The fluttering patient: an approach to the patient with palpitations

All doctors may encounter patients having palpitations or an abnormal electrocardiogram. Determining the diagnosis can be a challenge, while medical therapies and strategies can appear confusing to the uninitiated. Furthermore, invasive electrophysiological techniques have improved our diagnostic abilities as well providing an opportunity for curing pathology. This series of articles revises the commonly encountered tachyarrhythmias. The first article addresses the approach to the patient with palpitations.

Introduction

Palpitations are common and cause great distress (Mayou et al, 2003). Defined as an abnormally perceived heartbeat, the patient's definition may differ from the doctor's. Some mean chest discomfort or episodic breathlessness. Others refer to a single extra thud as 'palpitations' while others may suggest a racing heart was 'fluttering'. Palpitations can be frequent or seldom but are often alarming. Presentations may be arrhythmia specific, or with another medical illness, such as an exacerbation of obstructive airways disease or pneumonia. Few palpitations indicate clinically important arrhythmia but a careful approach is required to recognize high-risk individuals.

Taking the history

The history remains a powerful tool. First, ensure you understand what the patient is referring to. Do not assume that physicians who have seen the patient before you have got the diagnosis correct. Try to cover key areas shown in *Figure 1*.

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Each spell of palpitations may differ in the same patient. Therefore, generalized questions may not yield an accurate history; instead discuss key episodes separately. A sudden offset of fast palpitations suggests supraventricular tachycardia.

Pre-syncope and true syncope necessitate urgent assessment. Determine the exact circumstances of the collapse. Was it witnessed? If so, the witness can provide diagnostic information. Long pauses in heart rhythm lead to patients turning very pale, passing out suddenly, with the appearance that they have died, before flushing of the skin presages a return of consciousness, often with transient disorientation and agitation. Patients with collapse have poor recall and many deny loss of consciousness. Other aspects of the story can determine whether consciousness was lost. Was there any injury? How did the patient call for the ambulance?

Specifically seek features suggesting vasovagal syncope (Kapoor, 2000). Was the situation typical for vasovagal syncope – standing for a long period in a warm environment such as a bank queue? Was stress a component, e.g. having a blood test or procedure? The patient may complain of feeling distant, his/her vision fogging before the collapse. Urinary incontinence is common in vasovagal syncope, as are cerebral anoxic seizures if the patient is held upright. Recurrent fainters develop manoeuvres to terminate the sensation such as lying down or bending over.

Ask specifically for other signs of decompensation. Associated dyspnoea occurs in many tachyarrhythmias and the patient's exercise tolerance may be drastically reduced. Tachyarrhythmias are effectively an exercise stress test, and those with underlying coronary artery disease may develop angina. Alternatively, angina and ischaemia may trigger the arrhythmia.

Ask about a family history of an inherited cardiac disorder, arrhythmia or premature sudden cardiac death. Commonly, patients do not know the specifics of the death; a label of 'heart attack' is given. However, sudden deaths before the age of 40 years may be attributable to inherited arrhythmias such as arrhythmogenic right ventricular cardiomyopathy, Brugada syn-

Figure 1. Key questions to address in the history.

Describe what you mean by 'palpitations'	
Is it one extra thud or many?	
When it occurs, does the heart feel fast or slow?	
At what rate does the heart go?	
Ask the patient to tap out the heart beat – is it regular or irregular?	
How long does it last for? Probe to know with certainty	
Do the symptoms start suddenly? Probe to be sure	
How do the symptoms stop: suddenly or fade away over a period of time?	
Has the patient developed any techniques for terminating the symptoms (breath holding or gulping cold drinks)	
Is there a pattern to the symptoms – always on the bus to work? Do they occur when the patient is on holiday or more relaxed?	
Are there any triggers? Common triggers include:	Heightened anxiety or stress
	Caffeine
	Alcohol
	Other stimulants including illegal substances (cocaine, ecstasy) and medications (salbutamol inhalers)

drome or a long QT syndrome (Huikuri et al, 2001). Ask for specifics: how did the death occur, did an autopsy happen, was an underlying heart disorder known about?

Ask about a personal or family history of epilepsy. Some 'epilepsy' represents reflex anoxic seizures occurring secondary to arrhythmia. Alternatively, evidence suggests that seizures can cause acutely abnormal cardiac electrophysiological states, predisposing to arrhythmias and sudden death (sudden unexpected death in epilepsy) (Jehi, 2010).

Determining the presence of psychiatric symptoms is also important. Patients may be having panic attacks associated with physiological sinus tachycardia (Barsky et al, 1994; Mayou et al, 2003). A strong psychosocial interplay is involved (Weber and Kapoor, 1996). However, pathological tachycardias make patients feel anxious, particularly if symptoms are frequent and incapacitating.

When completing the history, consider the prognostic significance of any underlying structural heart disease. A comprehensive enquiry about previous cardiac history is essential, as is a complete drug history. Many drugs given for cardiovascular disease are potentially pro-arrhythmic and a full drug history must be obtained. Try to identify those that prolong the QT interval such as erythromycin, haloperidol and sotalol (Roden, 2004). Some drugs may induce atrial fibrillation (van der Hooft et al, 2004).

Physical examination

Physical examination is often normal. Look for features of thyrotoxicosis, anaemia, heart failure or airways disease. Look for signs of a pre-existing cardiac condition, including valvular disease, cardiomyopathy, hypertension, pulmonary hypertension and congenital heart disease.

Exceptionally, the patient may be experiencing the arrhythmia at the time of the consultation. Once a significantly abnormal pulse is detected, check the ABC of airway, breathing and circulation, and make strenuous efforts to obtain a 12-lead electrocardiogram recording immediately. Documentation of the arrhythmia may cut out the need for extensive subsequent investigations. Once an electrocardiogram has been recorded, look for features of atrioventricular dissociation during tachycardia, such as intermittent cannon waves in the jugular venous pulse, and variable intensity of the first heart sound, which are signs that a broad QRS tachycardia is ventricular rather than supraventricular in origin.

Investigations

Send blood tests for full blood count, electrolytes and thyroid function. Anaemia causes sinus tachycardia and dyspnoea, while hyperthyroidism commonly triggers atrial arrhythmias.

A resting 12-lead electrocardiogram may reveal an underlying abnormality to diagnose the arrhythmia or suggest an underlying cardiac disorder. Look for the short PR interval and delta waves of Wolff–Parkinson–White syndrome (*Figure 2*). Check the QT and the heart rate-corrected QT intervals for long QT syndrome (*Figure 3*), and precordial leads V1–V3 for the changes of Brugada syndrome (*Figure 4*). The presence of pathological Q waves may suggest a scar from previous myocardial infarction which can act as a substrate for re-entry pathways and tachyarrhythmias. Broadened QRS complexes demonstrate underlying conduction problems while PR interval prolongation and dropped beats indicate first or second degree heart block respectively. Patients with cardiomyopathy or heart failure rarely have a normal electrocardiogram.

Echocardiography should be undertaken in most patients. It is mandated in those with structural heart disease, those collapsing with impaired consciousness, and in patients with atrial fibrillation or flutter.

Continuous ambulatory electrocardiogram recording is frequently used, typically for 24–48 hours (Thavendiranathan et al, 2009). The patient wears a monitoring

Figure 2. Wolff–Parkinson–White syndrome. In resting sinus rhythm, a shortened PR interval and slurred upstroke of the QRS (delta wave) can be seen.

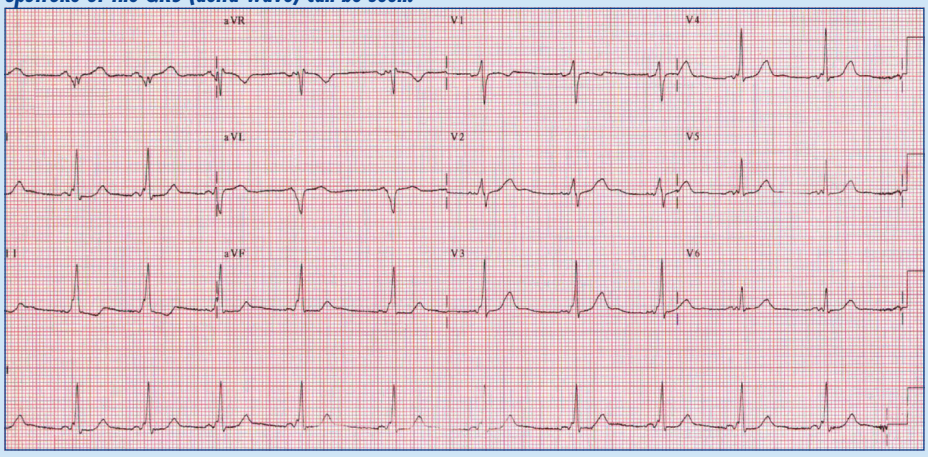
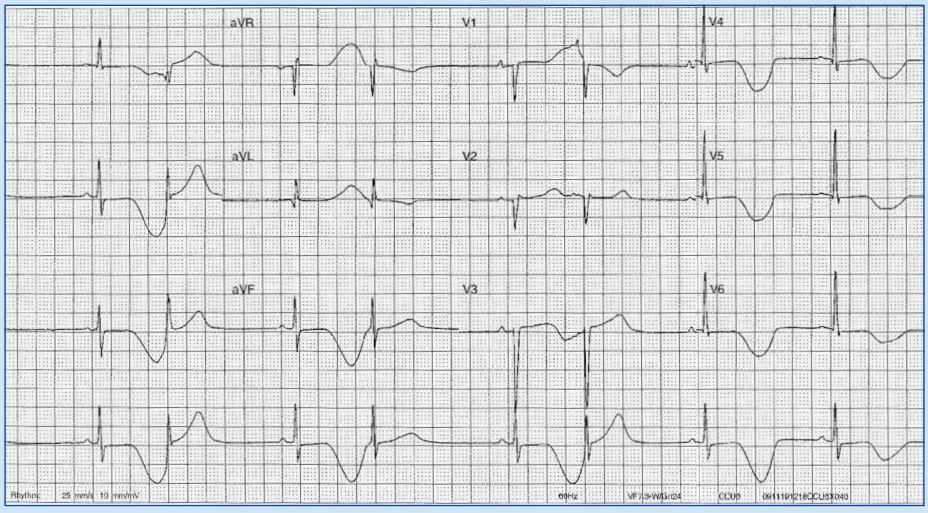


Figure 3. Long QT. Exaggerated long QT is shown here, with a QTc of 528 ms. A QTc of 440 ms is accepted as normal. Note gross T wave abnormalities inferolaterally.



device while continuing his/her normal activities, and should maintain a diary of symptoms which is returned for analysis. The purpose is both to document the arrhythmia causing the symptoms and to detect asymptomatic but potentially relevant arrhythmias. Benign arrhythmias are a universal finding when the period of monitoring is sufficiently prolonged. Typically these consist of atrial or ventricular ectopic beats occurring in isolation, periods of sinus tachycardia, or occasional dropped beats with relative pauses in rhythm. No action is needed when these arrhythmias are not associated with symptoms. If associated with the patient's usual symptoms of palpitation, only reassurance is required although a minority may require antiarrhythmic drug therapy.

If symptoms occur less than daily, a single Holter is unlikely to capture an episode of symptoms. Instead use a cardiac event monitor for a 1- or 2-week period. The heart rhythm is monitored continuously, but the electrocardiogram is only recorded to the device's memory on occasions when it falls outside certain preset limits of heart rate or if the patient presses an activation button. Such devices have several advantages over continuous ambulatory electrocardiogram recording. The quantity of recorded electrocardiogram data is much less which simplifies the analysis and reporting, longer periods of monitoring are practicable which makes capture of a symptomatic episode more likely, and the patient activation feature allows exact matching of symptoms to heart rhythm. A disadvantage is that potentially relevant arrhythmia may not fall outside the preset triggering parameters for recording and will therefore not be stored.

Longer term recording devices are available when patients have infrequent episodes of symptoms which elude diagnosis. Implantable loop recorders allow monitoring for up to 2 years, and have both automated and patient-activated electrocardiogram storage functions, similar to cardiac rhythm event monitors. Similar in size and shape as USB memory sticks, they are implanted subcutaneously under local anaesthetic in the left anterior chest wall. Full sterile operative technique and prophylactic antibiotic cover are used. The patient activates electrocardiogram storage by placing a wand over the device if a symptomatic bout occurs. Patients should then attend the electrocardiography department promptly to have the data downloaded and analysed. The diagnostic yield of these devices is excellent (Reiffel et al, 2005). They often resolve difficult arrhythmias and have allowed the diagnosis of true epilepsy when arrhythmias were questioned.

Ectopics

Ventricular and atrial ectopic beats are a common cause of palpitations. Frequently, an additional 'thump' is felt, followed by a pause and then another thump. Patients are often alarmed by the apparent pause and lack of heartbeat. The history alone can be diagnostic and Holter monitors are not required unless the burden of symptoms is to be determined.

Those with a significant burden of ectopic beats may need medical therapy with beta-blockers, or with calcium-channel blockers with antiarrhythmic action such as verapamil or diltiazem.

Where symptoms persist despite drug therapy, or when continued drug therapy is unacceptable to the patient, catheter ablation can be performed to reduce the frequency of the ectopic beats. Ablation is most likely to be successful when there are unifocal ventricular ectopics arising from the right or left ventricular outflow tract (i.e. the QRS morphology is always identical, and has an inferior QRS axis of +80° to +110°, strongly positive in electrocardiogram leads II, III, aVF with a left or right bundle–branch block pattern) (*Figure 5*). Multifocal ectopics and those associated with structural heart disease are more difficult to eradicate by ablation. In most, only reassurance is required with symptoms spontaneously settling, although recurrence is common.

Conclusions

This article discusses an approach to the patient with palpitations. Subsequent articles will cover atrial fibrillation, atrial flutter, supraventricular tachycardias and ventricular arrhythmias. While cardiac

Figure 4. Brugada syndrome. There is abnormal and highly characteristic ST elevation with upward convexity and right bundle–branch block pattern seen in V1 and V2. Brugada syndrome is caused by an inherited sodium channel defect that is associated with arrhythmia and sudden cardiac death.

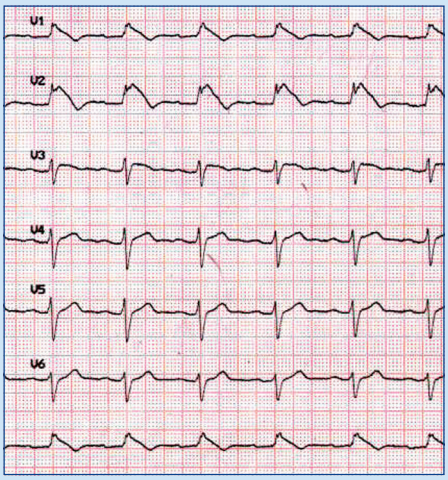
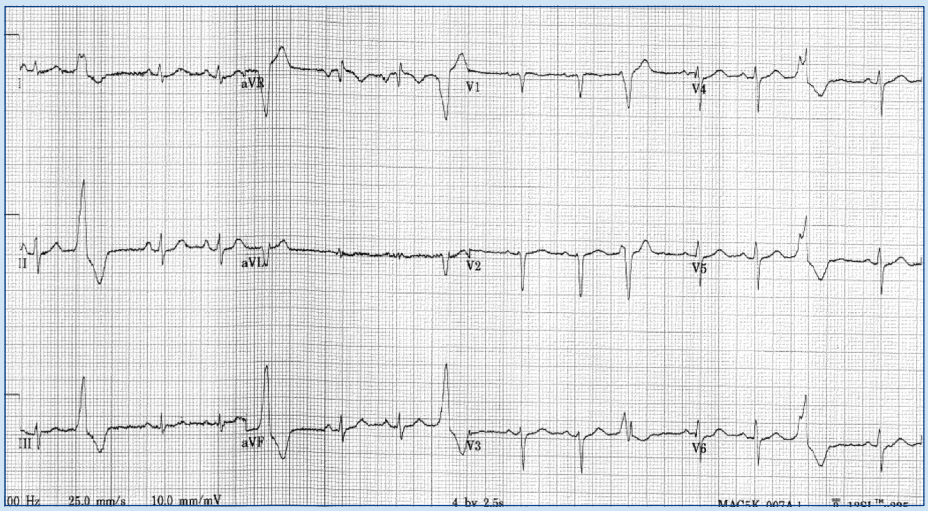


Figure 5. Frequent ventricular ectopics. Note that abnormal appearance compared to the other QRS complexes. Each is identical with an inferior axis. This is from right ventricular outflow tract and can be ablated if necessary.



arrhythmias can be difficult to diagnose, a sensible approach can make significant headway. BJHM

Conflict of interest: none.

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KEY POINTS

- A detailed history should be sought even if one was previously taken. Focus on how the palpitations started and stopped.
- Establish whether any associated breathlessness and chest pain started before or after the palpitations started.
- Always seek a family history of palpitations or of sudden death.
- Resting electrocardiographs are often normal and do not exclude underlying pathology.
- Cardiac event monitors that record for longer periods than 24 hours are often more useful than a single Holter recording.