# Supraventricular tachycardia

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## ABSTRACT

- Supraventricular tachycardia (SVT) is a common cardiac rhythm disturbance; it usually presents with recurrent episodes of tachycardia, which often increase in frequency and severity with time.
- Although SVT is usually not life-threatening, many patients suffer recurrent symptoms that have a major impact on their quality of life. The uncertain and sporadic nature of episodes of tachycardia can cause considerable anxiety — many patients curtail their lifestyle as a result, and many prefer curative treatment.
- SVT often terminates before presentation, and episodes may be erroneously attributed to anxiety.
- Sudden-onset, rapid, regular palpitations characterise SVT and, in most patients, a diagnosis can be made with a high degree of certainty from patient history alone. Repeated attempts at electrocardiographic documentation of the arrhythmia may be unnecessary.
- Treatment of SVT may not be necessary when the episodes are infrequent and self-terminating, and produce minimal symptoms.
- When episodes of tachycardia occur frequently, are prolonged or are associated with symptoms that affect quality of life, catheter ablation is the first choice of treatment; it is a low-risk procedure with a high success rate. Long-term preventive pharmacotherapy is an alternative approach in some patients.


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Supraventricular tachycardia (SVT) refers to a range of conditions in which atrial tissue or the atrioventricular node is essential for sustaining an arrhythmia. This review focuses on the three most common types of SVT — atrioventricular nodal re-entrant tachycardia (AVNRT), atrioventricular re-entrant tachycardia (AVRT) and atrial tachycardia (AT); it also describes inappropriate sinus tachycardia and postural orthostatic tachycardia syndrome, forms of sinus tachycardia (Box 1). Other types of SVT include atrial fibrillation and atrial flutter, which were the focus of a recent clinical update in the Journal.[1]

The incidence of SVT is about 35 cases per 100 000 population per year, with a prevalence of 2.25 cases per 1000 population.[2] SVT usually manifests as recurrent paroxysms of tachycardia. It is generally well tolerated but can produce uncomfortable symptoms that lead to acute presentation.

## Clinical features

Younger patients with SVT usually have structurally normal hearts, and are more than twice as likely to be female as male.[3] Most females with SVT present during their childbearing years (15–50 years),[4] and this has been linked to the effect of progesterone on the myocardium.[5] Women with SVT are more likely to have AVNRT than men,[4] whereas there is a male predominance in AVRT.[6]

For the three most common types of SVT, peak incidence of presentation for ablation occurs in the middle decades of life: at 36 years for AVRT, 48 years for AVNRT, and 50 years for AT.[7] The proportion of SVT caused by AVRT declines progressively with age, from 60% during the first decade of life to 9% after age 70. Correspondingly, there are steady increases in the proportions of AVNRT (from 33% to 68%) and AT (from 7% to 23%). In one study, AVNRT replaced AVRT as the dominant paroxysmal SVT mechanism at age 40 in males and at age 10 in females.[7] A significant proportion of patients have symptoms for a prolonged period (> 1 year) before the diagnosis of SVT is made, and occasionally episodes are misdiagnosed as anxiety or panic disorders.[8] These misdiagnoses occur more frequently in women.

## Symptoms

Palpitations and pounding in the neck or head are the most common symptoms of SVT, and may be accompanied by chest discomfort (chest pain is unusual), dyspnoea, anxiety, lightheadedness or, uncommonly, syncope. Syncope may occur at onset, before autonomic reflexes respond to blood pressure fall, particularly when heart rate is very rapid and occasionally during very prolonged episodes. It may also occur in response to rapidly conducted atrial fibrillation via an accessory pathway, or when SVT occurs in the presence of significant structural heart disease.

The severity of symptoms is highly variable and depends on features including heart rate, duration of tachycardia, underlying heart disease, and individual patient perception. Incessant SVT can result in tachycardia-mediated cardiomyopathy.[9] This left ventricular dysfunction is usually completely reversible on control or cure of the arrhythmia.[10] SVT occasionally results in myocardial ischaemia, or precipitates cardiac failure in patients with pre-existing coronary disease or myocardial dysfunction. The symptoms of SVT can be very similar to those of anxiety, and both may co-exist. Very rapid heart rate (around 180–200 beats/min) and termination of palpitations with the Valsalva manoeuvre are consistent with the diagnosis of SVT.

## Evaluating the patient with SVT

### History

Classical SVT history is characterised by an abrupt onset of rapid palpitations. This strongly suggests SVT, and diagnosis can usually be made without electrocardiographic documentation. Gradual onset of palpitations suggests sinus tachycardia,[11] and irregular palpitations often indicate atrial fibrillation.

Defining the frequency and duration of palpitations and associated symptoms enables an assessment of clinical severity. Episodes of SVT may be triggered by factors including caffeine and alcohol intake (which can increase the frequency with which ectopic beats are triggered), bending over, sudden movements, stress, physical exertion and fatigue. Patients will have a clear idea of whether any of these are common triggers in their own case. When triggers are present they should be avoided if possible, but there is no a priori reason to restrict caffeine or alcohol intake or limit exercise in patients for whom these are not triggers.

**1 Types of supraventricular tachycardia and their features**

Atrioventricular nodal re-entrant tachycardia
- Most common form
- Re-entrant circuit involves the atrioventricular node
- Retrograde P waves may be seen buried within or just after the QRS complex in tachycardia

Atrioventricular re-entrant tachycardia
- Second most common form
- Re-entrant circuit involves an accessory pathway
- Some pathways, termed "concealed" pathways, only conduct in a retrograde direction
- Pathways that conduct in an antegrade direction show pre-excitation on a surface electrocardiogram (Wolff–Parkinson–White syndrome)

Atrial tachycardia
- Third most common form
- Tachycardia arises from a localised focus of atrial tissue
- Foci arise from characteristic locations in the atrium
- P-wave morphology can be used to identify the site of tachycardia origin

Sinus tachycardias

*Physiological sinus tachycardia*
- Appropriate response to a physiological or pathological stress

*Inappropriate sinus tachycardia*
- Typically seen in women — usually in health care workers
- Persistent elevation of the sinus rate during the day, which normalises during sleep

*Postural orthostatic tachycardia syndrome*
- Inappropriate sinus tachycardia associated with upright posture
- Other autonomic symptoms may coexist

Rare forms of supraventricular tachycardia

*Permanent junctional reciprocating tachycardia*
- Typically seen in children
- Associated with tachycardia-mediated cardiomyopathy

*Junctional ectopic tachycardia*
- Occurs in children
- Tachycardia arises from a discrete focus in the atrioventricular node

*Mahaim tachycardia*
- Tachycardia due to an abnormal accessory pathway between the atrioventricular node and His–Purkinje system
- Pathway usually inserts from the right atrium into the right ventricle near the right bundle branch ◆

### Examination

Results of cardiovascular examination are usually normal for patients with SVT, but signs of structural heart disease should be sought.

### Electrocardiogram

In many cases, results of a baseline electrocardiogram (ECG) in patients with SVT are normal. However, the results should be carefully evaluated for evidence of pre-excitation, defined by a short PR interval (< 120 ms) and a delta wave (slurred upstroke at the onset of the QRS complex) (Box 2). This is the classical ECG appearance of Wolff–Parkinson–White (WPW) syndrome.[12]

If the arrhythmia is captured on an ECG it is usually a narrow-complex tachycardia (QRS duration, < 120 ms) (Box 3), but may have a prolonged QRS interval (> 120 ms) when associated with pre-existing or rate-related bundle branch block. In wide-complex tachycardia, however, it is safest to assume that the tachycardia is ventricular in origin until proven otherwise. A normal Holter monitor reading is seen in most patients with SVT because of the intermittent nature of episodes; thus a normal reading does not exclude the diagnosis of SVT. Often, prolonged and multiple unnecessary attempts at rhythm documentation are made when the diagnosis is evident from clinical history. Occasionally, in patients with infrequent palpitations and a less definite clinical history, cardiac event recorders or implantable monitors may be necessary to capture the underlying rhythm disturbance.

### Echocardiogram

An echocardiogram can be used to evaluate cardiac structure and function, but results are usually normal for patients with SVT.

### Exercise testing

Exercise testing is less useful for diagnosis of SVT unless the arrhythmia is typically triggered by exertion. Patients may complain of chest discomfort or pain during SVT episodes. These symptoms do not mandate an exercise stress test or angiography; decisions on further testing should be based on history and presence of vascular risk factors.

## Mechanisms of SVT

### Atrioventricular nodal re-entrant tachycardia

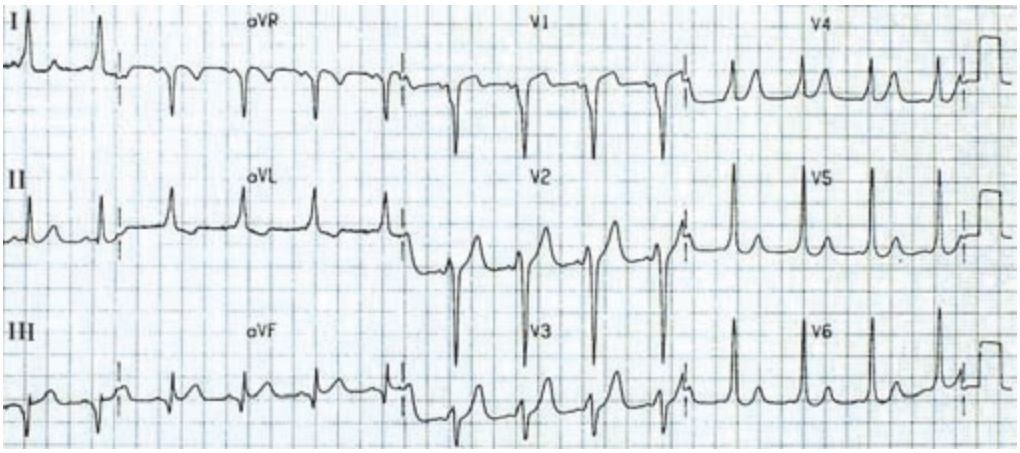
The most common type of SVT is AVNRT.[13] The mechanism involves a re-entrant circuit that includes the posterior inputs to the compact atrioventricular node, anterior inputs to the node, and probably perinodal atrial tissue. The tachycardia is often triggered by an appropriately timed atrial ectopic beat (Box 4).

### Atrioventricular re-entrant tachycardia

AVRT is the second most common type of SVT, and uses an accessory pathway to complete the re-entrant circuit. Accessory pathways are muscular connections composed of functional myocardial fibres that directly connect the atria and ventricles, bypassing the atrioventricular node. Many accessory pathways do not produce pre-excitation on the ECG during sinus rhythm, owing to an inability to conduct in an antegrade direction. When the pathway conducts from ventricle to atrium (retrograde conduction), with no evidence of antegrade conduction on the sinus rhythm ECG, the pathway is termed "concealed". In this situation, the tachycardia circuit involves antegrade conduction over the atrioventricular node and retrograde conduction over the accessory pathway.

When the accessory pathway also conducts in the antegrade direction during sinus rhythm, the ventricular myocardium is activated earlier than if conduction occurred only through the atrioventricular node, resulting in ventricular pre-excitation (WPW syndrome, Box 2).[12] In patients with WPW syndrome, episodes of SVT can trigger atrial fibrillation leading to rapid conduction of the atrial activity to the ventricle via the accessory pathway. Unlike the atrioventricular node, which acts as a filter between the atria and ventricles, an accessory pathway can transmit atrial rates of up to

**2 Electrocardiogram showing pre-excitation**




*The PR interval is short (< 120 ms) and there is a slurred onset of the QRS complex, with a widened QRS morphology. The appearance and degree of pre-excitation that is evident depends on the conduction of the pathway and atrioventicular nodal conduction.* ◆

300 beats/min directly to the ventricles (Box 5). This can lead to ventricular fibrillation and sudden death.[14]

## Atrial tachycardia

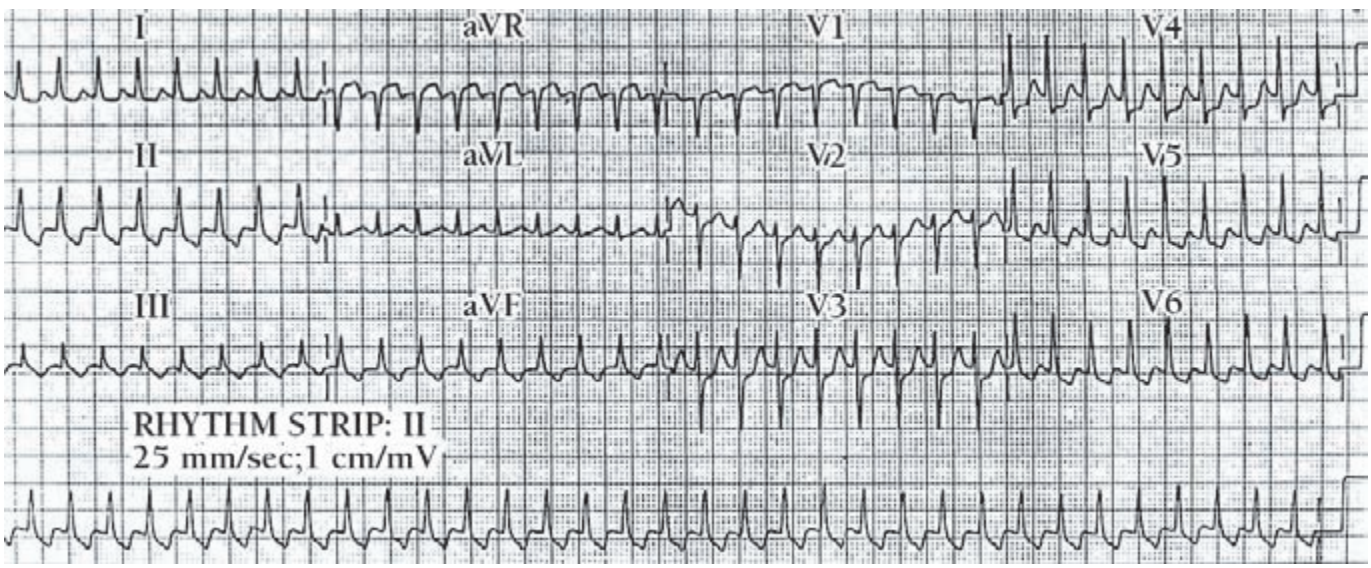
***Focal atrial tachycardia:*** This accounts for about 10% of cases of SVT, and originates from a single localised focus of atrial tissue.[15-17] The atrial rate can vary widely, from 120 beats/min to 300 beats/min. Depending on the atrial rate, and on atrioventricular node conduction, the atria may conduct 1:1 to the ventricles, or with varying degrees of atrioventricular block. Focal atrial tachycardia has characteristic anatomical sites of origin. The most common site in the right atrium is along the crista terminalis, and in the left atrium common sites are the ostia of the pulmonary veins.[16,18]

***Multifocal atrial tachycardia:*** This is characterised by electrocardiographic evidence of at least three different P-wave morphologies. It usually occurs in older patients with chronic lung disease or congestive cardiac failure, and may ultimately disorganise into atrial fibrillation.[19]

## Sinus tachycardias

***Inappropriate sinus tachycardia:*** This is an unusual clinical syndrome; it is characterised by a persistently elevated resting heart rate (> 100 beats/min) that is disproportionate to the degree of physiological and/or pathological stress. It is important to eliminate secondary causes of sinus tachycardia (eg, thyrotoxicosis, anaemia) before the diagnosis is made. Enhanced automaticity of the sinus node, excess sympathetic tone and reduced parasympathetic tone are the principal proposed mechanisms.[20] Most patients who are affected by inappropriate sinus tachycardia are women, and it is particularly common in health care workers — possibly because they are more likely to self-recognise tachycardia than the general population.[21] The condition is poorly understood and, after secondary causes have been excluded, patients may be misdiagnosed as having anxiety or a panic disorder. Monitoring electrocardiographic function over a 24-hour period using a Holter monitor is the most useful means of identifying inappropriate sinus tachycardia; classically, it reveals a persistently elevated sinus rate (> 100 beats/min) during the day and normalisation of the heart rate during sleep.[22]

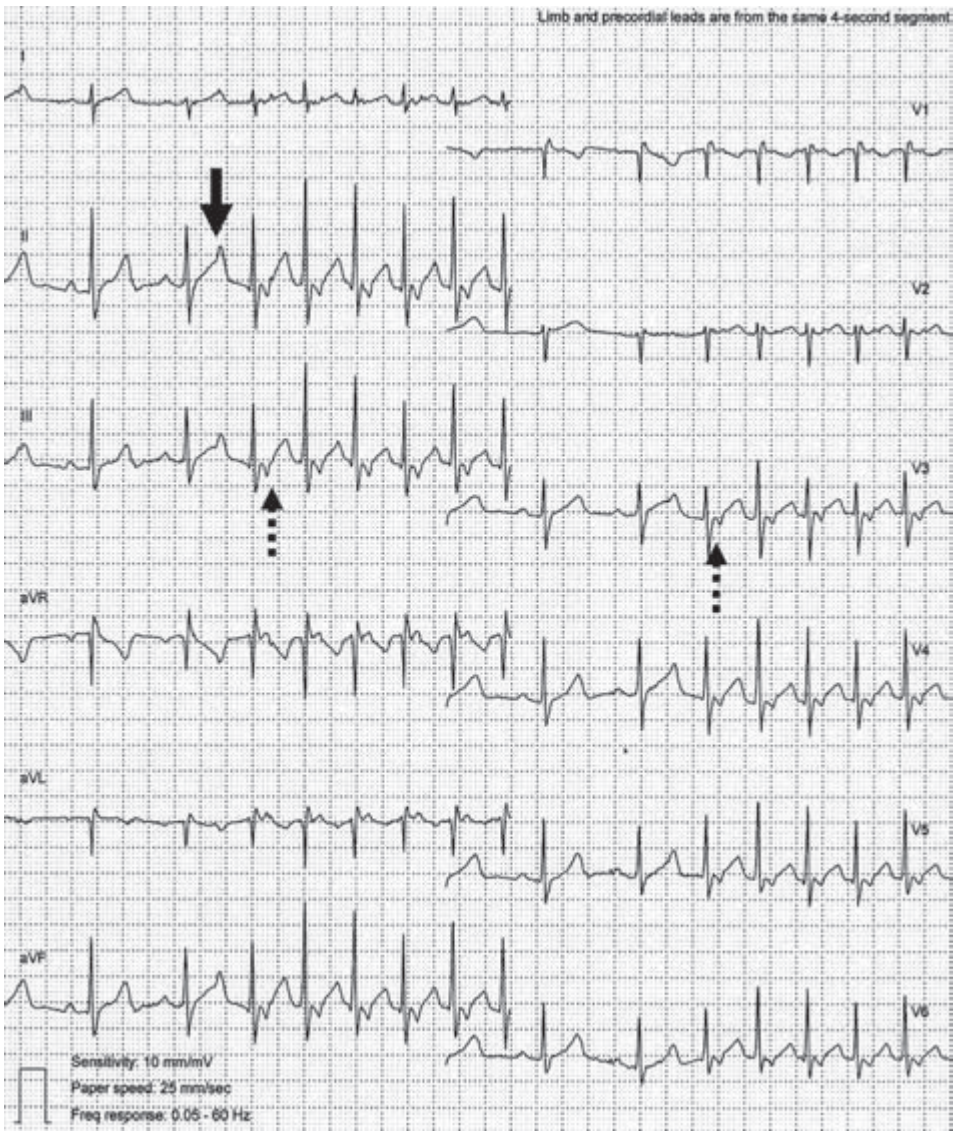
**3 Twelve-lead electrocardiogram of a narrow-complex tachycardia**




*The lack of visible P waves suggests that this tachycardia is due to atrioventricular nodal re-entrant tachycardia, or atrioventricular re-entrant tachycardia with a concealed pathway.* ◆

***Postural orthostatic tachycardia syndrome:*** In this syndrome an inappropriate sinus tachycardia is associated with upright posture, in the absence of postural hypotension or autonomic neuropathy.[23] Symptoms overlap with those of inappropriate sinus tachycardia, and additional autonomic symptoms can occur — tremor, constipation, bladder dysfunction, feeling cold, heat intolerance, marked fatigue and exercise intolerance.[24] The symptoms and the accompanying sinus tachycardia can be reproduced by tilt-table testing.[22]

**4 Holter monitor recording showing the initiation of supraventricular tachycardia triggered by an atrial ectopic beat**




*The P wave of the atrial ectopic beat is visible as a distortion of the T wave of the preceding beat (solid arrow). Retrograde P waves are visible immediately after the QRS complex (dotted arrows). This tachycardia may be due to atrioventricular re-entrant tachycardia with a concealed pathway, or atrioventricular node re-entry. This patient did not elect to undergo an electrophysiology study and ablation therapy, and is not on maintenance medical therapy.* ◆

## Management of SVT

### Short-term management

The goal of short-term management is to terminate acute episodes of tachycardia, which can often be achieved by manoeuvres that increase vagal tone, including the Valsalva manoeuvre, application of a cold stimulus to the face and carotid sinus massage. Carotid sinus massage can also provide diagnostic information by slowing atrioventricular nodal conduction and exposing the P wave; it is performed by applying gentle pressure over one carotid sinus for 5–10 seconds during held inspiration. This manoeuvre should not be performed if there is a history of carotid artery disease or if carotid bruits are detected on examination.

If vagal stimulation is unsuccessful, recommended drugs include adenosine, and calcium antagonists such as verapamil or diltiazem.[25] Adenosine is advantageous as its onset is instantaneous and it has an extremely brief duration of action. However, in rare cases it can aggravate bronchospasm, cause atypical chest discomfort or cause a sensation of impending doom. Administered by intravenous injection, a 6 mg dose of adenosine is successful in reverting SVT in 75% of patients, and a 12 mg dose is successful in more than 90% of patients.[26] If adenosine therapy is unsuccessful, intravenous boluses of either verapamil or diltiazem usually terminate tachycardia,[27,28] but carry the risk of potentiating hypotension and bradycardia. Intravenous verapamil is more readily available in most clinical settings than intravenous diltiazem. In adults, 5–10 mg of verapamil administered by intravenous injection over 2–3 minutes is often successful in reverting SVT. Patients given verapamil must be monitored due to the risk of bradycardia. SVT resulting in haemodynamic instability is rare but necessitates urgent direct-current cardioversion.

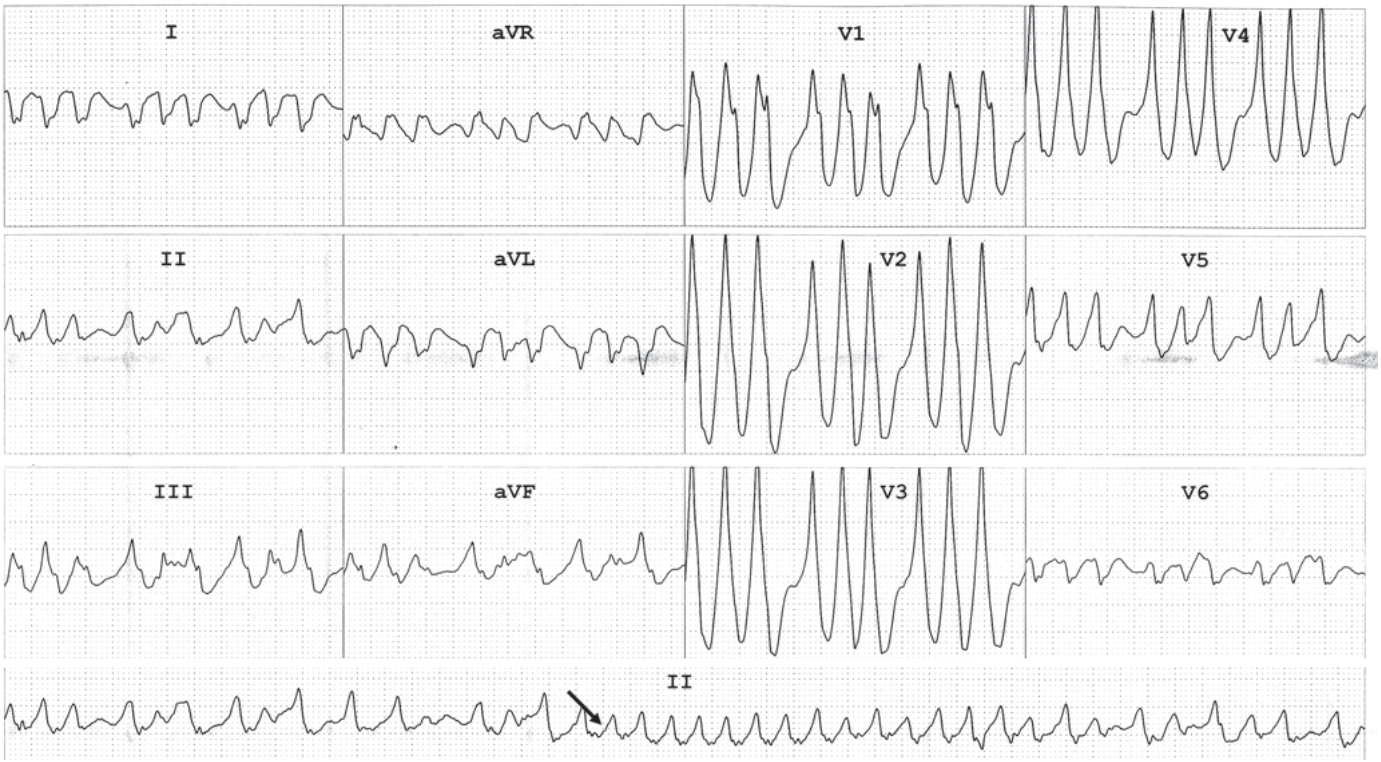
### Long-term management

Long-term management is individualised based on the frequency and severity of episodes and the impact of symptoms on quality of life.[29] For infrequent, self-terminating and minimally symptomatic episodes, treatment is not necessarily required; however, many patients will opt for a curative approach owing to the anxiety associated with possible recurrence of symptomatic episodes.

Definitive treatment of SVT is indicated in patients who:

- have recurrent symptomatic episodes of SVT that affect their quality of life;
- experience symptoms of SVT, and have WPW syndrome detected on ECG; and

**5 Twelve-lead electrocardiogram showing pre-excited atrial fibrillation**




*The accessory pathway is capable of very rapid conduction, resulting in a ventricular rate that is greater than if conduction occurred via the atrioventricular node. At times, the ventricular rate approximates 300 beats/min (arrow). Catheter ablation is mandatory in this situation.* ◆

- have infrequent episodes of SVT but are engaged in a profession or sport in which an episode of SVT could put them or others at risk (eg, pilots and divers).

Radiofrequency catheter ablation is recommended for most of these patients. It has a low risk of complications, and is curative in more than 95% of patients.[30] The procedure typically takes 1–1.5 hours; it can be performed under local anaesthesia with sedation, or under general anaesthesia. Patients usually stay in hospital overnight after the procedure for cardiac monitoring and observation.

### *Pharmacological management*

Long-term pharmacotherapy is generally used in patients who decline catheter ablation, and in whom the procedure carries an unacceptably high risk of atrioventricular node injury and pacemaker dependence. The goal of long-term pharmacotherapy is to reduce the frequency of episodes of SVT. In only a small minority of patients will episodes be completely abolished by antiarrhythmic drugs. Recommended drugs include atrioventricular nodal blocking drugs and antiarrhythmic drugs of Class Ic and Class III. Beta blockers and calcium-channel blockers (Class II and IV) are suitable first-line treatments when WPW syndrome is not detected on a surface ECG. Randomised studies have not demonstrated clinical superiority of any single agent, but beta blockers and calcium-channel blockers are perceived to be superior to digoxin as they provide better atrioventricular nodal blocking action during states of high sympathetic tone, such as exercise.[31] Digoxin should not be used in patients with WPW syndrome, as it may facilitate rapid conduction over the accessory pathway during atrial fibrillation — potentially leading to ventricular fibrillation.[32] Combining atrioventricular nodal blocking agents increases efficacy, but also increases adverse effects.[33]

For patients who do not respond to these drugs, or for those with WPW syndrome, alternative drugs include flecainide (Class Ic actions) and sotalol (Class II and Class III actions). Flecainide and sotalol are more effective than atrioventricular nodal blockers in terms of preventing SVT, but are associated with a small a risk of ventricular tachycardia. This risk is small in patients without structural heart disease, but it is has been reported to occur in 1%–3% of patients taking sotalol, particularly those taking higher doses.[34,35] Amiodarone has no role in long-term prevention of SVT, owing to the high incidence of serious toxicities associated with its long-term use.[36]

Beta blockers are first-line therapy for the management of inappropriate sinus tachycardia; the dose should be titrated to balance symptom control with prevention of hypotension and bradycardia.[22] Verapamil and diltiazem are alternatives for patients in whom beta blockers are contraindicated. A new agent, ivabradine, acts by blocking the sodium current responsible for spontaneous depolarisation in the sinus node ($I_f$), which results in sinus bradycardia.[37] Ivabradine has no negative inotropic effects but may produce visual disturbance that is reversible on discontinuation of the drug. It is licensed for treating angina and, although there is relatively little published data on its efficacy, it may be trialled off-label in patients with inappropriate sinus tachycardia who do not respond to beta blockers and calcium-channel blockers. In patients with postural orthostatic tachycardia syndrome, increased fluid and salt intake, resistance exercises, squatting and compressive stockings may be effective.[38] When non-pharmacological strategies are ineffective, beta blockers and/or fludrocortisone may be beneficial.[22]

## Competing interests

None identified.

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