ANSWER

The ventricular rate is around 56/min and is slightly irregular. The QRS complexes are wide, but are not typical of either left or right bundle branch block. Each QRS is preceded by a P-wave with a short PR interval. Wide, deep Q-waves are present in the inferior leads.

The QRS complexes are wide, about 140ms in duration. There is left axis deviation at -30°. The pattern superficially resembles LBBB, but the initial R-wave in V1 is too tall (equal to, or slightly larger than the S-wave) and too broad – at least 80ms. This, together with transition before V2, excludes LBBB. The terminal deflection in V1 is an S-wave, thus excluding RBBB.

What, then, is the reason for the wide QRS?
The following are possible reasons for a wide QRS complex:

- Bundle branch block (excluded)
- Non-specific intraventricular conduction delay
- Ventricular origin
- Idioventricular rhythm
- Paced rhythm
- Pre-excitation

Non-specific intraventricular conduction delay is unlikely, because most of the conduction delay occurs during the initial part of the QRS, with relatively rapid depolarisation in the latter part (see V1). This is more characteristic of pre-excitation where the wide QRS is a fusion of initial slow ventricular activation over the accessory pathway and then rapid ventricular depolarisation via the His Purkinje system. The rhythm is clearly not due to a ventricular pacemaker (the QRS complexes are not compatible with RV pacing and no pacing spikes are visible).

An accelerated idioventricular rhythm with isorhythmic AV dissociation should be considered. The QRS morphology may be consistent with a ventricular rhythm; however, such a rhythm is independent of atrial activity and is regular. The short PR interval could result from coincidental apparent association of the P-waves with the QRS complexes, because of similar rates – isorhythmic AV dissociation. Against this, however, is the constant PR interval. The PR remains constant despite significant variations in the P-P interval, due to sinus arrhythmia. This is clear evidence that the ventricular complexes are related to the atrial depolarisations and excludes a diagnosis of accelerated idioventricular rhythm.

Pre-excitation (Wolff-Parkinson-White pattern) is the best explanation for the combination of the wide QRS complexes with a slow upstroke (delta wave) and the short PR interval.

What about the inferior Q-waves? Does the young man have a previous inferior myocardial infarct as well as WPW?
The inferior Q-waves are deep and very broad, at least 70ms. Their onset coincides exactly with the onset of the delta waves in leads I and aVL. They are delta waves which mimic the Q-waves of inferior infarction. It is therefore not possible from this ECG to either diagnose or exclude a previous inferior infarct. Likewise, the tall initial R in V1 is a delta wave and not due to posterior infarction.

The ST segments are unusual and must be interpreted with caution in view of the depolarisation abnormality. Pre-excitation commonly results in secondary T-wave changes, with T-waves opposite in polarity to the delta waves. However, in this case there is saddle-shaped ST elevation in V1 to V5, in the same direction as the delta waves. This could possibly be the result of acute pericarditis, but there is no evidence of deviation of the PR segments in leads II and aVR which are characteristic of pericarditis. Although he complained of chest pain, this was not typical of either ischaemia or pericarditis. ST elevation was still present several months after successful catheter ablation of his accessory pathway (Figure 1). The appearance in this ECG is consistent with early repolarisation, which is commonly seen, particularly in young black males. Note the absence of inferior “Q”-waves. He did not have left ventricular hypertrophy, despite the high voltage in the chest leads.

The correct answer is therefore (b): WPW pattern.

(2) FURTHER INVESTIGATION AND TREATMENT

The assessment of the ECG indicates the presence of pre-excitation (WPW pattern). The diagnosis of previous or current myocardial infarction is neither made nor excluded by the ECG. Pericarditis is possible, but the ECG is not typical. Echo-cardiography is unlikely to be helpful.
Angiography would only be indicated if his history was strongly suggestive of ischaemia. His chest pain was not suggestive of ischaemia and was probably musculo-skeletal in origin.

While the ECG is diagnostic of pre-excitation, this is not an absolute indication for ablation of the accessory pathway in the absence of symptoms.
When a detailed history was obtained from this young man, it was clear that he had WPW syndrome and not just an asymptomatic pattern of pre-excitation. He had had many episodes of palpitations, several of which had required intervention to terminate them, including an episode of pre-excited atrial fibrillation with a rapid ventricular rate (Figure 2).

Catheter ablation is the treatment of choice for WPW syndrome, but is not generally indicated for asymptomatic people whose WPW pattern is picked up at the time of an ECG performed for another reason. This is controversial, and many would recommend non-invasive and even invasive electrophysiological testing to assess the potential risk in an individual. However, his history and documented rapid pre-excited atrial fibrillation made accessory pathway ablation mandatory.

The answer to (B) is (e): Base your decision on his current and previous history.

As noted above, radiofrequency catheter ablation of the left posterior paraseptal accessory pathway was successful from within the mouth of the coronary sinus.

LESSONS AND CONCLUSIONS

- The Wolff-Parkinson-White pattern can mimic myocardial infarction, both inferior and anterior.
- An early repolarisation pattern in the chest leads is common in young people.
- WPW pattern in the absence of symptoms is not an absolute indication for catheter ablation.
- Pre-excited atrial fibrillation with a rapid ventricular response (shortest R-R less than 250ms) is an absolute indication for accessory pathway ablation.

REFERENCES