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16.2.2 Syncope and palpitation 3285 and reduction in quality of life regardless of the underlying cause. The exceptions are cardiac causes of syncope, which have been re- ported to have 1-year mortality rates as high as 18–33%. An im- portant aim in the evaluation of syncope is to identify this subgroup of patients: clues may come from the history, examination, and the 12-lead ECG (Box 16.2.2.1). Differential diagnosis The initial evaluation of the patient with possible transient loss of consciousness should include history, examination, supine and up- right blood pressure, and 12-lead ECG (Fig. 16.2.2.1). It is important to establish that loss of consciousness (syncope) occurred to enable differentiation from nonsyncopal causes such as falls, drop attacks, and transient ischaemic attacks. In the absence of 'red flag' features of cardiac syncope (Box 16.2.2.1) and with a normal 12-lead ECG, a single episode of syncope requires no further investigation or treat- ment, other than reassurance. In patients with recurrent syncope, or a single episode in a high-risk individual, further investigation and treatment will depend on the suspected diagnosis. The causes of syncope can be subdivided on the basis of patho- physiology, namely (1) neurally mediated—or reflex—syncope, (2) orthostatic hypotension, (3) cardiac causes, and (4) cerebrovas- cular or psychogenic causes (Table 16.2.2.1). The patient history is most important. For example, it may strongly suggest a vasovagal origin, or an epileptic seizure. However, the diagnosis may be com- plicated by an overlap in features, such as convulsive movements during a vasovagal episode due to anoxic convulsive seizures. It is increasingly recognized that many patients who attend clinics for epilepsy have been misdiagnosed and are suffering from recurrent syncope: some of these patients have potentially lethal ventricular arrhythmias for which they should be receiving treatment. Neurally mediated syncope There are many disorders of autonomic control that can cause syn- cope. The most common is neurocardiogenic syncope, or simple faint, which is due to an increased sensitivity of normal reflex re- sponses. By contrast, autonomic dysfunction may produce abnormal neurovascular control that results in orthostatic hypotension. Vasovagal syncope Vasovagal syncope is the most common cause of syncope. It can affect all age groups and varies from infrequent episodes associated with obvious triggering factors to frequent unprovoked collapses, which may be debilitating. The pathophysiology most commonly involves venous pooling of blood and reduced venous return to the heart in response to upright posture. Reduced cardiac output and blood pressure stimulate arterial baroreceptors with resultant increased sympathetic activity and catecholamine levels. The vig- orous contraction of relatively empty ventricles results in the ac- tivation of mechanoreceptors that would normally respond to stretch in the left ventricular wall. Afferent nerve fibres conduct to the cerebral medulla and activate the reflex withdrawal of periph- eral sympathetic tone and activation of vagal parasympathetic ac- tivity. The resultant vasodilatation and bradycardia cause reduced cerebral perfusion and loss of consciousness. However, there is de- bate about these mechanisms and other factors may be involved in the aetiology of syncope, as illustrated by the documentation of neurocardiogenic syncope—despite cardiac denervation—in orthotopic heart transplant recipients. Certainly, it is well recog- nized that vasovagal syncope can result from other stimuli, such as pain, emotional shock, or the sight of blood: in these instances, the reflex activation is central in origin. The development of tilt testing has allowed the study of the patho- physiology of neurocardiogenic syncope. The patient is strapped to a tilt table and is tilted, head upright, usually at 70° for up to 45 min. Protocols that use additional provocation with isoprenaline or nitrates are commonly used. Blood pressure and cardiac rhythm are moni- tored throughout the tilt test. In neurocardiogenic syncope, the patient classically maintains normal blood pressure initially, until the sudden onset of syncope is associated with severe hypotension and brady- cardia, often preceded by tachycardia. These features resolve with re- turn to the supine posture. Some patients have a mainly vasodepressor

response, with hypotension and little change in heart rate, while others have a marked cardioinhibitory response, with severe bradycardia or asystole of several seconds duration (Fig. 16.2.2.2). Most have a mixed response of hypotension and bradycardia. Carotid sinus hypersensitivity An abnormal sensitivity of a normal reflex is responsible for syn- cope. Activation of the carotid sinus baroreceptors (e.g. by phys- ical pressure, such as carotid sinus massage) results in sympathetic withdrawal and parasympathetic activation. Bradycardia is usually a prominent feature. Situational reflex-mediated syncope In susceptible individuals, similar abnormal reflex sensitivity can result in syncope in response to afferent activity from some other Box 16.2.2.1 Features associated with cardiac syncope History of syncopal episode • Occurs during exertiona • Occurs when supinea • Lack of prodromal warninga • Associated palpitations (preceding syncope is higher risk)a • Associated chest pain • Traumatic injurya Past medical history • Known structural heart diseasea • Previous myocardial infarctiona • History of heart failurea • Valvular heart diseasea Family history • Family history of sudden deatha Examination • Presence of murmura • Signs of heart failurea • Carotid bruit 12-lead ECG • Evidence of atrioventricular blocka • Bundle branch blocka • Evidence of previous infarctiona • Left ventricular hypertrophya • Long QTc intervala • Features of Brugada syndromea a Denotes a ‘red flag’ feature of the history or examination that suggests urgent assessment is required and that there is a high chance of recurrent syncope.

section 16 Cardiovascular disorders 3286 mechanoreceptor activation. Syncopal responses to cough, mictur- ition, defecation, or swallowing have been reported. Orthostatic hypotension Hypotension may occur in patients in whom there are abnor- malities in the autonomic control of cardiovascular function. Abnormalities of afferent or efferent pathways, or of peripheral vascular control, can result in low blood pressure in the upright posture (i.e. orthostatic hypotension). The clinical presentation can be divided into four subgroups: • Initial orthostatic hypotension—symptoms occurring within 30 s of standing diagnosed with active standing blood pressure meas- urement (syncope is rare). • Classical orthostatic hypotension—symptoms occur between 30 s and 3 min diagnosed with active standing blood pressure meas- urement. It is usually due to classical autonomic failure or drug therapy (syncope is rare). • Delayed—symptoms occur between 3 and 45 min of standing and there is a more prolonged prodrome with a gradual fall in blood pressure resulting in syncope with or without a reflex (abrupt blood pressure fall) component, usually diagnosed with tilt table testing. • Reflex syncope—typically occurring in young healthy females, onset between 3 and 45 min; blood pressure is usually maintained until syncope and diagnosed by tilt table testing. Postural orthostatic tachycardia syndrome is not classically asso- ciated with syncope. TLOC present? (history) Presentation of patient with probable TLOC (may include ambulance or referral data) Initial syncose evaluation (H&P exam, ECG, supine, and standing BP) No TLOC (a) Syncope TLOC - non syncopal • Epileptic seizure • Psychogenic TLOC • TLOC, rare causes Certain or highly likely diagnosis High-risk of short-term serious events Early evaluation & treatment Ancillary tests followed by treatment Explanation, no further evaluation Low-risk but recurrent syncopes Low-risk single or rare recurrences Uncertain diagnosis (See panel (b)) Risk stratification (See panel (c)) Treat appropriately Act as needed Start treatment
Fig. 16.2.2.1 (a) Flow diagram for the initial evaluation and risk stratification of patients with syncope. (b) Clinical features that can suggest a diagnosis on initial evaluation. (c) High-risk features (that suggest a serious condition) and low-risk features (that suggest a benign condition) in patients with syncope at initial evaluation in the emergency department. AF, atrial fibrillation; ARVC, arrhythmogenic right ventricular cardiomyopathy; AV, atrioventricular; BBB, bundle branch

block;
BP, blood pressure; bpm, beats per minute; ECG, electrocardiogram; ED, emergency department; H&P exam, history and physical examination; ICD, implantable cardioverter defibrillator; LQTS, long QT syndrome; LVEF, left ventricular ejection fraction; OH, orthostatic hypotension; SCD, sudden cardiac death; SVT, supraventricular tachycardia; TLOC, transient loss of consciousness; VT, ventricular tachycardia. From Brignole M, et al. (2018). 2018 ESC Guidelines for the diagnosis and management of syncope. Eur Heart J, 39, 1883–948. By permission of Oxford University Press.

16.2.2 Syncope and palpitation 3287 (b) Fig. 16.2.2.1 Continued

section 16 Cardiovascular disorders 3288 (c) Fig. 16.2.2.1 Continued

16.2.2 Syncope and palpitation 3289 Orthostatic hypotension is more common in elderly patients, where it may be multifactorial, often exacerbated by drugs (Box 16.2.2.2). Nocturnal symptoms may occur, with a fall in blood pressure exacer- bated by sudden rising from a warm bed. Autonomic failure is an uncommon cause of syncope and patients may present with other features, including disturbances of bowel, bladder, or sexual function. Pure autonomic failure can be acute or chronic, primary (of unknown origin) or secondary to systemic dis- ease. Multiple system atrophy is characterized by autonomic dys- function, parkinsonism, and ataxia. Orthostatic hypotension may be a marked feature (the Shy–Drager syndrome), with additional parkinsonian features or cerebellar symptoms. Secondary auto- nomic failure can result from the central or peripheral involvement of certain diseases, including multiple sclerosis, cerebral tumour, diabetes, and amyloidosis. Cardiac syncope Loss of consciousness of cardiac origin may result from some sub- stantial disturbance of cardiovascular function or from abnormal- ities of heart rhythm, with resultant reduced cerebral perfusion. The importance in establishing the diagnosis of cardiac syncope is the associated adverse prognosis, which may be improved with appro- priate treatment. The probability of cardiac syncope is increased in the presence of structural cardiovascular disease identified from the history, clinical examination, or the ECG (Box 16.2.2.1). Tachycardia Syncope may be caused by tachycardia, most commonly ventricular, but supraventricular tachycardia can also be associated with loss of consciousness if it is very fast or in patients with structural heart disease. Syncope, rather than cardiac arrest, may result from self- terminating ventricular tachycardia or from sustained tachycardia with hypotension at the onset, but with a subsequent recovery of blood pressure. Whether or not a tachycardia causes syncope is re- lated to its rate, underlying left ventricular function, and the patient’s baroreceptor sensitivity. Cardiac arrhythmia should be considered in all patients with structural heart disease presenting with syncope. Ventricular tachy- cardia most commonly occurs in patients with structural heart dis- ease (e.g. prior myocardial infarction) but may also occur in patients with structurally normal hearts. For example, torsades de pointes in a patient with the long-QT syndrome is an important diagnosis to consider in young people with a history of loss of consciousness and possible epilepsy, in whom the episodes of collapse may be due to syncope caused by ventricular arrhythmia. Bradycardia A sudden decrease in heart rate, onset of ventricular standstill, or asystole may be a cause of syncope. When due to sinoatrial dysfunc- tion (sick sinus syndrome) this is not associated with a poor prog- nosis, but syncope due to intermittent complete atrioventricular block is. Syncope in a patient with a permanent pacemaker may in- dicate pacemaker malfunction. Structural cardiovascular disease Aortic stenosis may be associated with syncope, particularly during sudden exertion when the demand for increased cardiac output cannot be met because of the mechanical

obstruction. Hypertrophic cardiomyopathy may also be associated with syncope, either because (a) (b) Fig. 16.2.2.2 Cardioinhibitory response to tilt testing. (a) After 6 min of head-up tilting at 70° the patient complained of presyncope. Heart rate was 60/min but blood pressure was 70 mm Hg. (b) By 7 min the patient had lost consciousness, associated with an asystolic pause of 10 s duration and an unrecordable blood pressure. Recovery was rapid following the patient's return to the supine position. Table 16.2.2.1 Causes of syncope Neurally mediated Vasovagal or neurocardiogenic syncope Carotid sinus hypersensitivity Situational (micturition, defecation, cough, swallow) Orthostatic hypotension Primary autonomic failure Pure autonomic failure Multiple system atrophy (parkinsonian, cerebellar) Secondary autonomic failure (diabetic, amyloid neuropathy) Postural orthostatic tachycardia syndrome Drugs and alcohol Volume depletion (haemorrhage, diarrhoea) Cardiac syncope Bradycardia Atrioventricular block Sinoatrial disease Tachycardia Ventricular arrhythmia Structural heart disease Previous myocardial infarction Cardiomyopathy Structurally normal heart Long-QT or Brugada syndrome Supraventricular arrhythmia Aortic stenosis Structural cardiovascular disease Hypertrophic cardiomyopathy Atrial myxoma or thrombus Pulmonary embolism Cerebrovascular or psychogenic Neurological Migraine Subclavian steal Vertebrobasilar disease Psychogenic Anxiety, depression, and hyperventilation

section 16 Cardiovascular disorders 3290 of outflow obstruction or ventricular arrhythmia. Obstruction of blood flow through the mitral valve by an atrial myxoma or thrombus is an uncommon cause of syncope. Certain other cardiac diseases may be associated with loss of consciousness by a variety of mechanisms (arrhythmia, reflex-mediated, or haemodynamic), including myocardial infarction, pulmonary embolism, congenital heart disease, or cardiac tamponade. Vascular diseases may also be involved, such as aortic dissection and extracranial vascular disease. Cerebrovascular or psychogenic causes of syncope When epilepsy is excluded, neurological conditions are rare causes of loss of consciousness, but possible diagnoses include migraine, vertebrobasilar vascular disease, and subclavian steal syndrome. However, in most cases these will not result in true syncope. A psychogenic origin of loss of consciousness implies the absence of neurally mediated, neurological, or cardiac abnormalities, and may occur in association with anxiety, depression, and conversion disorders. For instance, apparent syncope may occur during tilt testing but with normal pulse and blood pressure. Hyperventilation may be an associated mechanistic factor in psychogenic syncope. Assessment of the patient with syncope Careful assessment of the patient's history, a full physical examin- ation and the 12-lead ECG will indicate a likely diagnosis in over 50% of patients with a history of syncope. Further investigations will be prompted by the initial evaluation (Fig. 16.2.2.1). History The importance of the clinical history in assessing a patient with syn- cope cannot be overemphasized. If possible, an eyewitness descrip- tion of the patient during the syncopal event should be obtained. Features associated with an increased risk of cardiac syncope should be sought (Box 16.2.2.1). Provocative factors Vasovagal syncope is classically associated with upright posture, often with aggravating circumstances such as prolonged standing, a hot environment, or hunger. However, episodes may also occur when seated, including while driving. Specific stimuli may be re- sponsible for neurocardiogenic syncope in susceptible individuals. Ventricular arrhythmia, in particular torsades de pointes in the long-QT syndrome, may be provoked by sudden stimuli such as a noise (e.g. an alarm clock), or exercise (particularly swimming). Exertional syncope is a feature of aortic stenosis or hypertrophic cardiomyopathy. Syncope may also be triggered by coughing, mic- turition, and more rarely, swallowing or laughing. Preceding symptoms Sweating and feeling hot or nauseated may precede vasovagal syn- cope. Cardiac arrhythmia may be associated with palpitation, chest pain, or

breathlessness. Bradycardia, such as intermittent complete heart block, may produce no preceding symptoms and may cause loss of consciousness without warning. Sinoatrial dysfunction is a cause of symptoms of dizziness and light-headedness in addition to syncope. A psychogenic origin may be suggested by multiple associ- ated symptoms including hyperventilation, paraesthesiae in fingers and lips, palpitation, and chest pain, which may precede syncope. Epilepsy may be preceded by a characteristic aura, which would strongly point away from cardiac syncope as the diagnosis. The syncopal episode In syncope the duration of loss of consciousness is usually short, with recovery after a few minutes. A longer duration of loss of con- sciousness suggests an alternative diagnosis. An exception to this is when the patient has remained upright during the attack, possibly aided by well-meaning but misguided helpers. Incontinence is a feature of epileptic seizure but may also occur (uncommonly) with syncope. Description of the patient during the episode is of great value. The classic description of an episode of syncope due to car- diac arrhythmia—in particular sudden-onset severe bradycardia— is of a sudden loss of colour, becoming deathly pale, with flushing on recovery (Stokes–Adams attack). Cyanosis may be a feature of an arrhythmic origin of syncope. Convulsive movements during the episode would raise the possibility of epilepsy, but they also occur with cardiac syncope. Although any cause of syncope can be associ- ated with injury, its absence may point to a nonsyncopal or psycho- genic origin. The recovery period By contrast to the postictal phase following epilepsy, there is com- monly a rapid recovery of cerebral function following syncope. Vasovagal syncope may be followed by persisting nausea or vomiting and general malaise; in older people this phase may be prolonged. Family history There are a few specific causes of syncope in which a family history of syncope or sudden death may have prognostic significance. Long- QT syndrome is hereditary and may be associated with sudden death. A family history of syncope is of adverse prognostic signifi- cance in hypertrophic cardiomyopathy. Investigation The investigation of cardiac disease and arrhythmia are dealt with in the appropriate chapters, but the approach to the patient with syn- cope is described briefly here. Dependent on the history, further in- vestigations may not be necessary with the exception of a 12-lead ECG. For example, the diagnosis of vasovagal syncope is a clinical one and other investigations are likely to have a low diagnostic yield. By contrast, if the history or examination points to a clear cause of Box 16.2.2.2 Common drugs that may cause postural hypotension • Diuretics • α-Adrenergic receptor blockers • β-Adrenergic receptor blockers • ACE inhibitors • Angiotensin II receptor antagonists • Calcium channel blockers • Nitrates • Opiates • Ethanol • Tricyclic antidepressants • Bromocriptine • Phenothiazines • Levodopa

 syncope, investigations appropriate to the underlying cause should be performed. Electrocardiogram An ECG should be performed on all patients with syncope. This may provide evidence of aetiology of syncope, such as the long-QT syndrome, or of structural heart disease, such as prior myocardial infarction or left ventricular hypertrophy. An arrhythmia may be documented if it is sustained, and there may be evidence of sino- atrial disease or conduction system disease, such as ‘trifasicular’ block, bundle branch block, or first-or second-degree block. In those over the age of 40 carotid sinus massage (CSM) with digital pressure to the carotid artery for up to 5 s may cause marked bradycardia in carotid sinus hypersensitivity, with pauses of more than 3 s duration. Although it is recommended that CSM is avoided in patients with carotid bruits or a stroke or TIA within past 3 months, the incidence of adverse events is extremely rare. Active standing Measurement of lying and standing blood pressure is made using a sphygmomanometer (not an automated blood pressure cuff, as is commonly done) for a period of 3 min or until diagnostic criteria are reached. The test is diagnostic when there is a symptomatic fall

in systolic blood pressure from baseline of 20 mm Hg or more or diastolic blood pressure of 10 mm Hg or more, or a decrease in sys- tolic blood pressure to less than 90 mm Hg. An asymptomatic fall, although suggestive, is not diagnostic when taken in context of the background incidence of postural hypotension which is up to 30% in the unselected elderly population and up to 60% in the hospital- ized population. Ambulatory monitoring Documentation of cardiac rhythm during syncope is extremely useful, especially if it was associated with palpitations, but is difficult to obtain because of the intermittent and usually infrequent nature of the symptom. In patients admitted with high-risk syncope, in- hospital monitoring is recommended. Holter monitoring is usually only useful in individuals with recurrent syncope occurring more than once a week or in those where there are underlying ECG ab- normalities (in particular conduction abnormalities which do not automatically meet the criteria for bradycardiac pacing). Real-time event recorders are of limited value in the investigation of syncope because they require a conscious patient to make the recording. Loop recorders, which have automatic rhythm detection algorithms and which can also be activated by the patient facilitating retro- spective rhythm analysis, can be used for periods of 1–4 weeks and are useful for investigating syncope with an intermediate frequency. Implantable loop recorders have a greater diagnostic yield than con- ventional monitoring and can now be implanted in an outpatient setting. They are indicated in the evaluation and treatment of infre- quent (>4-week interval), high risk, and recurrent syncope. Tilt testing When the history is suggestive of vasovagal syncope, the tilt test may be of value in confirming the diagnosis, but a negative test does not exclude the diagnosis. Adjuvant provocation (isoprenaline or ni- trate) may increase the sensitivity, but the incidence of false positive tests with tilt testing has been reported as 5–20%. As such, its use is probably best limited to investigation of recurrent symptoms with an atypical history in patients in whom there are no features to sug- gest cardiac syncope. Electrophysiological testing Abnormal sinus node function or evidence of atrioventricular con- duction disease may be elicited by electrophysiological testing, but demonstrating bradycardia during ambulatory monitoring more reliably makes both of these diagnoses. In patients with structural heart disease in whom arrhythmia is suspected, pro- grammed electrical stimulation of the ventricles can induce sus- tained monomorphic ventricular tachycardia. This is a relatively specific response which shows that the patient is at risk of recurrent ventricular arrhythmia and makes an arrhythmic origin of syncope likely. However, recent guidelines on device implantation suggest that an electrophysiological study is no longer routinely required in a patient with impaired ventricular function and syncope likely to be cardiac in origin. If there is enough clinical suspicion, then implantable cardioverter defibrillators can be offered to these pa- tients without electrophysiological testing. It is important to note that the diagnostic yield of electrophysiological testing is low in pa- tients with a structurally normal heart. Other investigations Assessment for structural heart disease is important. Physical exam- ination will detect most significant valve disease, but other diag- noses, such as hypertrophic cardiomyopathy or atrial myxoma, may produce little in the way of clinical signs. An echocardiogram is therefore worthwhile in cases where the diagnosis remains un- clear. Exercise testing is useful in patients with a history of syncope during or immediately after exercise. Exercise testing is diagnostic if Mobitz II second- degree or third-degree atrioventricular block de- velop during exercise even without syncope. Troponin measurement is not indicated in patients with syncope in the absence of features suggestive of an acute coronary syndrome. Approximately 10% of patients over the age of 60 presenting to the emergency department with syncope will have an elevated troponin, and although this is an independent risk factor for subsequent ser- ious events, the finding rarely changes management appropriately or contributes to the final diagnosis. A strong suspicion of

diagnoses other than syncope should lead to other investigations, including electroencephalography and brain imaging, but these have a low diagnostic yield in patients with syn- cope and should not be routine. Management Neurocardiogenic syncope may require no treatment other than re- assurance and avoidance of provocative factors. Syncope has several effects on lifestyle. Simple lifestyle measures may be employed to improve symptoms in specific situations: for example, increased fluid and salt intake. Where there is warning be- fore syncope occurs this may be used to prevent injury or complete syncope by adopting a position lying down or with feet elevated. It is crucial for those who suffer syncope to avoid situations that might put them at harm, such as swimming alone or bathing (showering is preferred). Management of vasovagal syncope, bradycardia, and cardiac ar- rhythmia are discussed in Chapters 16.4 and 24.5.4. In up to one-third

 of patients, the aetiology of syncope may not be found: these patients have a good outcome unless they have underlying heart disease. Palpitation The symptom of palpitation is defined as an awareness of one's heart beating. This may be due to an awareness of an abnormal heart rhythm, but it may also be due to an abnormal awareness of normal rhythm. A careful and detailed history can provide a likely diag- nosis. The most important aim in investigation is to correlate symp- toms with cardiac rhythm. History A description of the symptom should include an estimate of heart rate, duration of symptom, regularity of rhythm, suddenness of onset and offset. It may be helpful to ask the patient to tap with their finger to describe their palpitation. Trigger factors, including exer- cise, and aggravating factors such as alcohol and caffeine should be detailed. The length of history may be of interest. Sinus tachycardia An awareness of a rapid heart rate of gradual onset and offset is often associated with feelings of alarm and panic in patients with anxiety. Premature/ectopic beats Symptomatic atrial and ventricular premature or ectopic beats com- monly occur in normal individuals, and often generate considerable anxiety resulting in consultation. In the absence of coronary disease, premature ventricular ectopic beats (PVCs) at a frequency of 1 per hour or more were recorded during Holter monitoring in the Framingham study in 33% of men and 32% of women. PVCs have also been recorded in 0.8% of a healthy military population during a standard 12-lead ECG. These are im- portant factors to remember when discussing their significance with the patient. The patient may describe 'missed beats' or forceful beats. These symptoms relate to the pause that follows a premature beat. The premature beat produces a short diastolic filling interval and the low ventricular volume results in reduced ventricular contraction with a small stroke volume. However, the subsequent pause provides a long diastolic filling period and the resultant stretching of the ventricular walls is associated with an increased and forceful systolic contraction. The combination of the diminished premature beat and the enhanced postextrasystolic beat is responsible for the symptoms. Benign ectopy is indicated by the absence of a history of other cardiovascular symp- toms or family history of sudden death, their occurrence at rest and resolution with exercise, and a normal clinical cardiovascular exam- ination and resting ECG. Multifocal ventricular ectopy, and PVCs at a frequency of more than 20 000 in 24 h, are more indicative of poten- tially significant cardiac pathology and require further investigation. Atrial fibrillation This common arrhythmia may produce a variety of symptoms depending on ventricular rate, irregularity, and persistence. Paroxysmal atrial fibrillation is characterized by self-terminating episodes of atrial fibrillation, when there may be a rapid and irregular ventricular response. The patient is aware of an increased heart rate and often describes the irregular nature of the symptom. The vari- ations in diastolic interval produce symptoms by similar mechan- isms to that described earlier for premature beats, with

‘missed’ and ‘forceful’ beats. Patients with sinoatrial dysfunction may be most symptomatic on termination of the atrial fibrillation, which can be followed by sinus bradycardia or prolonged sinus pauses. Atrial fib- rillation may be persistent or permanent, and the severity of symp- toms will be related to the ventricular rate and irregularity. Paroxysmal supraventricular tachycardia A history of sudden-onset, rapid, regular palpitation in a healthy pa- tient with no underlying structural heart disease is suggestive of parox- ysmal supraventricular tachycardia. It may stop spontaneously or with vagotonic manoeuvres, or the patient may have had to attend hospital for intravenous therapy. In addition to palpitation, patients commonly report fatigue, malaise, light-headedness, or dyspnoea, but because they have normal hearts such episodes of tachycardia are usually well tolerated. Polyuria is a common associated symptom, which results from the release of atrial natriuretic peptide secondary to atrial stretch. Ventricular tachycardia Ventricular arrhythmias can present with the symptom of palpita- tion, but more severe symptoms such as syncope or cardiac arrest also occur. Characteristically the symptom of palpitation would be the sudden onset and offset of a rapid regular heart rhythm. A his- tory of structural heart disease should be sought. Investigation Electrocardiogram The first aim is to document cardiac rhythm during symptoms. This may be possible with a standard ECG if the arrhythmia is sustained or persistent. Atrial or ventricular premature beats, or evidence of struc- tural heart disease (e.g. myocardial infarction), may be documented. The presence of pre-excitation indicates the diagnosis of Wolff–Parkinson–White syndrome and suggests symptoms due to episodes of atrioventricular re-entry tachycardia. Other ECG signs indicative of primary electrical heart disease are: a corrected QT interval greater than 460 ms or less than 320 ms (long or short QT syndrome); right bundle branch block with ‘coved’ ST elevation (Brugada syndrome); epsilon waves and/or T wave inversion with QRS duration greater than 100 ms in the right precordial ECG leads (arrhythmogenic right ven- tricular cardiomyopathy); and high voltages in the precordial leads with Q wave formation and ST changes (hypertrophic cardiomyopathy). Ambulatory monitoring The success of ambulatory monitoring in documenting the rhythm during symptoms will be dependent on the frequency of symptoms. If they occur daily, then a 24 or 48 h Holter recording should suffice. However, palpitation is often infrequent and other patient-activated devices can be of more value. These include hand-held, patient- activated event recorders that allow the telephonic transmission of recordings. These devices do not allow retrospective recording and require symptoms of sufficient duration to allow their use. However, there are now devices producing high quality single lead ECG re- cording that can be used with a smartphone and purchased directly by the patient. Shorter episodes may also be captured using loop re- corders: the newest devices are the size of a large plaster, are attached

 on the left upper part of the chest, can record for up to 2 weeks, and are waterproof. Ultimately, implantable loop recorders may be helpful where symptoms are infrequent, and they may also be effective in monitoring therapy once implanted for diagnostic purposes. The most recent devices are small enough to be classed as ‘injectable’, and they may even be implanted in the outpatient clinic setting. Other investigations Thyroid function and a full blood count are of particular importance in patients with atrial arrhythmias or sinus tachycardia, respectively. Electrolytes are routinely analysed. In patients with paroxysmal symptoms, a history of hypertension, sweating, and anxiety during attacks, urinary metanephrines for the investigation of phaeo- chromocytoma are indicated. Echocardiography is performed in most patients with palpitations and documented arrhythmias: in pa- tients with ventricular ectopy, however, it is usually indicated only in those with suspected structural heart disease, a very high burden of ectopy, or those at a high risk of development of serious ventricular arrhythmias or sudden

cardiac death. Electrophysiological studies Invasive studies are of most value in determining the mechanism of a previously documented tachyarrhythmia, particularly with a view to treatments such as radiofrequency catheter ablation. Management Documentation of the cardiac rhythm during palpitation allows ap- propriate management, with reassurance as the only treatment in those with sinus tachycardia or premature beats. The treatment of other cardiac arrhythmias is discussed in Chapter 16.4. Lifestyle advice Advice regarding lifestyle with palpitations revolves around re- assurance where it is felt to be benign and avoiding precipitants where these can be identified. Although caffeine, other stimulants, alcohol, and stress are often quoted as potential triggers (and this may be true of ectopy, for example), it is much more common for many arrhythmias to occur without any avoidable trigger. Exercise as a trigger for palpitations is unusual and may signify adrenaline- dependent arrhythmias such as some forms of ventricular tachy- cardia (see Chapter 16.4). Driving restrictions may apply for both palpitations and syncope. In the United Kingdom clear guidance is provided by the Driver and Vehicle Licensing Agency (DVLA) as to who can and cannot drive with these symptoms, investigations that are required, and the dur- ation of driving bans for both a normal driving licence and heavy goods/passenger vehicle licences. FURTHER READING Benditt DG, Sutton R (2005). Tilt-table testing in the evaluation of syncope. J Cardiovasc Electrophysiol, 16, 356–8. Brignole M, et al. (2018). 2018 ESC guidelines for the diagnosis and management of syncope. Eur Heart J, 39, 1883–948. Brignole M, et al. (2018). Practical instructions for the 2018 ESC guide- lines for the diagnosis and management of syncope. Eur Heart J, 39, e43–e80. Grubb BP (2005). Neurocardiogenic syncope and related disorders of orthostatic intolerance. Circulation, 111, 2997–3006. NICE (2010). Transient loss of consciousness. Clinical guideline. https:// www.nice.org.uk/guidance/cg109 Raviele A, et al. (2011). Management of palpitations: a position paper from the European Heart Rhythm Association. Europace, 13, 920–34. Shen W-K, et al. (2017). ACC/AHA/HRS Guideline for the evalu- ation and management of patients with syncope: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines, and the Heart Rhythm Society. Circulation, 136(5), e60–e122.

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