

Chapter 1

Tachycardia

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In adults, tachycardia is defined as a heart rate faster than 100 beats per minute (bpm). This may represent a normal physiological response, a sign of systemic illness, or primary cardiac pathology [1]. There are several possible cardiac rhythms associated with tachycardia. Identifying the underlying rhythm is central to the diagnostic process and directs management. Classifying these rhythms according to the width of the QRS complex and the regularity of the rhythm (as seen on an ECG) helps to simplify this process (Table 1.1) [2].

SINUS TACHYCARDIA

Sinus tachycardia is the most commonly encountered rhythm disturbance. In the majority of cases, this is an appropriate physiological response mediated by the sympathetic nervous system to an identifiable cause, which may be benign or pathological (Box 1.1) [3]. In the context of mental health, sinus tachycardia may be experienced during episodes of agitation, anxiety or panic. Sympathomimetic and anticholinergic drugs are also important causes to consider, including clozapine which causes a transient sinus tachycardia in 25% of patients, usually limited to the first six weeks of treatment [4]. Among pathological causes, people with serious mental illness (SMI) are at higher risk of sepsis [5] and pulmonary embolism [5,6], while sinus tachycardia is also associated with clozapine-induced myocarditis [7], neuroleptic malignant syndrome, and serotonin syndrome [8]. Hyperthyroidism and, more rarely, phaeochromocytoma may present with both psychiatric symptoms and sinus tachycardia [9,10]. The reader is directed to other chapters for detailed information on sepsis (Chapter 72), venous thromboembolism (Chapter 18), myocarditis (Chapter 8), neuroleptic malignant syndrome (Chapter 85), serotonin syndrome (Chapter 86), and hyperthyroidism (Chapter 79).

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Table 1.1 Differential diagnosis of tachycardia according to the length of the QRS complex and regularity of rhythm [2].

| | Narrow QRS (≤ 120 ms) | Broad QRS (> 120 ms) |
|-----------|--|--|
| Regular | Sinus tachycardia Supraventricular tachycardia Atrioventricular re-entrant tachycardia (AVRT) Atrioventricular nodal re-entrant tachycardia (AVNRT) Atrial flutter (with regular atrioventricular block) Focal atrial tachycardia | Monomorphic ventricular tachycardia (VT) Any regular narrow-complex tachycardia with aberrant conduction (e.g. bundle branch block/accessory pathway) |
| Irregular | Atrial fibrillation (AF) Atrial flutter with varying atrioventricular block Multifocal atrial tachycardia | Torsade de pointes Polymorphic VT Ventricular fibrillation AF with aberrant conduction (bundle branch block/accessory pathway) |

ATRIAL FIBRILLATION

Atrial fibrillation (AF) is the second most prevalent rhythm disturbance, occurring in 0.4–1% of adults [11]. Risk increases significantly with age [11]. Alcohol and stimulant use, hyperthyroidism, heart failure, hypertension, and chronic lung disease are associated with AF, all of which are more prevalent in patients with SMI (Table 1.2) [12]. AF can cause acute cardiac decompensation presenting as pulmonary oedema or myocardial ischaemia (see Chapters 67 and 68), as well as longer-term complications such as thromboembolic disease (e.g. stroke; see Chapter 82) [13].

SUPRAVENTRICULAR TACHYCARDIA

Conventionally, supraventricular tachycardia (SVT) refers to any tachycardia other than AF that originates above the level of the ventricles, i.e. involving the atria, the atrioventricular node, or the bundle of His [2]. Atrioventricular nodal re-entrant tachycardia (AVNRT), atrioventricular re-entrant tachycardia (AVRT), atrial flutter, and focal atrial tachycardia are the most common forms of SVT and each is associated with its own distinct pathophysiology and management [14]. Among people with SMI, alcohol and stimulant use may precipitate SVT (see Box 1.1) [15]. Ischaemic heart disease is also an important risk factor [16], the incidence of which is higher in people with SMI [17]. Among the general population, SVT rates are higher in women and those older than 65, although in the absence of ischaemic heart disease, SVT tends to present in younger people with a mean age of 37 [16].

Box 1.1 Common or important causes of tachycardia: those associated with serious mental illness are highlighted in italic

Sinus tachycardia

Emotional/physical arousal: anxiety/panic/agitation

Pain

Circulatory compromise:

Sepsis

Pulmonary embolism

Hypovolaemia including haemorrhage

Heart failure

Myocardial ischaemia

Anaemia

Hyperthyroidism

Electrolyte disturbance (hypokalaemia, hypomagnesaemia)

Pregnancy

Postural orthostatic tachycardia syndrome

Inappropriate sinus tachycardia

Orthostatic intolerance

Alcohol/opiate/benzodiazepine withdrawal

Serotonin syndrome

Neuroleptic malignant syndrome

Drugs:

Salbutamol

Caffeine

Cocaine

Amphetamine

Cannabis

Clozapine

Tricyclic antidepressants

Carbamazepine

Methylphenidate

Supraventricular tachycardia [2,15,16]

Wolff–Parkinson–White syndrome (AVRT)

Electrolyte disturbances (hypokalaemia/hyperkalaemia, hypomagnesaemia)

Ischaemic heart disease

Drugs:

Alcohol

Cocaine

Amphetamine

Caffeine

Atrial fibrillation [12]

Older age

Sepsis

Pulmonary embolism

Heart failure

Valvular heart disease

Hypertension

Chronic lung disease and lung cancer
 Hyperthyroidism
Electrolyte disturbance (hypokalaemia, hypomagnesaemia)

Drugs:

Atropine
 Alcohol
 Caffeine
 Cocaine
 Amphetamine

Ventricular tachycardia [23–26]

Myocardial infarction
Cardiomyopathy
Structural heart disease
Electrolyte disturbances (hypokalaemia/hyperkalaemia, hypomagnesaemia)
Prolonged QTc interval (congenital or acquired)
Brugada syndrome (phenotype associated with antipsychotics)
Eating disorders

Drugs:

Cocaine
 Amphetamines
 Tricyclic antidepressants
QTc prolonging medication including antipsychotics (see Chapter 3)
 Digoxin

VENTRICULAR TACHYCARDIA

Ventricular tachycardia (VT) is less common but is associated with high mortality and is the leading cause of sudden cardiac death [18]. The majority of cases are experienced in the context of structural heart disease, myocardial infarction or cardiomyopathy (both ischaemic and non-ischaemic) [19]. Although a specific association between VT and SMI has not been investigated, sudden cardiac death is significantly more prevalent in the psychiatric population and particularly among those taking antipsychotic medication and people with eating disorders [20,21]. Torsades de pointes (TdP), an irregular polymorphic VT, is of particular relevance due to its association with many antipsychotics and other psychotropic medications that prolong the QT interval (see Chapter 3). Despite this, TdP is still relatively rare, with an annual incidence of 0.16% in general hospital inpatients [22].

DIAGNOSTIC PRINCIPLES

History

- 1 Define cardiac symptoms.
 - a Palpitations (Box 1.2): if these are paroxysmal (i.e. intermittent), ask the patient to tap out the rhythm; this may provide information on the rate and the regularity of

Box 1.2 Clinical assessment of paroxysmal palpitations

- Palpitations are defined as the abnormal sensation of one's own heartbeat.
- They may be associated with tachyarrhythmias but can also be experienced during other abnormal cardiac rhythms such as ectopic beats or bradyarrhythmias (see Chapter 1.2) [28].
- Common causes include anxiety and somatisation (31%), paroxysmal atrial fibrillation (16%), and paroxysmal supraventricular tachycardia (10%) [29].
- Although psychiatric symptoms are an important risk factor for a non-cardiac cause of palpitations, 13% of such patients have an underlying cardiac abnormality and so further investigation may be warranted in the presence of other cardiac symptoms or red flag features [30].

the heartbeat during the palpitations (if irregularly irregular, strongly suggestive of paroxysmal AF).

- b Symptoms of haemodynamic compromise, e.g. chest pain, shortness of breath, syncope/presyncope.
 - c Symptoms of heart failure, e.g. orthopnoea (shortness of breath on lying flat), paroxysmal nocturnal dyspnoea (sensation of shortness of breath that wakes a patient from their sleep), swollen ankles.
 - d Symptoms of myocardial infarction (assessment of chest pain including character, site, and radiation; nausea/vomiting, sweating).
- 2 Determine possible precipitants including exercise, stress, drugs, or alcohol.
 - 3 Symptoms suggestive of systemic illness.
 - a Sepsis: fever, rigors, presyncope/syncope, confusion, symptoms related to infective source.
 - b Dehydration/hypovolaemia: recent evidence of volume loss (diarrhoea/vomiting, reduced oral intake, blood loss).
 - c Hyperthyroidism: weight loss despite increased appetite, oligomenorrhoea, emotional lability, heat intolerance.
 - d Anxiety/panic: paraesthesia, breathlessness, association with psychosocial stressors.
 - 4 Medication history including any new medications or recent dose changes/withdrawal, paying particular attention to any sympathomimetic or anticholinergic drugs (e.g. clozapine).
 - 5 Past medical history, including:
 - a ischaemic, structural, or valvular heart disease or heart failure
 - b chronic lung disease (e.g. chronic obstructive pulmonary disease/obstructive sleep apnoea)
 - c thyroid disease
 - d diabetes mellitus
 - e previous tachyarrhythmias
 - f eating disorder (purging behaviour may be associated with electrolyte disturbance)
 - g panic attacks/anxiety.

- 6 Family history of sudden cardiac death/unexplained death under 40 or tachyarrhythmias [27].
- 7 Social history including alcohol and tobacco use, illicit substance use, and consumption of caffeinated drinks (see Chapter 46).
- 8 Perform a mental state examination, exploring for any psychiatric symptoms that may be associated with autonomic activation, e.g. panic, anxiety, or fear in the context of delusional beliefs or hallucinatory experiences.

Examination

- 1 In an emergency (e.g. the unconscious patient), resuscitate using the ABCDE approach and refer to emergency medical services.
- 2 Observations: heart rate, blood pressure, temperature, respiratory rate, oxygen saturations.
- 3 In stable patients, perform a cardiovascular examination paying particular attention to the following.
 - a Inspection: dyspnoea, raised jugular venous pressure, swollen ankles (evidence of heart failure).
 - b Palpation: examine pulse for rate, rhythm, character, and volume.
 - c Auscultation: murmurs (valvular heart disease), S3/S4 (heart failure), pulse deficit (additional heartbeats that do not correspond to a palpable pulse are a sign of AF), chest (pulmonary oedema).
 - d Palpation for sacral and pedal oedema (heart failure).
- 4 Focused examination if underlying cause suspected, as in the following examples.
 - a If infection is suspected, focused examination for potential sources (e.g. chest).
 - b If hyperthyroidism suspected: fine tremor, sweaty palms, exophthalmos/lid lag, palpate for goitre (see Chapter 12 for focused examination).
- 5 Lying and standing blood pressure may elicit postural drop in blood pressure indicative of haemodynamic compromise.

Investigations

- 1 ECG (see Box 1.3 for descriptions of ECGs for common or important tachyarrhythmias with examples).
- 2 Bloods:
 - a full blood count (anaemia, high/low neutrophil count)
 - b renal function (hypovolaemia, hypokalaemia/hyperkalaemia)
 - c bone profile (hypomagnesaemia, hypocalcaemia)
 - d C-reactive protein (infection)
 - e thyroid function (hyperthyroidism)
 - f antipsychotic levels (toxicity)
 - g troponin (if suspecting an ischaemic event or myocarditis)
 - h D-dimer (see Chapter 18 to guide use of this test)
 - i brain natriuretic peptide (heart failure)
 - j HbA_{1c} (diabetes mellitus)
 - k blood sugar (diabetic ketoacidosis/hyperosmolar hyperglycaemic state).

Box 1.3 ECG characteristics of different tachyarrhythmias [32]

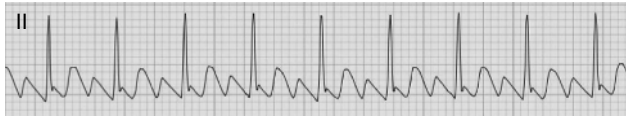
- Sinus tachycardia: P waves preceding every QRS complex, QRS complex following every P wave.



- Atrial fibrillation: irregularly irregular rhythm without P waves



- Atrial flutter: saw-tooth pattern reflecting atrial contractions at 300 bpm best seen in leads II, III and aVF. Usually narrow QRS complexes at 150 bpm (2 : 1 AV node conduction, as shown here).



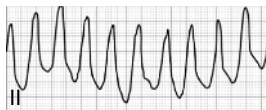
- Supraventricular tachycardia (atrioventricular re-entrant tachycardia or atrioventricular nodal re-entry tachycardia): regular tachycardia 140–300 bpm. Narrow QRS complexes (unless aberrant conduction). ST depression and T-wave inversion possible even in the absence of coronary artery disease.



- Wolff–Parkinson–White in sinus rhythm: short PR interval <120 ms. Delta wave: slowly rising QRS complex. QRS prolongation >110 ms. ST-segment and T-wave changes.



- Monomorphic ventricular tachycardia: regular rhythm, very broad QRS complexes (>160 ms) typically in the elderly or those with structural heart disease.



- Torsade de pointes: variable QRS complexes which 'twist' around isoelectric line. aVL



- 3 Urine toxicology (cocaine, amphetamines, cannabis).
- 4 Urine dipstick (urinary tract infection, ketonuria, glycosuria).
- 5 Chest X-ray (cardiomegaly, pulmonary oedema, pneumonia, lung cancer).

Specialist investigations

- 1 Echocardiogram (valvular heart disease, systolic/diastolic dysfunction, structural heart disease).
- 2 A 24-hour ECG (paroxysmal tachyarrhythmias, e.g. paroxysmal AF, SVTs) for symptomatic episodes that occur less than 24 hours apart, or suspected asymptomatic episodes.
- 3 Consider use of an event recorder if symptomatic episodes more than 24 hours apart and referral to cardiology [31].

MANAGEMENT

Sinus tachycardia

The management of sinus tachycardia depends entirely on the underlying cause. Indeed, since sinus tachycardia may represent a physiological response, attempts to slow the heart rate in this context may result in hypertension [33].

- If a specific cause is identified, management should focus on treatment of the underlying condition.
- In psychiatric inpatients, if agitation or panic is suspected but alternative diagnoses cannot initially be discounted (e.g. evolving infection), then increasing frequency of observations and clinical reviews may be indicated, even if only temporarily [8].
- The presence of red flag symptoms including persistent chest pain, syncope, hypotension, pyrexia, tachypnoea or hypoxia may necessitate transfer to the accident and emergency department (A&E) for further investigation, higher levels of monitoring, and acute management [28].

Clozapine-induced sinus tachycardia

Clozapine-induced sinus tachycardia is very common in the early stages of treatment but is usually benign and may be dose related. For asymptomatic patients without signs of myocarditis (e.g. fever, chest pain), clozapine-induced sinus tachycardia in the early stages of treatment can generally be managed conservatively with monitoring of clozapine levels, appropriate dose modification, and reassurance and daily observations [34]. If symptomatic, then rate control with beta-blockers such as bisoprolol (starting oral dose 1.25–2.5 mg once daily, titrate to response) may be used as first-line medical therapy, although the evidence base for beta-blockade in the context of clozapine use is limited [35], and may be associated with side effects such as fatigue, weight gain, postural hypotension and, in men, impotence. If beta-blockers are

contraindicated or not tolerated, ivabradine can be considered (5–7.5 mg, oral, twice daily) [36,37]. Seek advice from a cardiologist if first-line treatment fails, as untreated tachycardia has been associated with cardiomyopathy [38]. Evidence is currently lacking to support the treatment of asymptomatic clozapine-induced tachycardia, and therefore management should weigh the risks of rate-control medication against the potential risk of long-term tachycardia (i.e. cardiomyopathy). If pharmacological rate control is not pursued, ongoing monitoring of these patients is recommended, with consideration of annual echocardiograms to screen for cardiomyopathy, and where appropriate discussion with cardiology.

Atrial fibrillation

Guidelines published by the National Institute for Health and Care Excellence (NICE), the European Cardiac Society and the American College of Cardiology provide comprehensive algorithmic approaches to the management of AF. A brief summary is provided here, and readers are encouraged to consult the complete guidelines (accessed at <https://pathways.nice.org.uk/pathways/atrial-fibrillation>, <https://www.escardio.org/Guidelines> and <https://www.acc.org/guidelines>) [13]. General principles involve offering symptomatic patients rate control (e.g. a beta-blocker) and anticoagulation if the risk of thromboembolism is high (CHA₂DS₂VASc score ≥ 2 ; Table 1.2) but not outweighed by the risk of major bleeding (calculated using the HAS-BLED score; Table 1.3) [39,40]. Choice of anticoagulant should be made after a joint discussion of risks and benefits between doctor and patient. Options include warfarin or a direct oral

Table 1.2 CHA₂DS₂VASc score to assess risk of thromboembolic event in atrial fibrillation and need for anticoagulation [39].

| Risk criteria | Score |
|----------------------------|-------|
| Congestive heart failure | 1 |
| Hypertension | 1 |
| Female sex | 1 |
| Age: 65–74 | 1 |
| ≥ 75 | 2 |
| Diabetes | 1 |
| Stroke/TIA/thromboembolism | 2 |
| Vascular disease | 1 |

Score = 0: anticoagulation not indicated

Score = 1: consider anticoagulation in men

Score ≥ 2 : anticoagulate if it outweighs risk of bleeding

TIA, transient ischaemic attack.

Table 1.3 HAS-BLED score determines risk of major bleeding for people with atrial fibrillation^a [40].

| Risk criteria | Score |
|--|-------|
| Hypertension: uncontrolled, >160 mmHg systolic | 1 |
| Renal disease: dialysis, transplant, Cr >200 µmol/L | 1 |
| Liver disease: cirrhosis or bilirubin more than twice normal and AST/ALT/AP more than three times normal | 1 |
| Stroke history: prior major bleeding or predisposition to bleeding | 1 |
| Labile INR: unstable/high INRs, time in therapeutic range <60% | 1 |
| Age >65 years | 1 |
| Medication: aspirin, clopidogrel, NSAIDs | 1 |
| Alcohol: eight or more drinks per week | 1 |
| Score ≤0–1: low risk, consider anticoagulation | |
| Score = 2: moderate risk, consider anticoagulation | |
| Score ≥2: high risk, consider alternatives | |

^a HAS-BLED score does not relate directly to CHA₂DS₂-VASc score and cannot be compared quantitatively.

ALT, alanine aminotransferase; AP, alkaline phosphatase; AST, aspartate aminotransferase; Cr, creatinine; INR, international normalised ratio; NSAIDs, non-steroidal anti-inflammatory drugs.

anticoagulant (e.g. apixaban, dabigatran, rivaroxaban, and edoxaban). Referral to cardiology is recommended if anticoagulation is contraindicated. Anticoagulation, rate control, and symptoms should be reviewed at least annually alongside an assessment of cardiovascular risk and potential complications such as heart failure. Referral to cardiology is indicated if pharmacological or electrical cardioversion is being considered.

Supraventricular tachycardia

Persistent new-onset SVT requires *immediate transfer* to A&E due to risk of haemodynamic compromise [2]. Non-pharmacological interventions such as the Valsalva manoeuvre or carotid sinus massage can be attempted and immediate transfer to hospital can be avoided for people with known SVT who revert to sinus rhythm.

Further acute management may involve intravenous adenosine administration, synchronised cardioversion or intravenous antiarrhythmics such as diltiazem or beta-blockers [14]. Long-term management should be directed by a cardiologist. Lifestyle advice on alcohol, caffeine, and illicit drug use as potential precipitants should be offered, as well as strict control of general cardiac risk factors (e.g. smoking cessation) [2].

Broad-complex tachycardia

Any broad-complex tachycardia should be managed as VT until proven otherwise [28,29,41]. For psychiatric inpatients or people in the community, this is likely to

Box 1.4 Driving and working advice for patients with arrhythmia in the UK

In the UK, if the arrhythmia has caused or is likely to cause incapacity (e.g. syncope or VT), the patient should be advised to stop driving until:

- a satisfactory diagnosis is found
- the symptoms are controlled for at least 4 weeks (Group 1 entitlement: motor car or motorcycle) or 3 months (Group 2 entitlement: lorries, buses or HGVs) [42].

Note that guidelines differ somewhat internationally [43]. Please refer to local guidelines if not UK-based.

If the patient is working in potentially dangerous occupations (at height or with heavy machinery), they should be advised to:

- stop working until the condition is controlled
- notify their occupational health department if applicable [29].

require urgent transfer to A&E where further investigations and initial treatment can be enacted in a monitored environment, although immediate resuscitation will be required using the ABCDE approach if the patient loses cardiac output (see Chapter 70) [28,29,41]. Sinus tachycardia or AF in a patient with known bundle-branch block are the most likely exceptions to this rule. However, if in doubt, referral to emergency services is recommended given the high mortality associated with VT [28,29,41].

Advice from the UK government regarding driving and working for patients with arrhythmia is shown in Box 1.4. We advise reviewing the guidance at source as it is updated monthly (<https://www.gov.uk/guidance/cardiovascular-disorders-assessing-fitness-to-drive>).

When to refer to a specialist

Urgent transfer to A&E is indicated for the following conditions [28,29,41].

- Any tachycardia with ‘red flag’ features:
 - haemodynamic instability
 - significant breathlessness
 - chest pain
 - syncope or near syncope
 - family history of sudden cardiac death under 40
 - symptoms precipitated by exercise.
- Suspected VT.
- Persistent SVT.
- AF with evidence of serious complication (e.g. stroke or heart failure).
- Evidence of concurrent illness that necessitates admission (e.g. sepsis).

What information to include in a referral to a specialist

Referral to a specialist should include any positive findings and salient negative findings from the history, examination and investigations (summarized in Box 1.5). Where possible, include all relevant ECGs. In emergency situations, it may be possible to send scanned ECGs via secure email. Also include contact details of the patient's mental healthcare team/support network, and if the appointment should be sent to anyone in addition to the patient. Finally, provide details of any reasonable adjustments needed for the patient (e.g. time or duration of appointment, carer/support worker in attendance).

Box 1.5 Diagnostic summary for the tachycardic patient

History

- Define cardiac symptoms
- Determine possible precipitants
- Assessment of palpitations (if paroxysmal)
- Screen for symptoms of systemic illness
- Past medical and psychiatric history: any cardiac disease, hypertension, diabetes, thyroid disease, chronic lung disease, anxiety, panic
- Medication history
- Drug and alcohol history
- Family history of sudden cardiac death

Examination

- ABCDE assessment
- Cardiovascular examination
- Focused examination if systemic illness suspected

Investigations

- ECG
- Bloods
- Urine dipstick
- Consider echocardiogram or 24-hour tape

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