SYSTEMATIC ANALYSIS OF THE ECG

1. Rate and regularity: Irregularly irregular with a ventricular rate of 66 bpm.

2. Atrial activity: There is a P-wave preceding each QRS complex. The morphology of the P-waves is abnormal and not identical during this 10-second recording.

3. PR interval: Measures 4.5 small blocks x 0.04s = 180ms and it is constant.

4. QRS complexes are narrow, just under 100ms in duration, with a normal axis of around 30º. There is no suggestion of myocardial infarction. Minor notching due to non-specific conduction abnormality is noted in V1. Although there may be an impression of a slurred upstroke of the QRS in V1, there is no delta wave or pre-excitation. The appearance is due to the ascending terminal component of the negative component of the biphasic P-waves in V1.

5. ST- and T-waves are unremarkable. The QT interval is 360ms, normal.

IN SUMMARY
The ECG shows atrial rhythms with normal AV conduction.

FURTHER DISCUSSION
The atrial activity requires further analysis.

Although the patient’s pulse and rhythm are irregularly irregular, which is the characteristic finding in atrial fibrillation, well-formed atrial activity with P-waves excludes this diagnosis.

There are 2 distinct morphologies of the P-waves (Figure 1):

- The majority, those related to the faster rhythm and also the last P-wave; vs.
- The 1st, 6th and 7th P-waves which are all identical but different.

The first mentioned P-waves are slightly positive in Lead I and positive in aVF; their axis is 75º and compatible with sinus node origin. These P-waves are clearly abnormal being almost 120ms (3 small blocks) and biphasic in V1 with a predominantly negative component – both suggestive of left atrial abnormality, probably enlargement. Their rhythm is not regular. Some irregularity, varying with respiration, called sinus arrhythmia, is normal and physiological. However, the irregularity noted here is excessive with 3 pauses of 1.2 - 1.3 seconds – clear evidence of sinus node dysfunction. The last P-wave on the ECG after a pause is one of these P-waves.

The other P-wave morphology is seen in the very 1st and also the 6th and 7th beats. That they are different from the “faster” P-waves can be early seen in aVF for the 6th P- and V1 for the 7th P-wave. All 3 P-waves are identical as can be confirmed in the V1 rhythm strip at the bottom. (Since the morphology is not multifocal or from ≥3 sites this is not multifocal atrial rhythm.) The axis of these P-waves is minus 60º - these are not arising from the sinus node. These are not atrial premature beats/complexes (APBs) because they are not early; rather, the 6th and 7th P-waves occur after sinus pauses and are escape beats and would not be seen if there had been no pauses or bradycardia.

DIAGNOSIS
Thus, the diagnosis is clear: There is sinus node dysfunction, which also encompasses the term “sick sinus syndrome” and which may at times present with a “tachy-brady” syndrome.

Attributing bradycardia to intrinsic sinus dysfunction should occur only after ruling out AV block and then by exclusion of important conditions such as hypothyroidism, electrolyte abnormalities and drugs (Table 1).

A resting ECG done a few days earlier (Figure 2) shows more marked pauses in keeping with sinus node dysfunction, but again the patient was asymptomatic.

PACING OR NOT?
The question now is what to do about this arrhythmia, especially in anticipation of a procedure under general anaesthesia and taking
into account a history of syncope. Should the patient be paced temporarily, permanently or not?

If he were symptomatic due to the bradycardia, chronotropic incompetence, pauses or paroxysmal arrest of sinus node dysfunction, the answer to pacing would be “yes”. The decision to pace is straightforward with the obvious symptoms of syncope and dizziness. However, the diagnosis and hence pacing may be overlooked when the symptoms are limited to fatigue, reduced effort tolerance, dyspnoea and even cognitive impairment.

The syncope that he had in 2008 led to his referral for evaluation because at the time the ECG showed the same arrhythmia as the current ECG, namely short pauses and bradycardia. A very careful history of the syncopal episode suggested typical reflex neurally mediated or neurocardiogenic/vasovagal syncope. A tilt test was positive (with a vasodepressor effect and no cardio-inhibition, i.e. hypotension without bradycardia) and reproduced the symptoms at the time of the syncope. Furthermore, a prolonged ECG recording by Holter did not reveal any prolonged pauses: Even if

### TABLE 1: Approach to bradycardias

Any bradycardia, whether persistent, regular or intermittent with pauses, should prompt search for P-waves and analysis of the ECG for failure of conduction or failure of impulse generation. Although sinus node dysfunction or sick sinus syndrome are a cause of faulty impulse generation, the diagnosis is made only after excluding extrinsic causes.

<table>
<thead>
<tr>
<th>Bradycardias</th>
<th>Failure or suppression of impulse generation (i.e. sinus node problem)</th>
<th>Failure of impulse conduction (i.e. AV node problem)</th>
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<tr>
<td>Intrinsic</td>
<td>Failure or suppression of impulse generation (i.e. sinus node problem)</td>
<td>Failure of impulse conduction (i.e. AV node problem)</td>
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<td>Extrinsic</td>
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<td>Failure of impulse conduction (i.e. AV node problem)</td>
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<td>Hyperplasia</td>
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<td>Hyperautonomia</td>
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claimed to be asymptomatic, pauses of >2.5 seconds during waking hours or >4 seconds during sleep are recommendations for pacing. As he was otherwise asymptomatic and has remained so, there was/is no indication for permanent pacing.

MORBIDITY AND MORTALITY
Is sinus node dysfunction or sick sinus syndrome benign? Regarding prognosis, unlike AV block, mortality directly due to sinus node disease is rare. Where death has occurred, it is thought to have been related not so much to sinus arrest and asystole but more to bradycardia-induced tachycardia, the mechanism of which is likely to be bradycardia induced long QT and polymorphic VT or torsades de pointes. There is some association with “sick sinus syndrome” and the development of atrial fibrillation with its attendant risks of thromboembolism and requirements for anti-coagulation. However, it is mainly the morbidity related to this condition that impacts on routine cardiology. The symptoms may be non-specific and the ECG signs imprecise and frequently over-interpreted with diagnoses of dizziness and syncope incorrectly attributed to the chance finding of co-existent sinus node dysfunction. This has resulted in very significant levels of unnecessary and inappropriate pacing especially in certain sectors of the population.

PERI-OP MANAGEMENT
There is little doubt that this patient has sinus node dysfunction. Despite his not having experienced, or tests not demonstrating, symptomatic paroxysmal more severe sinus dysfunction, it is not impossible that this could develop. However, this possibility would be regarded as remote and no temporary pacing in anticipation of a surgical procedure or general anaesthetic is therefore indicated. It would be prudent in any patient with a risk of bradycardia to have external pacing facilities and atropine on hand in the operating room.

The correct answer is (e). The patient subsequently underwent the procedure under GA. It was uneventful. The finding was an oesophageal web.

CONCLUSIONS/LESSONS
- Sinus node dysfunction or sick sinus syndrome may be an incidental finding.
- The recognition of sinus node dysfunction is easily made if the sinus rhythm shows: irregularity that is not physiological, inappropriate tachycardia, chronotropic incompetence or inappropriate bradycardia.
- In a bradycardia, sinus node dysfunction is a diagnosis of exclusion of other potential life-threatening conditions.
- Pacing for sinus node dysfunction is indicated only if symptomatic and these symptoms cannot be accounted for by other diagnoses.
- Not all irregularly irregular pulses are atrial fibrillation; an ECG is required to make the diagnosis.