A 50-year-old man is referred to hospital by his GP. Mr Frederick Abel awoke in the early hours of this morning with an uncomfortable awareness of a fast heartbeat; the sensation has continued since then. Mr Abel complains that his chest feels ‘tight’ and he is slightly short of breath. While walking to the GP’s surgery Mr Abel felt light-headed, but did not faint. The GP noted a fast, irregular pulse with a rate of around 140 beats/min.

What challenges will this patient present?

- The combination of Mr Abel’s description of a rapid heartbeat and the GP’s clinical findings suggest some form of tachyarrhythmia
- The differential diagnosis for a patient with ‘palpitations’ is quite wide (see Table 20); however, if the problem persists, a combination of history, examination and ECG or rhythm strip will usually uncover the underlying cause and enable treatment to be started
- In some cases, a rhythm disturbance may come and go (paroxysmal arrhythmia), in which case the problem may have resolved by the time the patient presents to the doctor; this may present a difficult diagnostic challenge (see Case 15); ambulatory monitoring of the heart rhythm (24-hour tape or Holter monitor) may be diagnostic if the episodes occur frequently

On arrival in hospital Mr Abel is alert and talking, but appears pale and slightly sweaty, with cool peripheres. Initial observations reveal a pulse rate of 140 beats/min, which appears to be irregular. Mr Abel’s blood pressure is 130/70. Respiratory rate is increased at 20 breaths/min with oxygen saturation of 94% while breathing room air.

What immediate management is required?

- Check patency of airway and apply high-flow oxygen via a mask
- Establish intravenous access
- Connect the patient to a cardiac monitor
- Send blood for full blood count, electrolytes, liver function, glucose and thyroid function tests
- Obtain an ECG: if the ECG shows evidence of acute ST elevation myocardial infarction the patient should be managed according to the principles outlined in Case 1
- Request a chest radiograph

Having initiated these measures, it is important to establish the cause of Mr Abel’s fast heart rate. A careful history, clinical examination and close inspection of the ECG are required.

The patient’s breathing feels more comfortable following application of the oxygen, although the palpitations continue.

Mr Abel describes having a number of similar episodes in the past, usually on waking and settling within about 30 min. Mr Abel has never blacked out during an episode. His chest usually feels ‘tight’ during the palpitation with slight shortness of breath, but no significant chest pain. The palpitations usually feel irregular and fast, with some beats stronger than others. Mr Abel has never previously had an episode that has persisted and required hospital admission. Mr Abel was investigated one year ago for similar symptoms, at which time an ECG, blood tests and a 24-hour tape were normal.

Mr Abel has recently noticed that he has been getting increasingly breathless on exertion, particularly when walking briskly uphill. He is also slightly breathless when lying flat, which is eased by sleeping with three pillows. Mr Abel’s ankles swell during the day but are back to normal by morning.

In the past Mr Abel has suffered from high blood pressure, for which he takes bendrofluamide 2.5 mg daily. He also has mild asthma, and uses salbutamol via an inhaler as required. Mr Abel’s father had ischaemic heart disease and died from a myocardial infarction at the age of 60. He smokes 20 cigarettes per day and drinks two or
three glasses of wine most nights. On the night before his admission Mr Abel had consumed about double this quantity. Mr Abel works as a buildings inspector for the council and drives a car to work each day.

On examination he appears overweight, with an irregular pulse of 140–150 beats/min; the strength of the radial pulse appears to vary from beat to beat. Blood pressure remains 130/70. Oxygen saturation is 100% while breathing oxygen. Jugular venous pulsations are not visible.

Heart sounds are normal. Chest examination reveals bibasal inspiratory crepitations and quiet expiratory wheeze throughout both lung fields. There is pitting oedema of both ankles.

Inspection of the cardiac monitor and ECG rhythm strip reveals an irregular tachycardia with a rate between 150 and 170 beats/min. No P-waves are visible and the QRS complexes all appear similar in morphology (see Fig. 25).

### Table 20 Common causes of ‘palpitations’

<table>
<thead>
<tr>
<th>Cause</th>
<th>Symptoms</th>
<th>ECG</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ectopic beats</td>
<td>Patient describes intermittent, ‘missed’ beats or erratic ‘forceful’ beats, usually not fast. Systemically well</td>
<td>Broad or bizarre complexes interspersed within normal sinus rhythm (see p. 16)</td>
<td>Usually benign, but can be uncomfortable if frequent. Often exacerbated by anxiety, caffeine or sympathetic stimulants (e.g. salbutamol)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>Irregular, often fast, heart rhythm. May be associated light-headedness/breathlessness</td>
<td>Irregularly irregular rhythm; no P-waves; QRS complexes all appear similar to each other (see p. 15)</td>
<td>See text</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>Fast, regular palpitation</td>
<td>Rate usually close to 150/min and very regular. May be possible to see jagged ‘saw-tooth’ flutter waves between QRS complexes (see p. 17)</td>
<td>Atrium ‘flutters’ at 300 beats/min; ventricular rate determined by degree of ‘block’ at AV node; commonest is 2:1 block resulting in rate of 150 beats/min. 3:1 (100/min) or 4:1 (75 beats/min) also occur as can variable block leading to symptoms similar to atrial fibrillation</td>
</tr>
<tr>
<td>Sinus tachycardia</td>
<td>Fast, regular palpitation – often intermittent. May be symptoms of associated illness (e.g. thyroid disease) or anxiety</td>
<td>Rate usually &lt;150 beats/min. P-waves before QRS complexes; some variability in rate – unlike atrial flutter</td>
<td>Sinus tachycardia is often a feature of other serious illness – e.g. pulmonary embolism, sepsis, hypovolaemia; generally such patients do not present with palpitations as their main symptom. Anxiety is the commonest cause of symptomatic sinus tachycardia</td>
</tr>
<tr>
<td>Supraventricular tachycardia</td>
<td>Often very fast (‘too fast to count’); very regular; may be associated syncope</td>
<td>Usually narrow QRS complexes (although see Case 4); very regular; rate can be &gt;200 beats/min</td>
<td>Structurally normal heart in the majority of cases; some patients with recurrent episodes may have associated electrical anomalies (e.g. Wolff–Parkinson–White syndrome)</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>Fast, and regular; usually quite unwell – often present with syncope or acute shortness of breath</td>
<td>Always broad QRS complexes (&gt;12 s); regular. If P-waves are visible they do not relate to QRS complexes (‘dissociated’)</td>
<td>See Case 4 for more detailed discussion of the management of broad complex tachycardia</td>
</tr>
</tbody>
</table>
What is the likely cause of Mr Abel’s symptoms?
Common causes of palpitations, along with their historical and electrocardiographic features, are shown in Table 20.

The symptoms, signs and ECG features point to a diagnosis of acute-onset, fast atrial fibrillation as the cause for Mr Abel’s palpitations, as indicated by:
- Mr Abel’s description of a rapid, irregular heartbeat
- The irregular pulse on examination with variability in pulse volume
- The characteristic ECG findings (see p. 16)

What is the cause of the breathlessness and ankle oedema?
It is likely that Mr Abel has some pre-existing cardiac failure, as suggested by:
- Recent exertional dyspnoea
- Ankle swelling
- Orthopnoea
The onset of fast atrial fibrillation may have exacerbated this problem.
- The lack of coordinated atrial contraction prior to ventricular systole results in reduced cardiac output
- A rapid heart rate reduces the time for ventricular filling during diastole, again reducing cardiac output

What is the relevance of the previous episodes of palpitations?
- Mr Abel’s prior symptoms suggest a previous diagnosis of paroxysmal atrial fibrillation (see Table 21)

Table 21 Classification of atrial fibrillation (AF)

<table>
<thead>
<tr>
<th>Terminology</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>New-onset AF</td>
<td>AF of recent onset</td>
</tr>
<tr>
<td>Paroxysmal AF</td>
<td>Episodes of AF, terminating spontaneously within 7 days (and usually within 48 h)</td>
</tr>
<tr>
<td>Persistent AF</td>
<td>AF lasting more than 7 days</td>
</tr>
<tr>
<td>Permanent AF</td>
<td>AF lasting &gt;1 year, even with attempts to restore sinus rhythm</td>
</tr>
<tr>
<td>‘Lone’ AF</td>
<td>AF occurring in the absence of any structural heart disease or other precipitant</td>
</tr>
</tbody>
</table>

- It is possible that this episode will also resolve spontaneously
- However, given that the symptoms are continuing, with evidence of cardiac failure, Mr Abel will require further inpatient treatment and investigation

Results of further investigations are now available. Blood test results are as follows (for normal ranges see p. 229):
- haemoglobin 121; white blood cells 11.8; platelets 256
- Na 133; K 3.05; urea 6.7; creatinine 75
- albumin 38; alanine transaminase 121; alkaline phosphatase 140; bilirubin 13
- glucose 4.2
- thyroid function normal
What treatment is required?
When considering the treatment of a patient with fast atrial fibrillation, two key questions need to be considered.

- Should an attempt be made to restore sinus rhythm (‘rhythm control’)?
- Should the heart rate simply be slowed down (‘rate control’)?
  - the complex issues surrounding rate versus rhythm control are summarised in Box 8
  - in this case there is good evidence that the episode started acutely this morning, which increases the likelihood that sinus rhythm may be restored (‘cardioversion’)
  - although this may occur spontaneously, this is not guaranteed
  - techniques for cardioversion are summarised below

Are there any ‘correctable’ precipitating factors?
Potential precipitants for acute atrial fibrillation are summarised in Table 22. Failure to correct a precipitant such as electrolyte imbalance or thyroid dysfunction will reduce the likelihood of the success of other treatment measures, whereas correction may increase the chance of spontaneous resolution.

- In this case Mr Abel is noted to be hypokalaemic (probably as a result of his thiazide diuretic), which may have contributed. Potassium needs to be diluted when infused through a peripheral vein, and care will be required not to give fluid too rapidly in view of his pulmonary oedema
- Mr Abel’s heavy alcohol intake the night before his admission may also be implicated as a cause, which may need to be addressed in the future

Does the patient require immediate anticoagulation with heparin?
Atrial fibrillation (AF) results in incomplete emptying of the atrium during systole. Blood may ‘stagnate’ in the left atrial appendage leading to the formation of thrombus.

### Box 8 Rate control versus rhythm control

Restoration of sinus rhythm (‘rhythm control’) is usually considered the ‘gold standard’ treatment for atrial fibrillation. However, this is not always possible and even where it is achieved, the effect may be only temporary; a large proportion of patients will have reverted back into AF within 1 year. In some cases, therefore, clinicians will choose to adopt an approach which aims simply to control the heart rate (‘rate control’), accepting that the patient remains in AF. The patient’s symptoms are often well controlled with this approach, and the drugs required to achieve this are often better tolerated than those designed to induce and maintain sinus rhythm.

Although the decision is often complex, the following can be used as a guide.

- Cardioversion is generally recommended for new-onset AF unless the patient is elderly or has structural heart disease
- Rhythm control is preferred in patients with paroxysmal or persistent AF who are highly symptomatic.
- Pharmacological rhythm control of AF does not reduce the risk of stroke
- If drug therapy fails in either rate or rhythm control, referral to a cardiac electrophysiologist for catheter ablation therapy should be considered

### Table 22 Causes and precipitants of atrial fibrillation

<table>
<thead>
<tr>
<th>Type</th>
<th>Precipitants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac</td>
<td>Ischaemic heart disease, Valvular heart disease, Cardiomyopathies, Hypertensive heart disease</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Pulmonary embolism, Pneumonia (and other severe systemic infections), Bronchial carcinoma</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Electrolyte imbalances (e.g. hypokalaemia), Acute renal failure, Thyrotoxicosis</td>
</tr>
<tr>
<td>Toxic</td>
<td>Alcohol intoxication, Caffeine excess, Drug overdose (e.g. tricyclic antidepressant), Recreational drugs (e.g. cocaine)</td>
</tr>
<tr>
<td>Iatrogenic</td>
<td>Postoperative (especially following cardiac surgery), Sympathetic stimulant drugs (e.g. salbutamol, theophyllines)</td>
</tr>
</tbody>
</table>
If this moves from the heart (‘embolisation’) it may block an artery resulting in acute ischaemia or infarction of the area that the artery supplies. The commonest and often most devastating manifestation of this is an acute ischaemic stroke.

Embolisation can occur at any time, but is most common following electrical cardioversion, particularly where the AF has persisted for more than 48 h. Formal anticoagulation will reduce the risk of stroke in patients with AF. Recommendations for anticoagulation in AF have been produced by the National Institute for Health and Clinical Excellence (see Table 23). In practice, unless there is evidence of recent or ongoing bleeding, most patients presenting to hospital with acute AF will require treatment with heparin while plans are made for further treatment.

• Mr Abel fulfils these criteria and should be given initial treatment with heparin (e.g. enoxaparin 1 mg/kg twice daily).

Mr Abel is given 1 mg/kg of enoxaparin, which is prescribed twice daily. An intravenous infusion of 500 mL of normal saline containing 3 g of potassium chloride is commenced to run in over 4 h.

Following senior review by your consultant, it is decided that an attempt should be made to restore sinus rhythm.

How should sinus rhythm be restored?

• Restoration of sinus rhythm can be achieved by using drugs (‘chemical’ cardioversion) or by delivering an electric shock to the myocardium (‘electrical’ cardioversion); the latter usually requires a general anaesthetic.

• In the emergency setting, when the patient is severely unwell (hypotensive, persistent tachycardia >160 beats/min, ongoing chest pain, severe pulmonary oedema), immediate electrical cardioversion is recommended.

• If the patient is more stable, as in this case, an initial attempt at chemical cardioversion is usually undertaken, followed by electrical cardioversion if unsuccessful. A list of drugs that may be considered for chemical cardioversion is given in Table 24.

Mr Abel is treated with an infusion of intravenous amiodarone, 300 mg over 30 min, followed by oral amiodarone 200 mg three times daily. He has been prescribed enoxaparin 1 mg/kg twice daily.

Mr Abel’s heart rate is noted to slow to 110 beats/min over the next 2 h, but he remains in atrial fibrillation.

Repeat measurement of electrolytes after 12 h reveals correction of the potassium concentration (now 4.1 mmol/L).

Mr Abel is observed on a cardiac monitor in the medical admissions unit. He is noted to be less breathless while breathing oxygen, which is reduced to 28% via a Venturi mask, maintaining his saturation at 99%. Examination of Mr Abel’s chest reveals clearing of the bibasal crackles.

A transthoracic echocardiogram is undertaken, which reveals dilatation of the left ventricle and left atrium with moderate left ventricular systolic dysfunction. There is no evidence of valvular heart disease.

What further treatment options are available?

Initial attempts at chemical cardioversion have apparently failed, although Mr Abel’s heart rate has reduced, which may have accounted for the improvement in the symptoms of cardiac failure.

A number of further treatment options can now be considered.

---

Table 23 National Institute for Health and Clinical Excellence recommendations for antithrombotic therapy in acute atrial fibrillation (AF)

<table>
<thead>
<tr>
<th>1</th>
<th>A patient with acute AF who is receiving no or subtherapeutic anticoagulation therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• In the absence of contraindication, heparin should be started at initial presentation</td>
</tr>
<tr>
<td></td>
<td>• Heparin should be continued until full assessment has been made and appropriate oral antithrombotic has been initiated</td>
</tr>
<tr>
<td>2</td>
<td>For a patient with a confirmed diagnosis of acute AF of recent onset (&lt;48 h), oral anticoagulation should be used if:</td>
</tr>
<tr>
<td></td>
<td>• Stable sinus rhythm is not restored within the same 48 h period following onset of acute AF, or</td>
</tr>
<tr>
<td></td>
<td>• There are factors indicating a high risk of AF recurrence or</td>
</tr>
<tr>
<td></td>
<td>• It is recommended by the stroke risk stratification algorithm</td>
</tr>
<tr>
<td>3</td>
<td>In a patient with acute AF where there is uncertainty over the precise time of onset, oral anticoagulation should be used as for persistent AF</td>
</tr>
<tr>
<td>4</td>
<td>In cases of acute AF where the patient is haemodynamically unstable, any emergency intervention should be performed as soon as possible and the initiation of anticoagulation should not delay any emergency intervention</td>
</tr>
</tbody>
</table>
Inpatient electrical cardioversion prior to discharge

This has the advantage of potentially restoring sinus rhythm before discharge from hospital. In this case there is good reason to believe that the onset of AF occurred shortly before admission. If cardioversion can be performed within 48 h of the onset of AF (or if anticoagulation was initiated shortly after the onset of AF) the risk of embolisation is minimal. If the duration of AF is more prolonged, or is uncertain, it is usually recommended to anticoagulate the patient for at least three weeks before electrical cardioversion. If more urgent cardioversion is required, a transoesophageal echocardiogram can be used to confirm the absence of clot from the atrium before cardioversion.

Continuation of amiodarone and oral anticoagulation with warfarin, followed by outpatient electrical cardioversion if sinus rhythm is not restored (additional rate control as required)

This approach has the advantage of giving the patient the opportunity of spontaneous cardioversion (assisted by amiodarone), thereby avoiding the potential risk of the anaesthesia. However, the patient may remain symptomatic while in AF, particularly if rate control is difficult.

Long-term rate control and anticoagulation with warfarin

Adequate symptomatic control can often be achieved simply by controlling the heart rate. Furthermore, a large proportion of patients whose sinus rhythm is restored by cardioversion will revert to AF within one year. Although the patient will usually have to take anti-arrhythmic drugs lifelong as well as warfarin, some patients may prefer this to cardioversion.

This approach is generally preferred in older patients who are relatively asymptomatic, those with a longer duration of AF and those with structural or valvular heart disease.

Where possible it is important to involve the patient in this decision in relation to the risks and benefits of drugs, long-term anticoagulation and general anaesthesia.

Following discussion with Mr Abel it is decided to undertake inpatient electrical cardioversion. This is conducted under general anaesthesia the following day. Following 3 DC shocks at 200 J, sinus rhythm is restored; however, shortly after returning to the ward Mr Abel is noted to be back in AF with a heart rate of 110 beats/min. This is confirmed by 12-lead ECG, which shows no new acute ischaemic changes.

Mr Abel is no longer breathless at rest, and is not aware of any palpitations. He mobilises uneventfully on the ward with mild dyspnoea on exertion.

It is decided that further management should concentrate on providing rate control.

How can the heart rate be controlled for patients who remain in atrial fibrillation?

- Rate control in AF is achieved by reducing the transmission of electrical impulses from the atrium to the ventricle at the atrioventricular (AV) node
- A variety of drugs that can block transmission at the AV node are available and are listed in Table 25
- The choice will be dependent on factors related to the individual patient

Table 24 Drugs used for ‘chemical’ cardioversion

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage/administration</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone</td>
<td>300 mg intravenously or oral loading with 200 mg tds for 1/52, bd for 1/52 then 200 mg daily</td>
<td>Will require administration via a central venous cannula if prolonged intravenous use required; long half-life – slow onset of action following oral administration</td>
</tr>
<tr>
<td>Flecaïnide</td>
<td>300 mg orally or 2 mg/kg by intravenous infusion over 30 min</td>
<td>Avoid in ischaemic or other structural heart disease</td>
</tr>
<tr>
<td>Sotalol</td>
<td>80–240 mg bd orally</td>
<td>Avoid in asthma and uncontrolled heart failure</td>
</tr>
<tr>
<td>Propafenone</td>
<td>150 mg tds orally</td>
<td>Avoid in structural heart disease</td>
</tr>
<tr>
<td>Esmolol</td>
<td>5–200 μg/kg/min i.v.</td>
<td>Avoid in asthma and uncontrolled heart failure</td>
</tr>
</tbody>
</table>
Severe asthma is an absolute contraindication to treatment with beta-blockers. Mr Abel’s ‘asthma’ is described as mild, and may in fact represent smoking-induced chronic obstructive pulmonary disease (COPD) rather than ‘true’ asthma. The wheeze noted on admission may have been related to pulmonary oedema rather than bronchospasm. This should be considered a ‘relative’ contraindication and a small dose of a cardioselective beta-blocker (e.g. bisoprolol 2.5 mg daily) may be tolerated. The patient must be advised to stop immediately if they become wheezy.

Mr Abel’s amiodarone is stopped. He starts taking digoxin, with three oral doses of 500 μg, followed by 125 μg daily. Mr Abel also starts taking ramipril 2.5 mg daily, in view of his left ventricular failure, and his bendroflumethiazide is stopped. Mr Abel is given warfarin, orally. It is decided not to commence beta blockade unless the above measures fail to control the heart rate.

Over the next two days Mr Abel’s heart rate settles to 80/min, although he remains in atrial fibrillation. Mr Abel is mobile around the ward without breathlessness and is weaned off his oxygen.

What further management/follow-up is required following discharge from hospital?

A number of issues need to be considered on discharge.

- **Anticoagulation monitoring**: warfarin requires regular blood testing to measure the international normalised ratio (INR). Stabilisation of the dose usually requires at least five days of treatment with daily monitoring. If the patient has already been on the drug for more than five days the stable dose may already have been established. Further testing after discharge is normally undertaken by the patient’s GP. It is often helpful to speak directly to him/her to discuss the timing of the next blood test.

  - **Monitoring of electrolytes**: Mr Abel was hypokalaemic on admission; although the bendroflumethiazide has been stopped, he has started taking ramipril, which can also cause electrolyte imbalances. Digoxin toxicity may be precipitated by hypokalaemia, which may also increase the risk of further arrhythmias.

  - **Digoxin level**: although not essential if the patient’s heart rate is controlled, measurement of digoxin level after one week of treatment may give an indication whether the dose is appropriate. This is more important for elderly patients and those with renal impairment, in whom accumulation of the drug may result in toxicity.

  - **Heart rate control**: confirmation that the heart rate is controlled following discharge may be achieved by asking the GP to review the patient after one week. A 24-hour tape will also help to identify episodes of poor rate control, which may necessitate the introduction of a beta-blocker or increase in the digoxin dose (if the level is subtherapeutic).

  - **Alcohol**: heavy alcohol intake may precipitate arrhythmias, and chronic use may result in a cardiomyopathy; the patient should be advised to cut down alcohol consumption to <21 units per week (14 units for women).

  - **Cigarette smoking**: discontinuation of smoking should reduce thrombotic risk and may also be important if there is underlying ischaemic heart disease. The patient may require help to do this, such as with the use of nicotine replacement.

### Table 25

<table>
<thead>
<tr>
<th>Drug name</th>
<th>Dosage/administration</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digoxin</td>
<td>1.5 mg orally in three divided doses over 24 h followed by 125–250 μg daily</td>
<td>Slow onset of action – intravenous administration rarely justified. Lower doses in elderly patients or those with renal impairment</td>
</tr>
<tr>
<td></td>
<td>Or 500 μg i.v. infusion over 30 min</td>
<td></td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>Bisoprolol 2.5–5 mg daily orally; atenolol 25–50 mg daily orally</td>
<td>Avoid in asthma and uncontrolled heart failure</td>
</tr>
<tr>
<td>Calcium antagonists</td>
<td>Verapamil 40 mg orally tds or diltiazem 90–180 mg bd</td>
<td>Avoid in severe heart failure</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>200 mg orally tds for 1/52 reducing to bd for 1/52 then 200 mg daily</td>
<td>Generally avoided for rate control due to long-term side effects</td>
</tr>
</tbody>
</table>

Mr Abel’s amiodarone is stopped. He starts taking digoxin, with three oral doses of 500 μg, followed by 125 μg daily. Mr Abel also starts taking ramipril 2.5 mg daily, in view of his left ventricular failure, and his bendroflumethiazide is stopped. Mr Abel is given warfarin, orally. It is decided not to commence beta blockade unless the above measures fail to control the heart rate.

Over the next two days Mr Abel’s heart rate settles to 80/min, although he remains in atrial fibrillation. Mr Abel is mobile around the ward without breathlessness and is weaned off his oxygen.
Caffeine: excessive caffeine consumption should be avoided; ideally the patient should be advised to consume decaffeinated coffee, although complete abstinence from caffeine consumption is not an absolute requirement.

Driving: if the patient has experienced any syncopal episodes they should cease driving for a period of three months. In the absence of syncope (as in this case) the patient may drive providing the symptoms from the arrhythmia are not likely to impair their driving ability in the view of the doctor treating them.

Consideration of underlying myocardial ischaemia: this is an important cause of atrial fibrillation and Mr Abel has risk factors for ischaemic heart disease: cigarette smoking, hypertension and a family history of myocardial infarction. Exercise testing, myocardial perfusion scanning or coronary angiography should be considered following discharge.

Mr Abel returns to the outpatient clinic six weeks after discharge. He has felt much better for the past two weeks with no further feelings of ‘palpitation’. Mr Abel’s exercise tolerance has improved and he no longer experiences orthopnoea. Mr Abel has reduced his alcohol intake dramatically since discharge and has stopped smoking. Clinical examination reveals that his pulse now feels regular and ECG demonstrates that he has reverted to sinus rhythm. Chest examination is normal with a blood pressure of 135/70. Mr Abel has not needed to use the salbutamol since discharge. His digoxin is stopped and he starts taking bisoprolol 2.5 mg daily, as a means of reducing the risk of further episodes. Mr Abel is advised to stop this immediately if he becomes wheezy. It is determined to continue his warfarin in view of his previous history, which was suggestive of ‘paroxysmal’ atrial fibrillation. Mr Abel is booked for an outpatient treadmill ECG and further follow-up to establish whether there is evidence of myocardial ischaemia.

CASE REVIEW

This case describes a 50-year-old man who presented to his GP with a prolonged episode of palpitations. ECG revealed fast AF, and symptoms suggested that there was co-existent cardiac failure. Possible precipitants for the arrhythmia included heavy alcohol consumption on the previous night, hypokalaemia (probably diuretic induced) and underlying myocardial ischaemia.

After initial stabilisation with oxygen and correction of the hypokalaemia Mr Abel was given intravenous amiodarone in an attempt to restore sinus rhythm. Mr Abel was also prescribed subcutaneous LMWH to reduce the risk of thrombus formation during the arrhythmia and subsequent embolisation. Although the heart rate slowed following the amiodarone he remained in atrial fibrillation. An echocardiogram confirmed a degree of left ventricular impairment with dilatation of the left atrium and ventricle. Electrical cardioversion was attempted, but only restored sinus rhythm for a short period; he was therefore commenced on digoxin to control his heart rate and discharged from hospital on this combined with warfarin and ramipril for the heart failure. He was advised to reduce his alcohol intake and stop smoking at the time of discharge from hospital.

At follow-up he had ‘spontaneously’ reverted to sinus rhythm, enabling the digoxin to be stopped. He was started on a small dose of the beta-blocker bisoprolol as prophylaxis against further episodes. An outpatient treadmill ECG was arranged to investigate the possibility of underlying myocardial ischaemia.

KEY POINTS

- Palpitations usually imply a cardiac tachyarrhythmia.
- Careful history, clinical examination and ECG during the episode will usually enable identification of the causative rhythm disturbance.
- AF is a common arrhythmia that can compromise cardiac output, particularly when the rate is very fast.
- Treatment of AF can involve either rate control or attempts to restore sinus rhythm.
- Anticoagulation should be considered for all patients presenting to hospital with AF to reduce the risk of systemic embolisation.
- Precipitating factors, such as electrolyte imbalances, should always be sought and, where possible, corrected.
- Underlying valvular, ischaemic or other structural heart disease should be sought by careful investigation including echocardiography and exercise testing.
Further reading