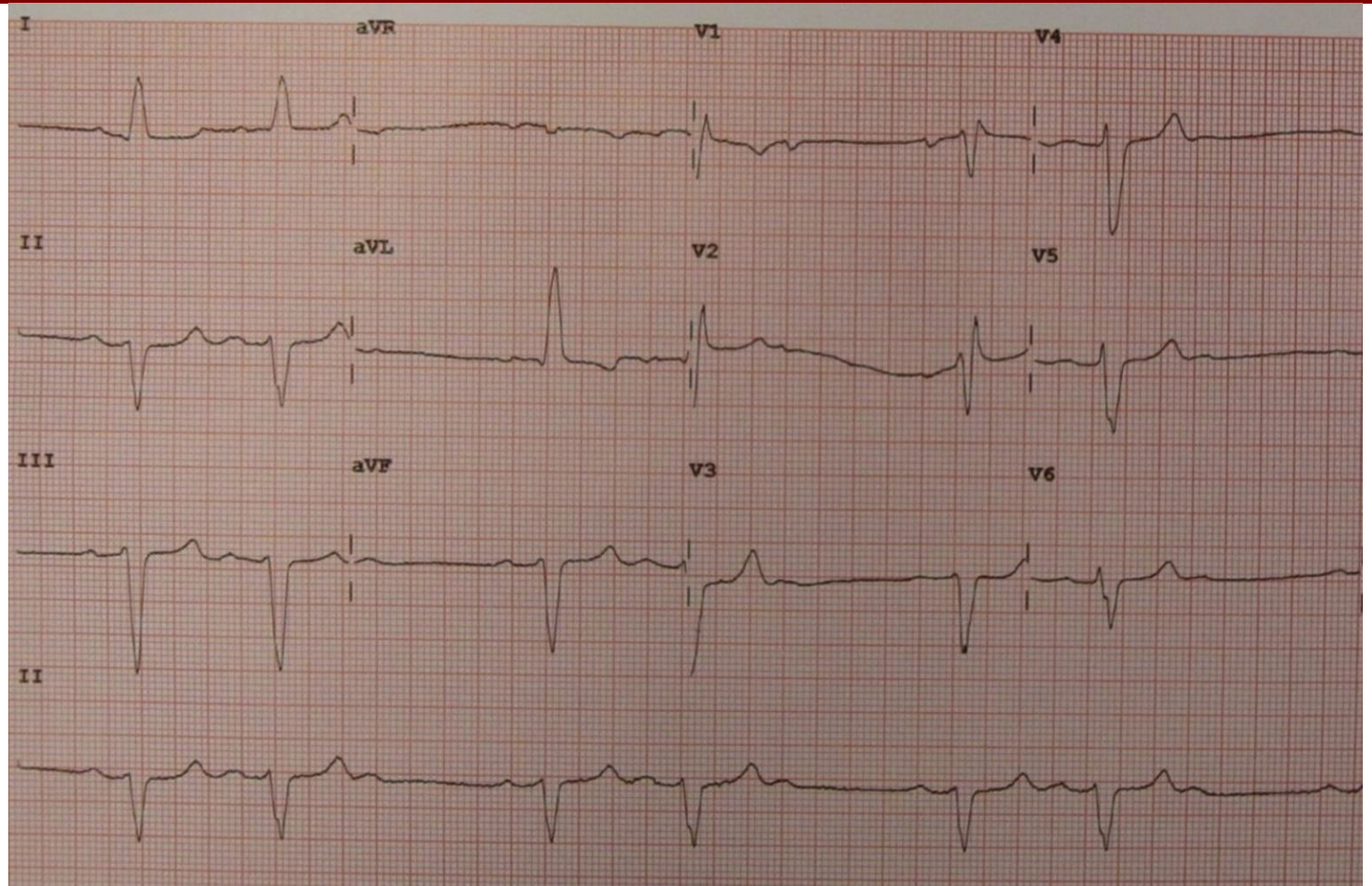
WAMSS SGR 2022

Trigger

You are an RMO in the ED at a peripheral hospital. It is currently 5:00pm and Quentin, a 62M patient presents following a syncopal event in the parking lot of his local grocery store. He reports that he is currently feeling lightheaded. Mr. Q has a long-standing history of hypertension which is poorly managed. His hypertension is currently treated with ramipril and diltiazem.

On examination Quentin is responsive. He sustained some small cuts and bruises from his fall but is otherwise well. He is bradycardic and his blood pressure is 158/102 mmHg (which is normal for him), with no postural hypotension.

Task: Interpret the ECG and provide a diagnosis.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Rate	Atrial: ~70 bpm, Ventricular: ~40 bpm
Rhythm	Regularly irregular
Axis	Left axis deviation
Intervals (ref. ranges)	PR (120-200) – AV dissociation (P and QRS occurring at different rates at 3:2, when conduction does occur PR: ~240ms) QRS (<120) - broad (> 200 ms) QT (<440) – WNL
Segments	Occasional loss of AV conduction, conducting in a 3:2 pattern. PR interval when conducted is elevated at ~240 ms but still constant, indicating Mobitz type II 2nd degree AV block. No ST elevation seen M waveform in V1 and W waveform in V6 is suggestive of RBBB
Other morphology	T-wave inversion in aVL
Interpretation	In summary, this is an ECG of a 62M presenting with syncope and lightheadedness, likely of cardiac origin. On examination he is hypertensive and bradycardic. The ECG is abnormal, with a regularly irregular rate, left axis deviation, AV dissociation in a 3:2 conductance pattern and RBBB. My working differential is a **Mobitz type II 2nd degree AV block**.

Follow-up Questions

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1. What are your initial investigations for Quentin?

2. How will you manage Quentin's condition?

3. What is the pathophysiology underlying PR prolongation or PR absence in the different types of AV block?

Question 1

- **Serum troponin**
  - To rule out any ischaemic causes of syncope and AV block in Mr Q
- **UECs and CMP (K^+ and Ca^{2+})**
  - Abnormal calcium and potassium levels are reversible causes of AV block
  - Patient is on ramipril which can cause hyperkalaemia
- **Other potential investigations:**
  - **Transthoracic echocardiogram** – to identify potential structural causes of MR Q's AV block especially given his history of hypertension
  - **Holter monitor** – for further evidence of AV block

Question 2

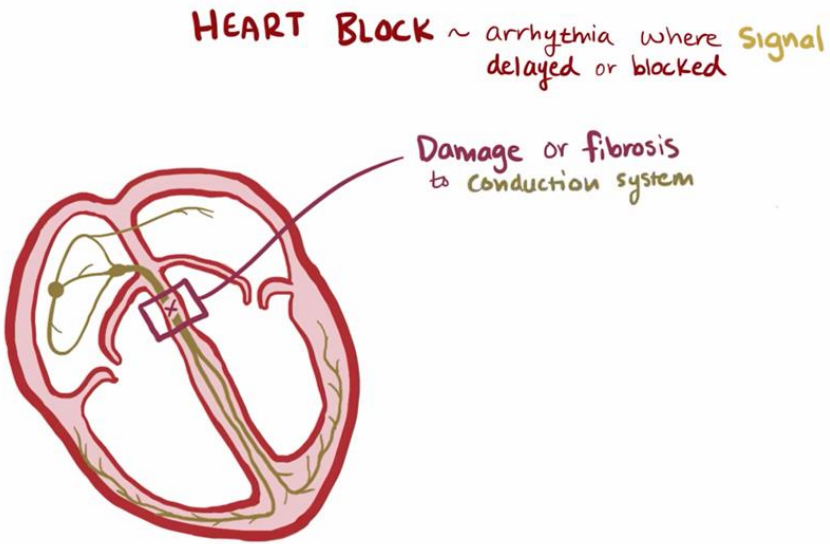
- **If reversible:**
  - Treat reversible causes:
    - Acute myocardial infarction
    - Electrolyte abnormalities
    - pH derangement
    - AV nodal blocking drugs (non-dihydropyridine calcium channel blockers, beta-blockers, digoxin)
  - If showing minimal/no response to treatment consider placement of permanent pacemaker (PPM)
- **If irreversible:**
  - Consider PPM

Question 2 cont.

- **Specific management for Quentin:**
  - Consider changing hypertension management from ACE + CCB to ACE + thiazide diuretic
  - If refractory to treatment: consider PPM

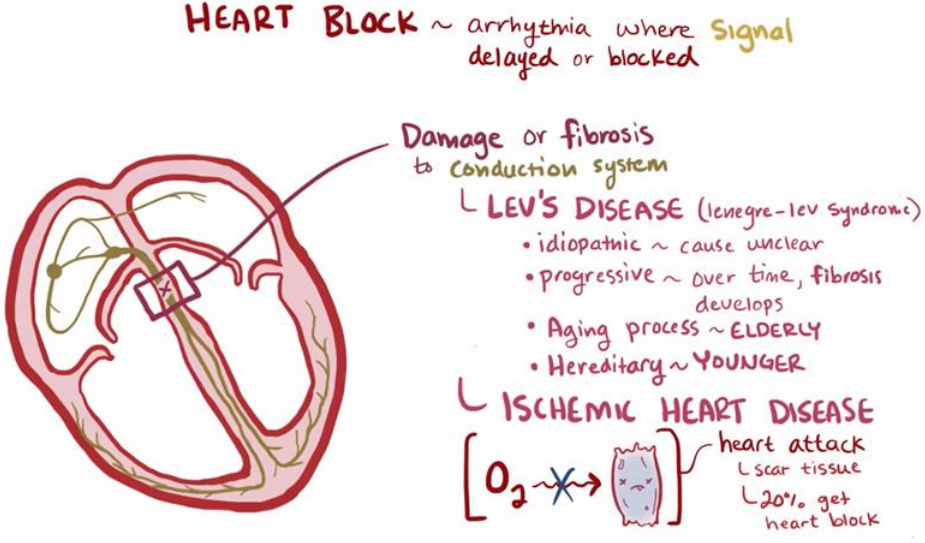
Question 3

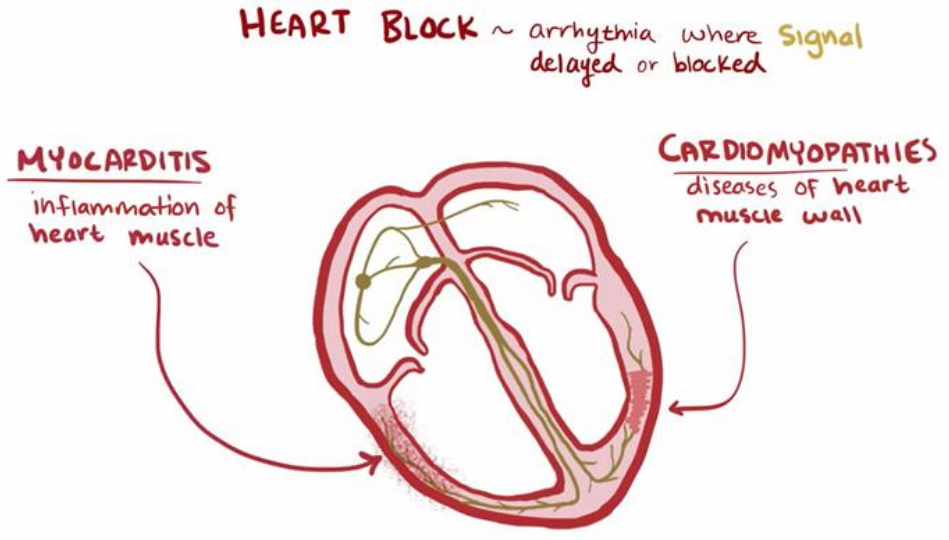
- Damage or fibrosis to the electrical conduction system between the AV node and the His-Purkinje fibres of the ventricles



Question 3

- The damage or fibrosis can be idiopathic, or caused by ischaemic heart disease, cardiomyopathies, myocarditis or iatrogenic (e.g. meds)





Thank you!

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