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Understanding the modern-era management of cardiac tachyarrhythmias

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Abstract

Diagnosis and treatment modalities of supraventricular arrhythmias have been changed over the past decade. This article mainly outlines advances in diagnosis and modern-day management of supraventricular tachycardia as well as an overview of the management of wide complex tachyarrhythmias. The role of cardiac electrophysiology study and radiofrequency catheter ablation in the diagnosis and management of a wide variety of supraventricular arrhythmias is also discussed. This article also emphasizes the current evidence-based management of atrial-origin tachyarrhythmias such as atrial flutter and fibrillation.

The management of cardiac arrhythmias has changed over the past decade, which is mainly due to improvements in catheter ablation including 3D mapping catheter ablation, improvements in device therapy such as implantable cardioverter defibrillator (ICD) and biventricular pacing therapy (CRT) which have evolved to treat many conditions. Catheter ablation has moved from being a treatment considered only for patients with refractory arrhythmias to a first-line treatment. In the new era, catheter ablation is also used to treat atrial origin tachyarrhythmias such as atrial fibrillation (AF) and atrial flutter as well as ventricular tachycardia (epicardial VT ablation). Improvement of imaging modalities such as 2D/3D echocardiogram and cardiac MRI also play a vital role in the prognosis of ablation particularly in AF and epicardial VT ablation. Improvement in device implantation such as ICD and biventricular pacing also helpful in managing tachyarrhythmia. Modern advancements in cardiac electrophysiology help patients to improve their quality of life and prevent sudden cardiac deaths due to cardiac arrhythmia.

Key words: cardiac, tachyarrhythmia, ablation, EP study

Introduction

Supraventricular arrhythmias (SVT) are common arrhythmias. With these arrhythmias, patients are often symptomatic and require management with drugs and electrophysiological procedures. There are several coexistent conditions associated with SVTs. To understand the mechanisms of SVTs, we might need to have adequate knowledge regarding the cardiac conduction system and its anatomy. In general management of SVT is based on the principles of evidence-based medicine. Even though there are many varieties of SVTs, we will discuss the most common SVTs that we encounter in our day-to-day clinical practice and their management.

Mechanisms of tachycardia

Three main mechanisms have been identified.

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1. Re-entry

This is the most common tachycardias (70%). Reentry contains two distinct electrical conduction pathways or tissues with different electrophysiologic properties that are linked proximally and distally, which form a close reentrant circuit. One pathway is generally inactive whilst one is in sinus rhythm. Tachycardia initiates when this closed pathway opens due to early extra beats and changing refractory periods. The reentrant circuit may become repetitively activated, producing a sustained reentrant tachycardia (e.g., atrioventricular re-entry tachycardia [AVRT], atrioventricular nodal re-entry tachycardia [AVNRT]).

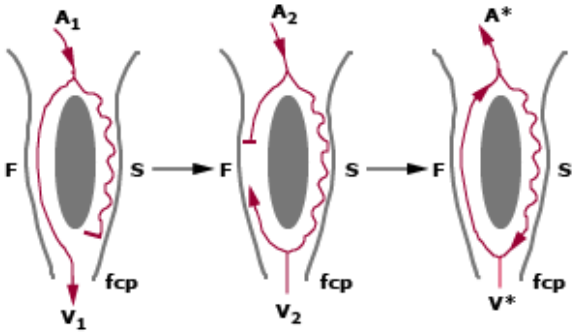

Figure 1. Electrical conducting pathways in reentry tachycardia.

A normal sinus beat (A_1) is conducted through the fast pathway (F) to the final common pathway (fcp) in the AV node to bundle of HIS (Figure 1). The conduction through the slow pathway (S) runs into the refractory period of the impulse through the fast pathway. A critically timed atrial premature beat (A_2) finds the fast pathway refractory in the antegrade direction but can conduct antegrade through the slow pathway, which has a shorter refractory period. If excitability in the fast pathway has recovered by the time the impulse reaches the fcp, there may be retrograde activation of the fast pathway. The retrograde impulse throws off an echo to the atrium (A*), and, if the slow pathway has recovered its excitability, the impulse reenters the slow pathway and produces ventricular depolarization (V*) and creates a sustained reentrant tachycardia.

2. Increased automaticity

It is mainly due to increased normal automaticity of the sinus node and abnormal automaticity resulting in an ectopic atrial or junctional tachycardia.

3. Triggered activity

Rapid depolarization during the re-polarization phase can generate early (EAD) or delayed after depolarization (DAD). This can generate tachycardia. (e.g., atrial tachycardia with or without AV block due to digitalis intoxication or arrhythmias in the setting of acute ischemia or infarction).

Clinical manifestations

Usually, the symptoms are sudden onset. Most commonly, patients present with palpitations, which are associated with diaphoresis, lightheadedness, or dizziness.

Patients with supraventricular tachycardia also present with shortness of breath or chest discomfort. Particularly those with underlying cardiac comorbidities (e.g., coronary heart disease, cardiomyopathy, or valvular heart disease with or without HF) and higher heart rates (>150 bpm). Syncope is a rare presentation, however, it may occur with a very rapid ventricular rate (>250 bpm, as might be seen in persons with AF or atrial flutter and an accessory pathway) or when SVTs degenerate into ventricular tachycardia or fibrillation (pre-excited AF may degenerate into VF in patients with accessory pathway).

The physical examination in a patient supra ventricular tachycardia reveals a rapid pulse which may be regular or irregular depending on the underlying cardiac rhythm. Distended Jugular veins are seen in AVNRT due to Atrio-ventricular dissociation. While other physical examination findings may be present in situations where the tachycardia has led to or exacerbated another condition (e.g., hypotension following syncope, lung congestion in a patient with HF).

Types of supraventricular tachycardia

a. Atrial tachycardias (AT)

1. Sinus tachycardia
   - Physiological sinus tachycardia
   - Inappropriate sinus tachycardia
   - Sinus nodal re-entrant tachycardia
2. Focal AT
3. Multifocal AT
4. Macro reentrant atrial tachycardia (MRAT)
   - Cavotricuspid isthmus-dependent MRAT
     - Typical atrial flutter, counter-clockwise (common) or clockwise (reverse)
     - Other cavotricuspid isthmus-dependent MRAT

- Non-cavotricuspid isthmus-dependent MRAT
  - Right atrial MRAT
  - Left atrial MRAT

5. Atrial fibrillation

b. AV junctional tachycardias

1. Atrioventricular nodal re-entrant tachycardia (AVNRT)
   - Typical
   - Atypical
2. Non-re-entrant junctional tachycardia
   - JET (junctional ectopic or focal junctional tachycardia)
   - Other non-re-entrant variants

c. Atrioventricular re-entrant tachycardia (AVRT)

- Orthodromic
- Antidromic

Management of supraventricular tachycardia

Initial diagnostic approach

In a patient with tachyarrhythmia, a quick clinical assessment (including short history and examination) should be done, and a 12-lead electrocardiogram should be performed. If the patient is hemodynamically unstable, at least a rhythm strip should be done prior to urgent cardioversion.[1]

If the patient is clinically (or haemodynamically) unstable (Figure 2).

Features suggestive of hemodynamic instability are hypotension, shortness of breath, chest pain suggestive of coronary ischemia, shock, and/or decreased level of consciousness.

- As quickly as possible determine whether the rhythm is sinus tachycardia.
- If the rhythm is not sinus tachycardia, or if there is any doubt that the rhythm is sinus tachycardia, urgent cardioversion is recommended.[1,2]

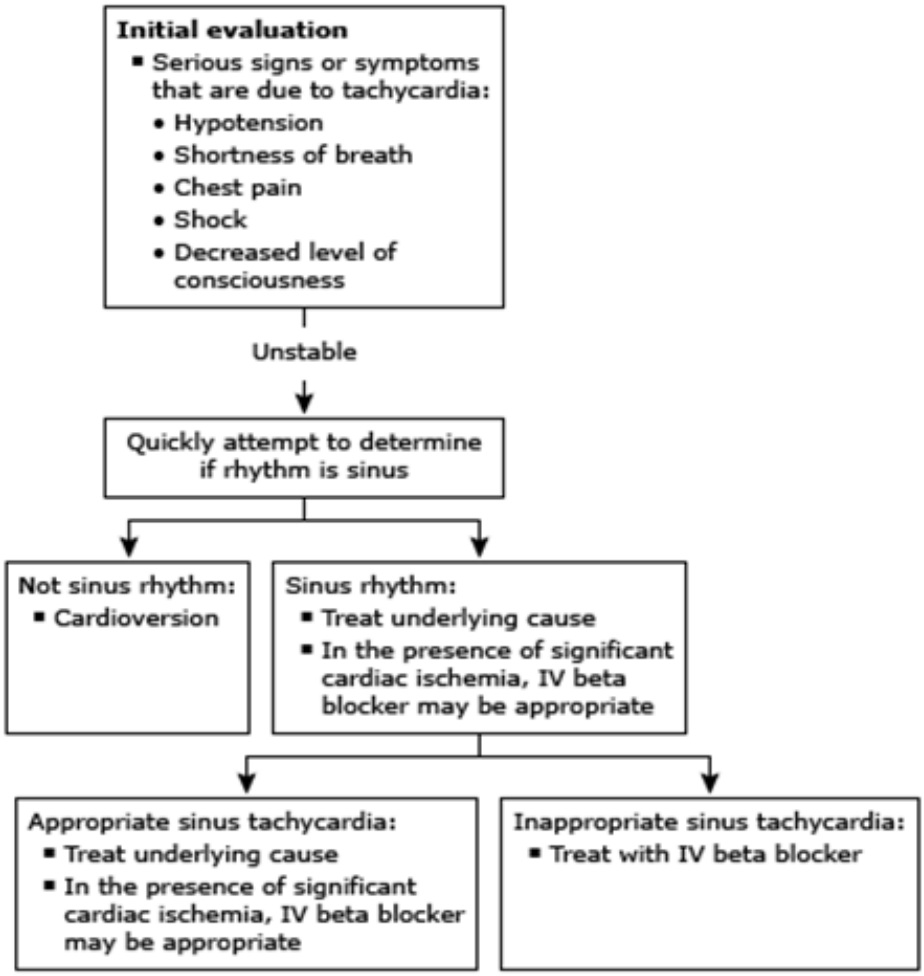

Figure 2. Management of haemodynamically unstable patient.

If the patient is clinically (or haemodynamically) stable (Figure 3).

Need to look for whether the QRS complex is narrow or wide and regular or irregular. The patient should be managed according to ECG findings.

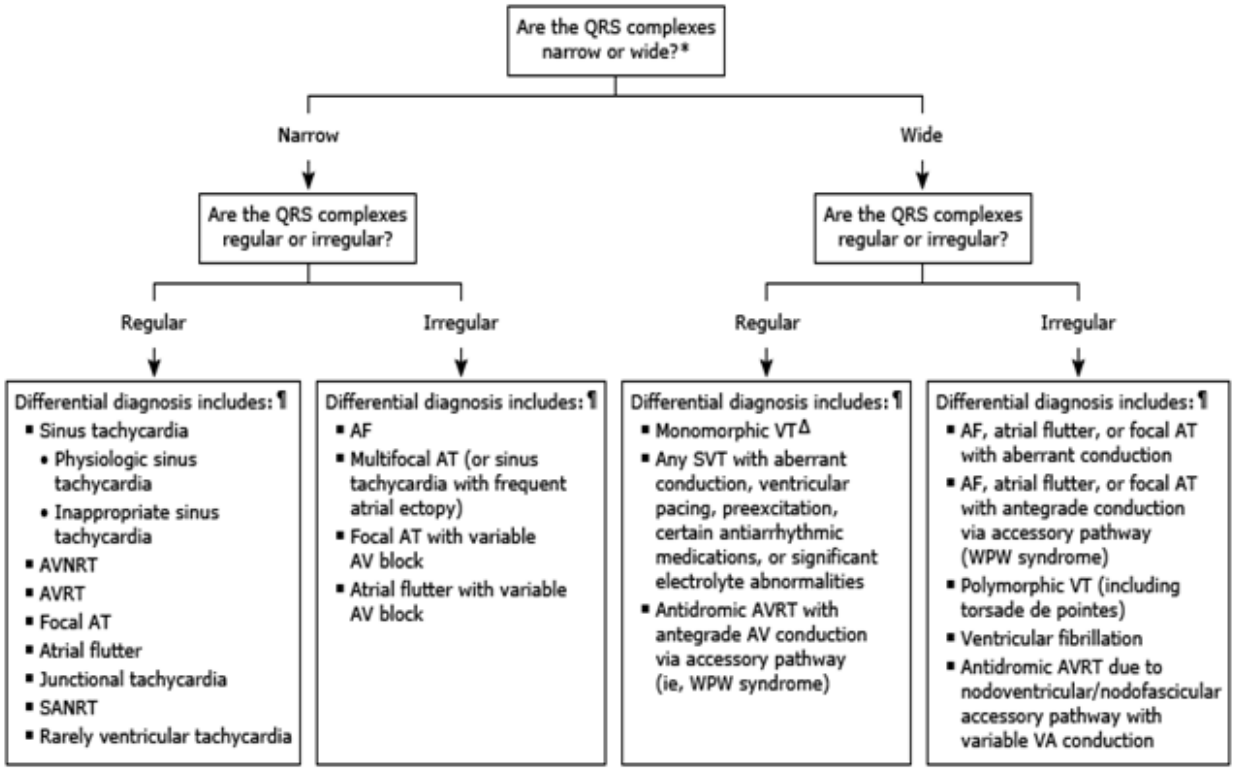

Figure 3. Management of haemodynamically stable patient.

Assessment and management of narrow complex tachycardia

1. Atrioventricular nodal reentrant tachycardia (AVNRT)

This is the most common (50%) of regular SVT. The onset of AVNRT occurs bimodally, and in many patients, attacks manifest in early life, whereas in a substantial proportion of patients, AVNRT starts after 40 years of age. More than 50% of the patients present with minimal symptoms, and short-lasting, infrequent episodes. However, a proportion of patients present with hemodynamic compromise or severe symptoms due to tachycardia and warrant aggressive treatment. AVNRT may also present with atrial fibrillation in a certain proportion of patients.[3] In patients with AVNRT, tachycardia rates are generally between 160-200 bpm. ECG typically shows absent P wave tachycardia (Figure 4). However, more commonly short RP tachycardia is seen. AVNRT is further divided into typical and atypical AVNRT[1,2], even though 12 lead ECG gives basic clues regarding AVNRT, most of the patients need a cardiac electrophysiology (EP) study. Usually, it's a 3-catheter study, where catheters are placed in coronary sinus, bundle of his and right ventricle. EP study gives a definitive diagnosis of AVNRT which also helps to map the slow pathway and radiofrequency catheter ablation of slow pathway of AVNRT. The success rate of RF ablation of AVNRT is more than 95% and recurrence is very rare after successful RF ablation.[1]

2. Accessory pathway-related tachycardia

It is the second most common cause of regular SVT and is predominantly seen in older children and young adults. Accessory pathways (AP) are single or multiple strands of myocardial cells that bypass the physiological conduction system and directly connect atrial and ventricular myocardium. It is due to incomplete embryological development of the AV annuli, without complete separation of the atria and ventricles. The most common APs connect the atrium and the

ventricle along the mitral or tricuspid annulus. APs present characteristic electrophysiological features that differ from AV nodal conduction properties. They typically exhibit fast conduction and a longer refractory period than the AV node and act as fast pathways (except atypical accessory pathways such as the Mahim pathway). Although most of the APs conduct both antegradely and retrogradely, some propagate impulses in only one direction.

When the AP conducts antegradely, ventricular pre-excitation (short PR interval and delta wave and wide QRS complex) is usually evident at rest during sinus rhythm which is called Wolf-Parkinson-White syndrome (WPW) (Figure 5). When AP is conducted only in a retrograde manner, ventricular pre-excitation would not be present in sinus ECG. During tachycardia, ECG shows one of three types.[1,2]

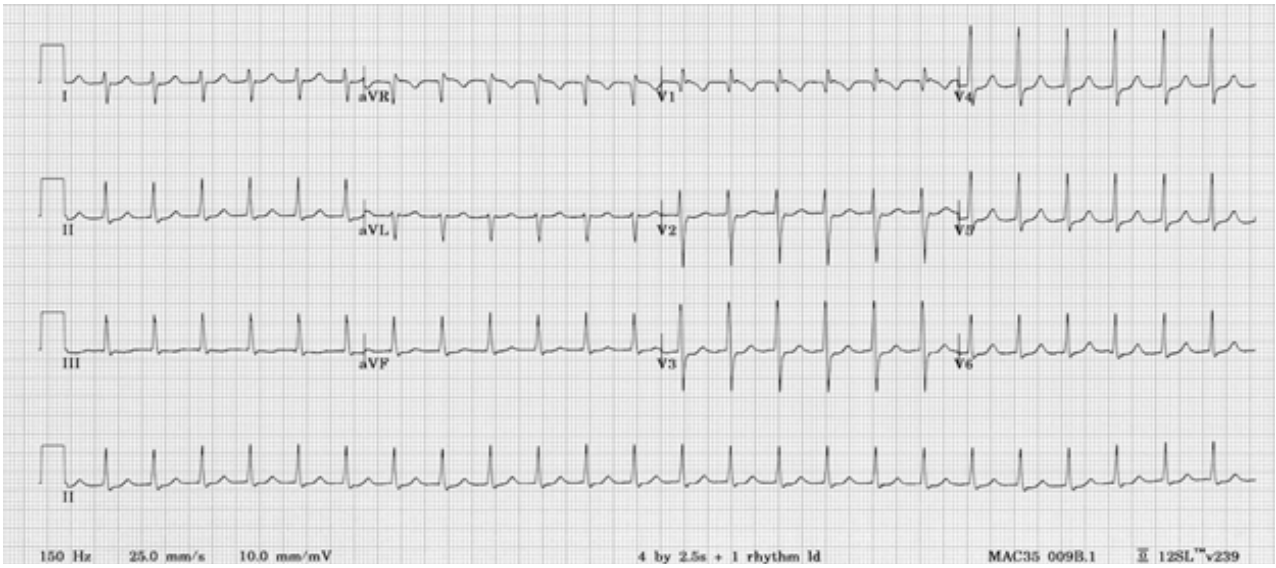

Figure 4. ECG of typical AVNRT, P waves are often hidden – being embedded in the QRS complexes. Pseudo r' wave may be seen in V1, and Pseudo S waves may be seen in leads II, III, or aVF.

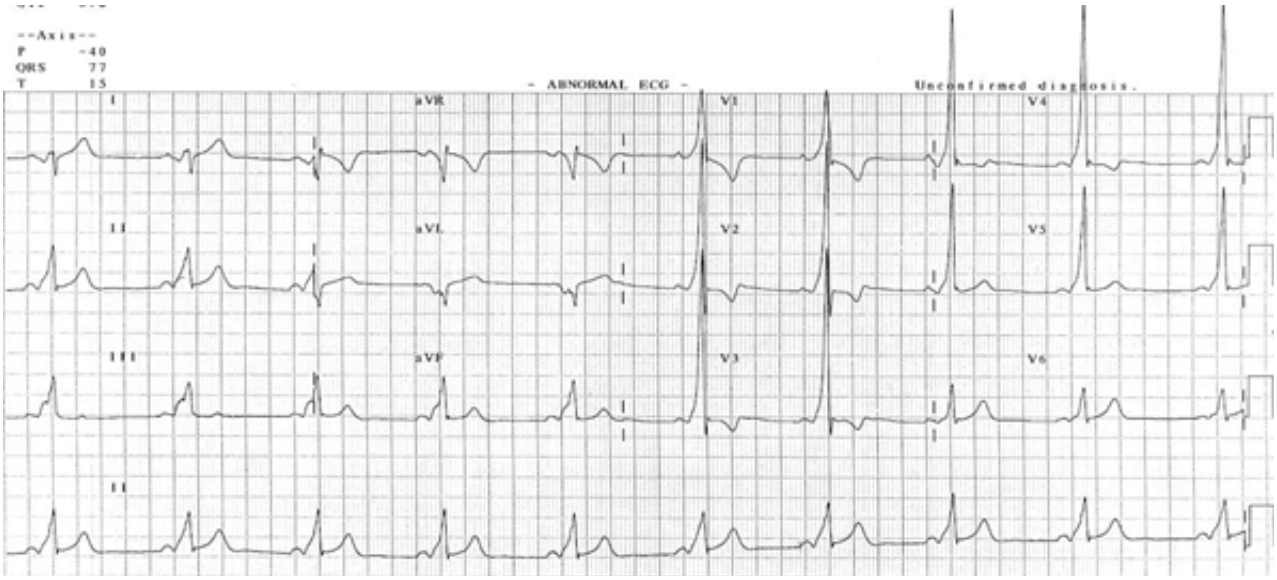

Figure 5. ECG of WPW syndrome in sinus rhythm manifest with ventricular pre-excitation (short PR interval and delta wave and wide QRS complex).

- Regular narrow complex SVT – short RP tachycardia (Figure 6)
- Pre-excited wide complex SVT (Figure 7)
- Pre-excited atrial fibrillation (Figure 8)

There is a 5% incidence of sudden cardiac death in WPW syndrome. This is due to pre-excited AF degenerating into ventricular fibrillation.

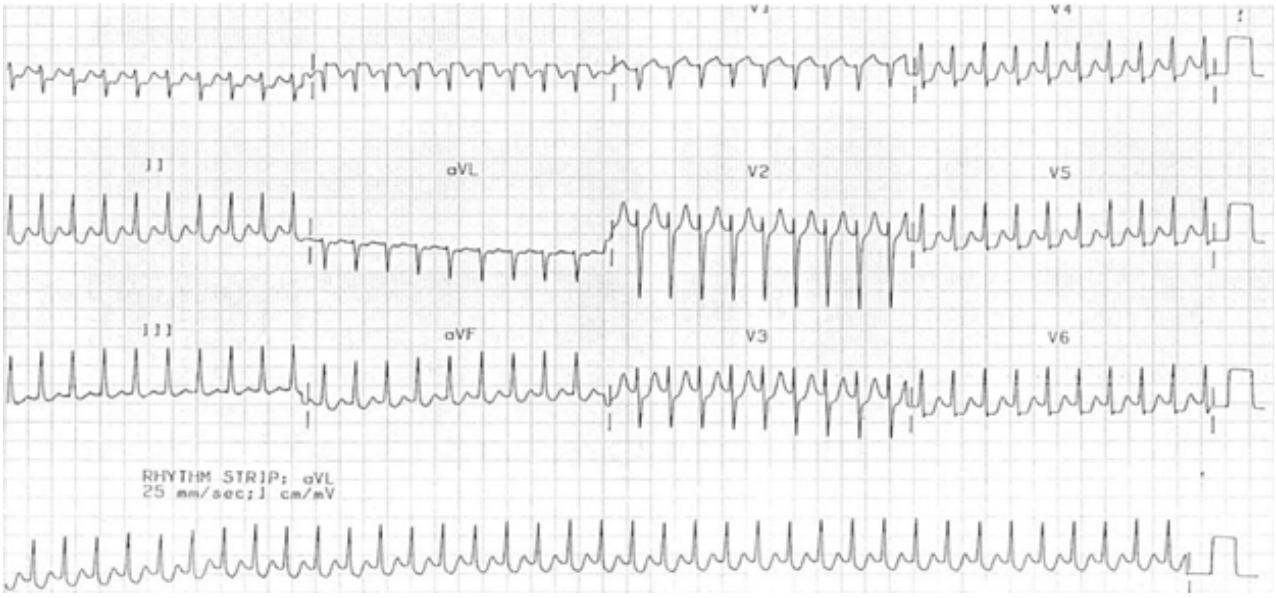

Figure 6. Regular narrow complex short RP tachycardia.

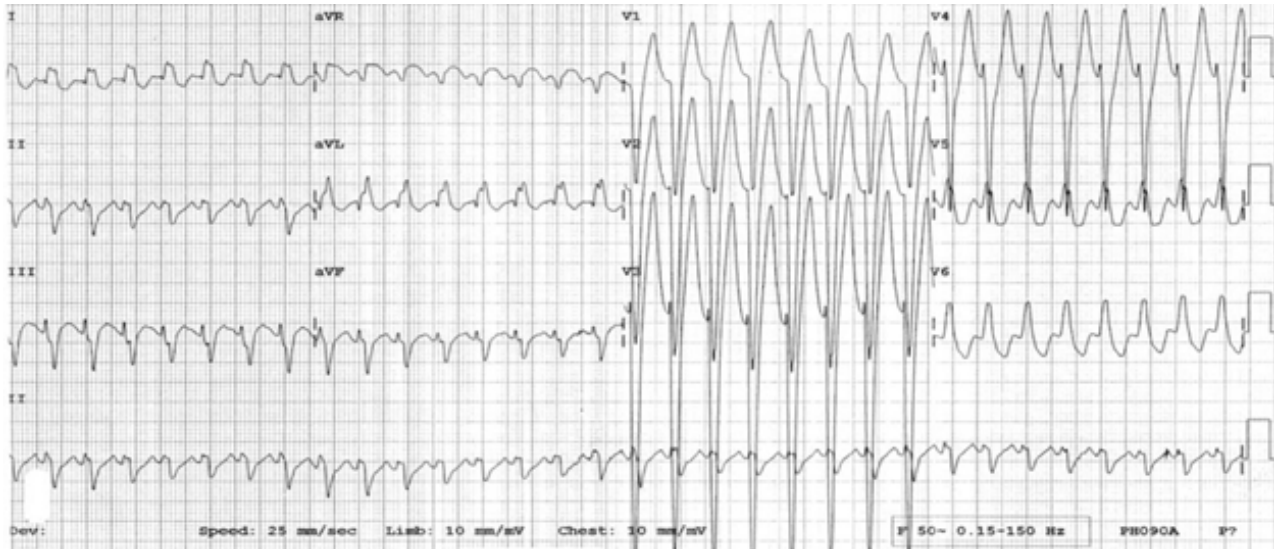

Figure 7. Pre-excited wide complex SVT.

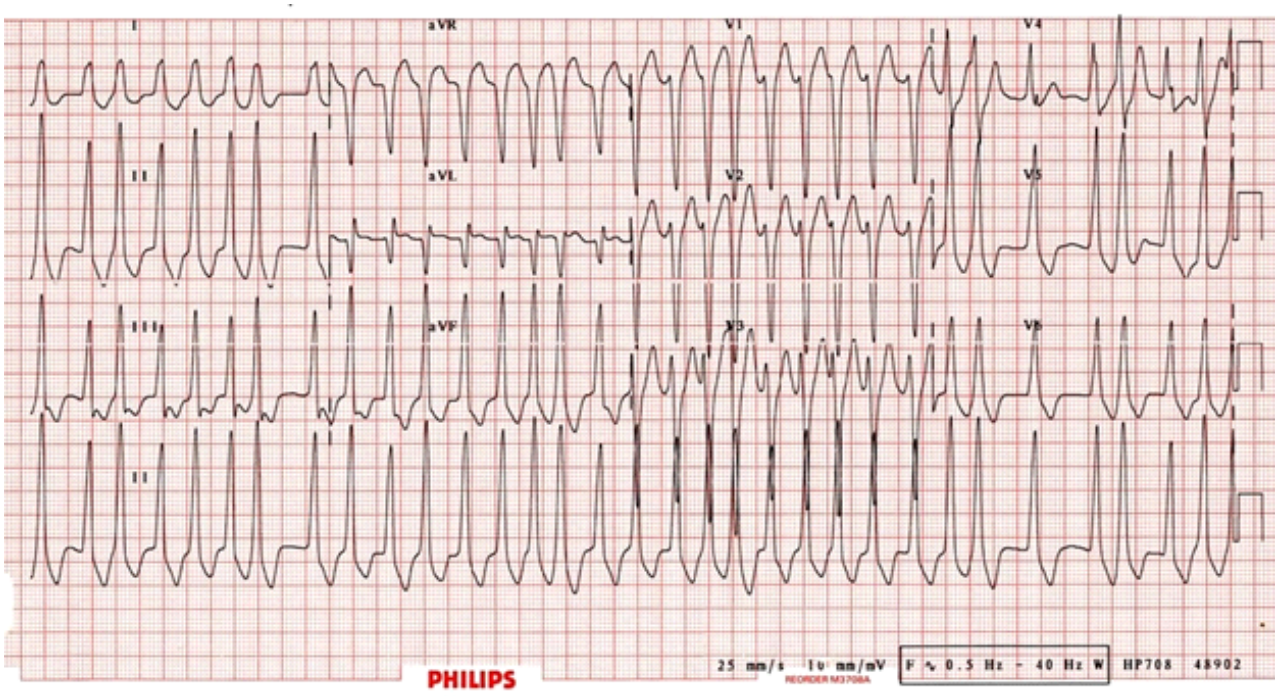

Figure 8. Pre-excited atrial fibrillation in WPW syndrome.

In the management of AVRT, AV nodal blocking agents such as beta blocker, verapamil, digoxin and adenosine can be used in patients with short RP tachycardia. They are contraindicated for patients with pre-exited SVT and atrial fibrillation because it can lead to ventricular fibrillation.[1,3] Such patients should be managed with anti-arrhythmic medications such as procainamide and ibutilide. Amiodarone is not indicated in patients with pre-excited atrial fibrillation. Definitive treatment of accessory pathway-mediated tachyarrhythmia is to do an EP study, which helps to identify the location and RF ablation of the accessory pathway.

Sinus tachycardia

It is the most common tachycardia. In certain patients, it might present with clinically significant cardiac symptoms such as palpitation and dyspnea. Management is targeted towards the treatment of underlying causes such as coronary ischemia, pulmonary embolism, respiratory or cardiac failure, hypovolemia, anaemia, and hyperthyroidism. In some cases (e.g., coronary disease, aortic stenosis, and thyroid storm), rate-controlling drugs such as oral or intravenous beta-blocker might be needed.

Inappropriate sinus tachycardia (IST)

IST is defined as a fast sinus rhythm (>100 bpm) at rest or minimal activity that is out of proportion with the level of physical, emotional, pathological, or pharmacological stress.[3] The tachycardia tends to be persistent, and most of the affected patients are young and female. The underlying mechanisms are multifactorial (e.g. dysautonomia, neurohormonal dysregulation, and intrinsic sinus node hyperactivity). The prognosis of IST is generally considered benign.

The diagnosis is by 24-hour Holter monitoring which demonstrates a mean heart rate >90 bpm with an exaggerated heart rate response >100 bpm during waking hours. The main treatment modalities are reassurance and lifestyle interventions such as exercise training, volume expansion, and avoidance of cardiac stimulants which should be tried before drug treatment.[3] Ivabradine and selective beta blockers are the mainstay of drug treatments, usually a combination of drugs is more effective.

Sinus node re-entrant tachycardia

This tachycardia is due to a re-entry circuit involving the sinus node. In contrast to IST, is characterized by paroxysmal episodes of tachycardia.[3] On the ECG, the polarity and configuration of the P waves are like the configuration of sinus P waves. It is mainly diagnosed by EP study. Verapamil and amiodarone have demonstrated variable success, but beta-blockers are ineffective.[2,3] It can be effectively and safely treated with catheter ablation.

Atrial tachycardia

Atrial tachycardia predominantly seen in those under 10 and over 60 years of age. Atrial tachycardia is divided into focal and multifocal atrial tachycardia.[2,3] Focal atrial tachycardia is usually paroxysmal and self-limited, and arises from a single site or area of micro reentry or enhanced automaticity outside of the sinus node (Figure 9). Multifocal atrial tachycardia is a rapid, irregular rhythm with at least three distinct organized morphologies of P waves on the surface ECG (Figure 10). Multifocal AT is commonly associated with underlying conditions, including pulmonary disease, pulmonary hypertension, coronary disease, and valvular heart disease. Common sites of atrial tachycardia originations are from crista terminalis, pulmonary veins, annulus of valves and triangle of Koch's.

Most patients with atrial tachycardia present with palpitations and with rapid fluttering sensation in the chest or neck. The symptom burden is typically higher in focal compared with reentrant supraventricular tachycardia. The diagnosis of atrial tachycardia is based primarily on electrocardiographic (ECG) findings demonstrating an atrial arrhythmia of >100 beats per minute originating from outside the sinus node with long RP tachycardia. The P wave could be upright or inverted depending on the focus. EP study is the best way to diagnose the atrial tachycardia and its focus.

Hemodynamically unstable patients due to atrial tachycardia will need immediate DC cardioversion. A hemodynamically stable patient with symptomatic atrial tachycardia in acute setting is treated with an oral or intravenous beta-blocker or non-dihydropyridine calcium channel blocker. Intravenous amiodarone is useful in patients who are not responding to calcium channel blocker or beta-blockers.[1,2]

Atrial flutter

This is not a very common tachycardia. The mechanism is due to a macro re-entrant circuit.[3] Atrial flutter usually presents as a regular narrow complex tachycardia; however, it may occasionally present as an irregular ventricular response. It could be categorized as typical or atypical. Typical atrial flutter is the most frequent cavotricuspid isthmus (CTI)-dependent flutter (a macro-reentry circuit around the tricuspid annulus using the CTI as a critical passage at the inferior boundary. (Figure 10). Usually, activation occurs in counter-clockwise pattern which starts from RA free wall, through the CTI and ascends in the right septum. When activation occurs in the opposite direction (clockwise) it results in a different ECG pattern, then called typical reverse flutter.[2,3] ECG findings during typical counter-clockwise atrial flutter show, negative flutter waves in inferior leads (Figure 12) whereas positive flutter waves are seen in a clockwise flutter. Atypical flutters are scar-related, and their presentation varies from asymptomatic to very rapid tachycardia. Diagnosis of atrial flutter is based on ECG and Holter monitoring. EP study helps for definitive diagnosis of atrial flutter as well as RF ablation of a macro reentrant circuit.

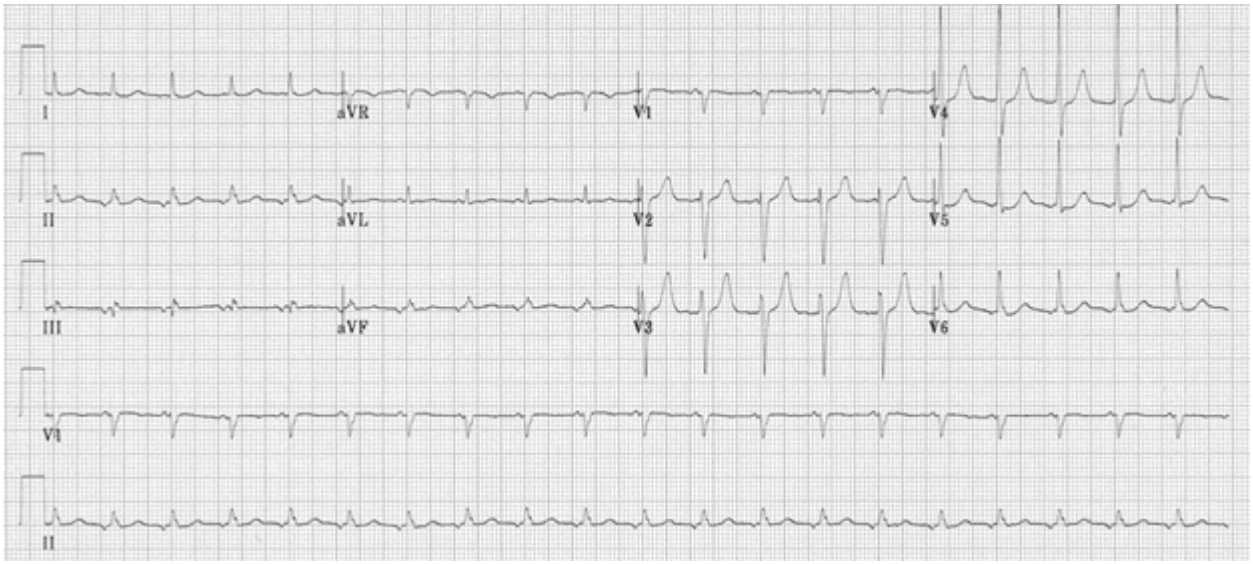

Figure 9. Focal atrial tachycardia.

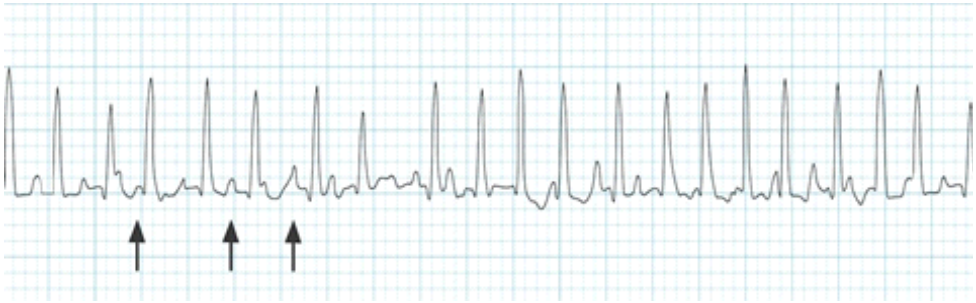

Figure 10. Multifocal atrial tachycardia.

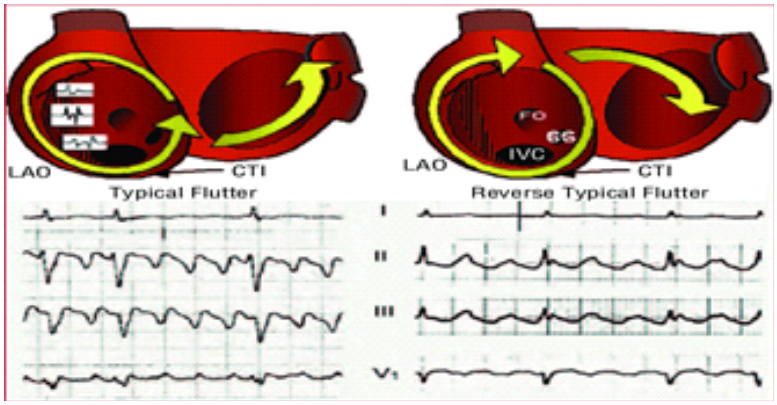

Figure 11. Cavotricuspid isthmus dependent macro reentrant circuit of typical atrial flutter.

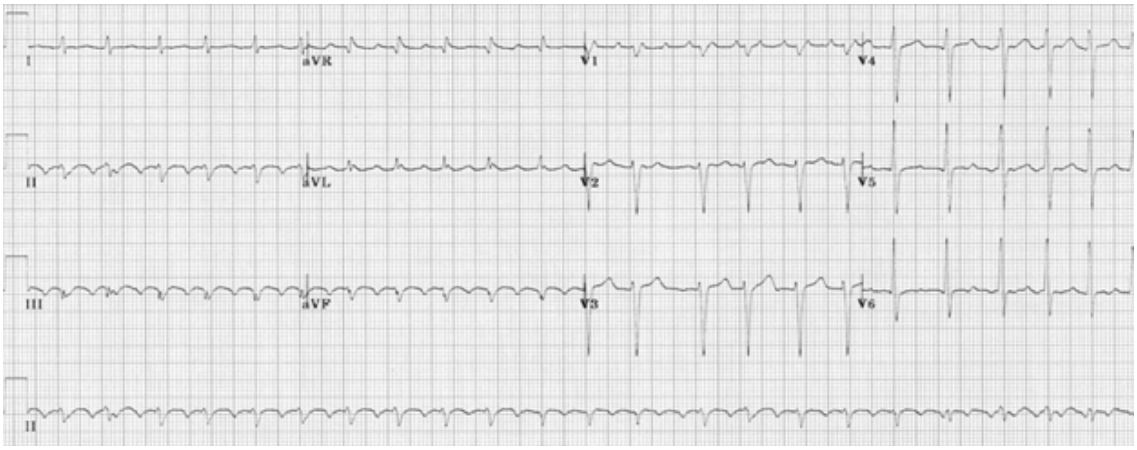

Figure 12. Atrial flutter with 2:1 Block.

Since atrial flutter carries a risk of stroke, the initial approach to the management of atrial flutter is the same as atrial fibrillation. If the patient with new-onset atrial flutter is hemodynamically unstable may need DC cardioversion. A hemodynamically stable patient is managed with rate-controlling medications such as beta blocker, digoxin and calcium channel blocker, or rhythm control medication and antithrombotic therapy. Catheter ablation is the most effective therapy to maintain sinus rhythm and is superior to rhythm controlling agents. Ablation of CTI with confirmed bidirectional conduction block results in less than 10% recurrence of atrial flutter.

Atrial fibrillation

Atrial fibrillation is defined as supraventricular tachyarrhythmia with uncoordinated atrial electrical activation and consequently causing ineffective atrial contraction.[3] It is the most common arrhythmia in clinical practice. Mechanisms of AF are poorly understood but factors leading to electrical instability of the left atrium. Rapidly firing foci from pulmonary veins have been identified as the primary cause of AF. The incidence of AF is related to atrial size and the extent of fibrosis. Some of the factors that may play a role in the mechanisms of AF include autonomic tone, inflammation, atrial pressure and wall stress, and genetics.[2,3] Electrocardiographic features of AF include irregularly irregular R-R intervals (when atrioventricular conduction is not impaired), absence of distinct repeating P waves, and irregular atrial activations (Figure 13).

Even though there are several classifications of AF, for the management purpose AF is mainly classified as acute onset AF (onset <48 hours) and chronic AF (onset >48 hours). AF with a rapid rate presents with symptoms related to the arrhythmia such as palpitations, and syncope. In some patients first presentation of AF could be an embolic phenomenon such as a stroke. Urgent DC cardioversion should be considered for patients with active ischemia, significant hypotension, severe heart failure, or the presence of a pre-excitation syndrome associated with rapid conduction using the accessory pathway. For hemodynamically stable patients rate control is indicated to improve symptoms and to reduce the risk of tachycardia-mediated cardiomyopathy. A targeted heart rate of less than 110 beats per minute is reasonable for an asymptomatic patient with a normal ejection fraction. AV nodal blocking agents (beta-blockers and non-dihydropyridine calcium channel blockers) are preferred as first-line agents in most patients, and digoxin should only rarely be used. Intravenous preparations are preferred to oral preparations when rapid rate control is necessary. Since there are evidence to suggest that rhythm control improvs the quality of life in the symptomatic patient with AF, it's better to maintain sinus rhythm by pharmacological or planned electrical cardioversion. Patients should be commenced on oral anticoagulation according to CHA2DS2 VAS Score.

The key factors of long-term management of AF are lifestyle modifications such as alcohol moderation, weight reduction and physical activity and identification and treatment of precipitating etiology such as hyperthyroidism. Pulmonary venous catheter ablation can be useful in patients who respond poorly to rhythm control medication. The recurrence rate of AF after catheter ablation is related to LA size, AF duration, patient age, and renal function. Left atrial appendage device closure is useful in patients with AF and high-risk factors for stroke.

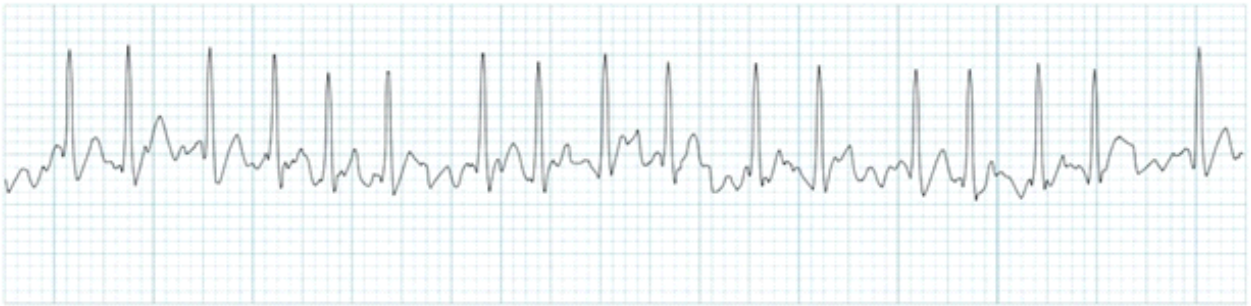

Figure 13. Atrial fibrillation.

Management of wide complex tachycardia

A widened QRS (≥120 milliseconds) occurs when ventricular activation is abnormally slow. Common aetiologies for wide complex tachyarrhythmias are arrhythmia originating outside the normal conduction system (ventricular tachycardia), abnormalities within the His-Purkinje system (SVT with aberrancy), pre-excitation with an SVT conducting antegrade over an accessory pathway. Acute management of wide complex tachyarrhythmia consists of immediate assessment of the symptoms, vital signs, and level of consciousness to determine if they are hemodynamically stable or unstable. In hemodynamically unstable patients, need to obtain an immediate 12 ECG (at least a rhythm strip) and electrical cardioversion/defibrillation should be given regardless of the aetiology to prevent further clinical deterioration or sudden cardiac arrest (SCA). In stable patients, if the ECG is suggestive of SVT with aberrancy, vagal manoeuver and intravenous adenosine can be given.[4,5] If ECG features suggestive of ventricular arrhythmia, it can be treated with anti-arrhythmic medications (IV amiodarone and lignocaine) and AV nodal blocking agents such as beta blockers. Long-term management depends on the aetiology of wide complex tachycardia. Some cases of ventricular tachycardia might need oral antiarrhythmic treatment. Some such as right or left ventricular outflow tract ventricular tachycardia and idiopathic fascicular left ventricular tachycardia can be curable with catheter ablation. However, some ventricular tachycardias are non-ablatable such as channelopathy-related tachycardia (e.g., Brugada syndrome and long QT syndrome). These tachycardias may need device therapy such as ICD implantation.

Conclusion

The modern approach to the management of tachyarrhythmia has changed significantly over the past decade. This is due to the advancement in RF catheter ablation, the introduction of new devices, improvements in imaging modalities, and the practice of new evidence-based medicine. These advancements in patient management improve patient quality of life and reduce mortality.

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