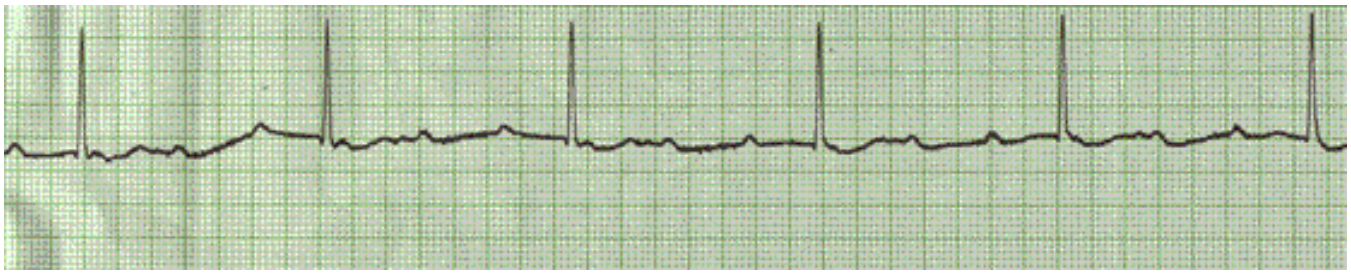
QUICK QUIZ
WHAT IS THE DIAGNOSIS?

BACKGROUND

A 68-year-old woman of Asian descent presents to the emergency department complaining of intermittent substernal chest pressure and progressive dyspnoea for 4 days. She denies having similar symptoms in the past. Today, her symptoms worsened significantly and she is now dyspnoeic at rest. Her chest pressure is constant, moderate in intensity, non-radiating, non-pleuritic and non-positional. She has no paroxysmal nocturnal dyspnoea, orthopnoea or lower-extremity oedema. She denies long-distance travel, recent surgery, or immobilization. Her medical history is significant for diabetes mellitus for which she takes an oral hypoglycaemic agent.

During triage, the patient is awake, calm, and alert. She has a respiratory rate of 30 breaths per minute, a heart rate of 40 bpm, a blood pressure of 138/76 mm Hg, and an oxygen saturation of 82% on room air. The patient was immediately given oxygen by means of a nonrebreather face mask and attached to a cardiac monitor with continuous pulse oximetry. An intravenous line is started, external cardiac-pacing pads are placed on her chest, and a cardiac resuscitation cart is brought to the bedside. An initial rhythm strip is obtained.



QUESTIONS

1. What is the diagnosis?
2. What are the clinically significant findings?
3. How is the condition treated?

ANSWER & DISCUSSION

1. Diagnosis

Mobitz type II atrioventricular (AV) block with variable conduction delay.

Mobitz II mimics must also be considered and ruled out. Two possibilities are nonconducting premature atrial contractions and a physiologic block of atrial tachycardia or flutter. In the first, multiple P waves precede a QRS complex, but the morphologies of the P waves typically vary, as might the P-P intervals. In the second, the physiologic refractoriness of the AV node prevents underlying atrial tachycardia from leading to a corresponding ventricular tachycardia. The ventricular rate is likely to be rapid in a physiologic block in contrast to a Mobitz II block, in which bradycardia is expected.

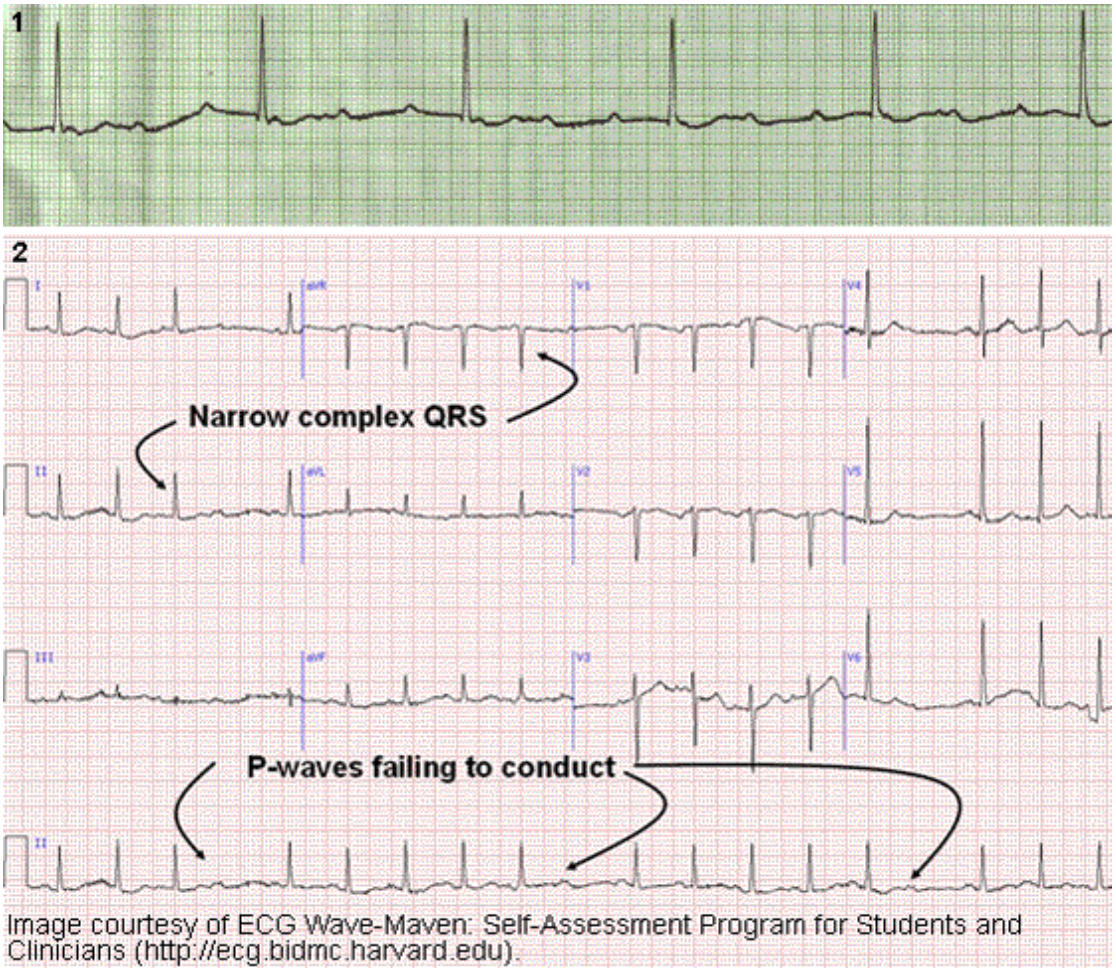
In addition to the rhythm, the presumed location of the block also has implications on the patient's care. Conduction delays arise from either the AV node itself or along the His-Purkinje system. Unlike first-degree AV blocks and Mobitz I second-degree blocks, which occur in the AV node itself, Mobitz II blocks typically occur in an infranodal location, usually in the bundle branches or Purkinje system. As a consequence, QRS widening or a bundle-branch pattern is often present. In 25% of patients, the block is in the His bundle, and the QRS complexes have normal morphology and duration.

Conduction delays can occur as a result of several medical conditions. Fibrosis, sclerodegenerative changes, and ischemic disease are the most common. Medications, including digitalis, verapamil, and beta-blockers, are also common causes. Other causes are cardiomyopathies, familial disease, increased vagal tone, hyperkalemia, and many other rare conditions. Coronary ischemia must be of highest concern because of heart block secondary to infarction is associated with a high mortality rate.

2. Clinically significant findings

This patient had a profound bradycardia due to a second-degree type II (Mobitz II) AV block.

This block can be differentiated from complete heart block because a P wave precedes every QRS complex with constant PR length, unlike complete heart block in which the P waves and QRS complexes are not related. This type of block also differs from a second-degree type I block (Mobitz I block or Wenckebach pattern) because the PR distance does not change. In a Wenckebach pattern, the PR interval progressively lengthens until a QRS complex is dropped. If a 2:1 block is present, type I and type II second-degree blocks are difficult to distinguish (as with the 12-lead ECG in this case); therefore, the rhythm strip (Image 1) in this case provides further information. It shows evidence of a transition to a 3:2 block, which helps differentiate the type I and II second-degree blocks. This distinction between a Wenckebach block and Mobitz II is important because the latter, as in this case, is considered a high-degree, unstable block with the potential to progress to complete heart block.



3. Treatment

The treatment for a Mobitz II block consists of atropine, isoprenaline, and pacing. However, pharmacologic treatment with atropine or isoprenaline carries a potential harm and should be used with caution. In general, these pharmacologic agents should be considered a temporizing or bridging measure to a stabilizing temporary pacing system. Additionally, these agents are contraindicated in the setting of acute ischemic disease because they can increase myocardial oxygen demand. Unlike a Wenkebach pattern or a first-degree AV nodal block in which the direct effects of atropine at the node improve conduction, a Mobitz II block may not significantly respond to atropine because of its infranodal location. In fact, atropine may paradoxically exacerbate the block by allowing additional sinus impulses to pass the AV node, increasing the refractoriness of the infranodal tissues. If no improvement is seen after a cumulative dose of 2 mg of atropine, it should be discontinued. Isoprenaline may be used as a constant infusion starting at 0.5-2 µg/min and titrated to 10 µg/min to maintain a heart rate of 60 bpm.

Finally, transcutaneous or transvenous pacing is the definitive treatment and should be initiated in symptomatic patients, and possibly even in asymptomatic patients, in anticipation of deterioration. A permanent pacemaker is placed after the patient's condition is sufficiently stabilized. A cardiologist should be consulted on an urgent basis for all patients with a Mobitz II block.