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EC CARDIOLOGY
Review Article

Basic Understanding of Supraventricular Tachycardia for Post Graduates

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Abstract

While dealing with ECG of tachycardia, it is important to differentiate between ventricular and supraventricular tachycardia, further it is important to determine type of supraventricular tachycardia. Different type of SVT requires individually different approach and management. Present article summarize briefly about different types of SVT and how to approach them.

Keywords: *SVT: Supraventricular Tachycardia; WCT: Wide Complex Tachycardia; WPW: Wolff Parkinson White Syndrome*

Introduction

Tachyarrhythmias are divided into supraventricular tachycardia (SVT) and ventricular tachycardia (VT). SVT originates either from sinus node, atria, AV node or his bundle. Focus of SVT lies above the ventricle whereas focus of VT located in ventricle itself. Then depending upon the QRS duration tachycardia are divided into narrow complex (QRS ≤ 120 msec) tachycardia or (QRS ≥ 120 msec) tachycardia. The narrow complex tachycardia is always supraventricular in origin, whereas wide complex tachycardia is mostly ventricular tachycardia. Some of wide complex tachycardia are having supraventricular origin e.g. SVT with concurrent bundle branch block, SVT with aberrant conduction over accessory pathway [1].

There are different criteria's to differentiate between SVT and VT like Brugada criteria, Miller criteria, Wellens criteria and Kindwall Criteria. ECG criteria to differentiate between SVT and VT are shown in table 1. Brugada criteria's to differentiate between SVT and VT are described in table 2 and in table 3.

Favour supraventricular tachycardia	Favour ventricular tachycardia
Tachycardia complexes identical to those in sinus rhythm	Tachycardia beats identical to PVCs during sinus rhythm
Initiation with premature P wave	Initiation with premature QRS complex
QRS contours consistent with aberrant conduction (V_1, V_6)	QRS contours inconsistent with aberrant conduction (V_1, V_6)
Slowing or termination by vagal tone	AV dissociation, Fusion beat, Capture beat
QRS duration < 140 msec	QRS duration > 140 msec Left axis deviation (between -90^0 to -180^0) Concordant R wave progression pattern Contra lateral bundle branch block pattern from resting rhythm Initial R, q, or r > 40 msec or notched Q in aVR Absence of an "rS" complex in any precordial lead

Table 1: *Electrographic distinction for diagnosis of wide complex Tachycardia.*

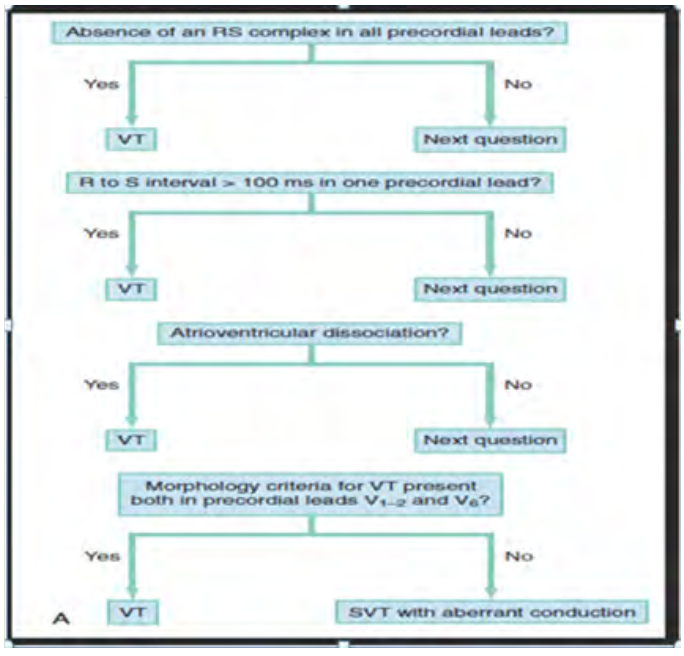


Table 2*: Brugada algorithm to differentiate VT from SVT with aberrant conduction.*

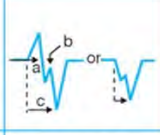
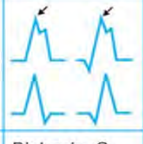
	LBBB		RBBB	
	VT	**SVT**	**VT**	**SVT**
Lead V_1	In V_1, V_2 any of: (a) r ≥ 0.04 sec (b) Notched S downstroke (c) Delayed S nadir > 0.06 sec	In V_1, V_2 absence of: (a) r ≥ 0.04 sec (b) Notched S downstroke (c) Delayed S nadir > 0.06 sec	Taller left peak Biphasic RS or QR	Triphasic rsR′ or rR′
				
Lead V_6	Monophasic QS		Biphasic rS	Triphasic qRs
				

Table 3: *Morphologic criteria in V1 and V6 for VT and SVT differentiation, as described in brugada criteria.*

Types of Supraventricular tachycardia

SVTs (excluding atrial fibrillation or flutter and multifocal AT) have an estimated incidence of 35 per 100,000 person-years, with a prevalence of 2.29 per 1,000 persons [4]. Although AVNRT is the most common SVT in adults (approximately 50 to 60 percent) [2], AVRT is most common in children (accounts for approximately 30 percent of all SVTs) [3]. Types of SVT are:

1. Sinus tachycardia
2. Atrial tachycardia

3. Multifocal atrial tachycardia (MAT)
4. Atrial flutter and fibrillation
5. Atrioventricular nodal reentrant tachycardia (AVNRT)
6. Atrioventricular reentrant tachycardia (AVRT).

Differentiation of SVT based on many factors [5] (Figure 1-3)

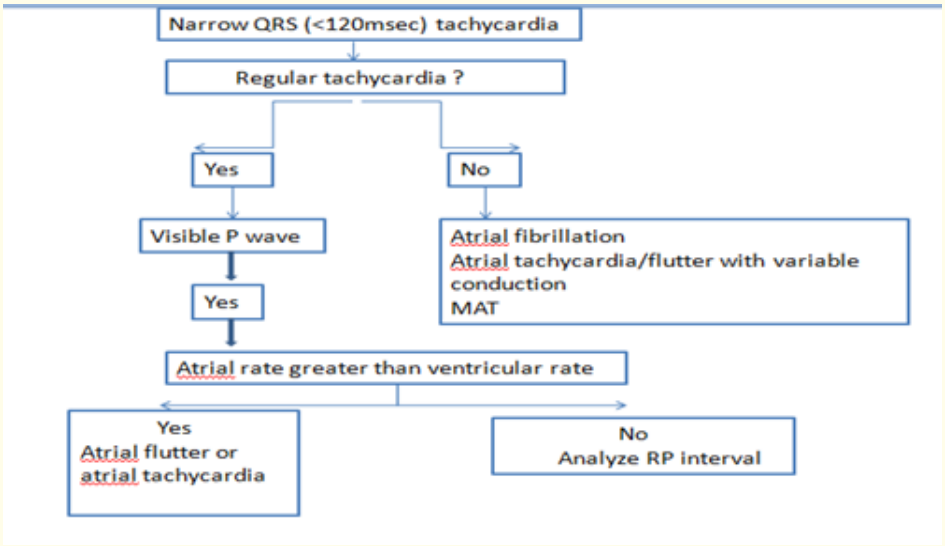


Figure 1: *Algorithm for diagnosis of narrow complex tachycardia.*

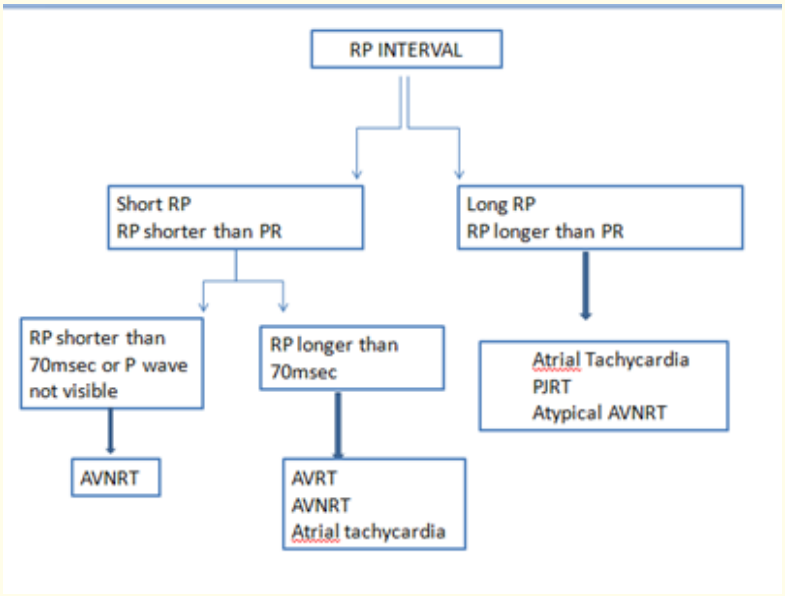


Figure 2: *Differentiation of SVT based on RP interval.*

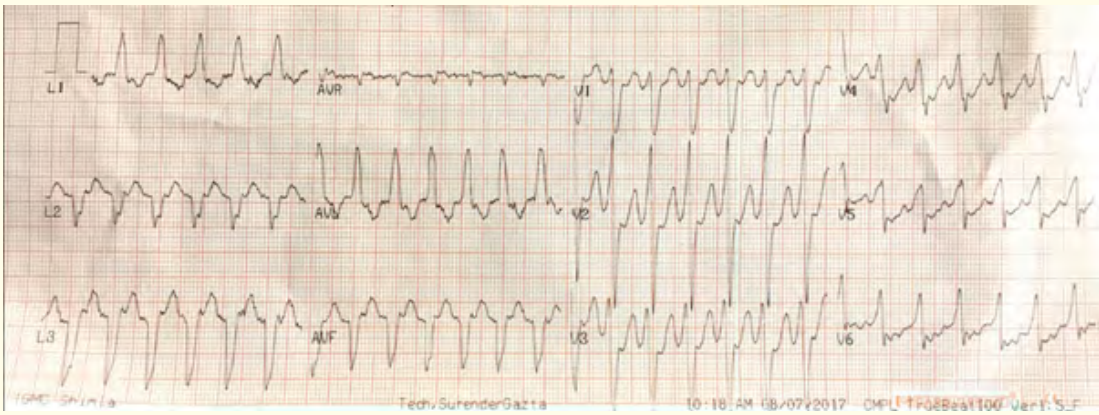


Figure 3: *ECG showing tachycardia HR around 150/min and QRS < 120 msec, No concordant R wave progression in precordial leads, no fusion or capture beat, no tall R wave in avR and P wave falls on ST segment after every QRS complex, these findings are suggestive of SVT.*

1. Whether rhythm is regular or not
2. If regular, whether P waves are visible or not
3. If P waves are visible then whether atrial rate is greater than ventricular rate or not
4. If atrial rate is not more than ventricular rate, then look for RP interval.

Short RP tachycardias are

1. Typical AVNRT
2. AVRT

Long RP tachycardias are

1. Atrial tachycardia,
2. PJRT
3. Atypical AVNRT.

Sinus tachycardia

Sinus tachycardia arises as a response to physiologic (exercise, fever), emotional, pathological (acidosis, hypoxia), and pharmacologic stimuli (alcohol, caffeine, amphetamines). There is gradual increase in heart rate to more than 100 bpm/min and ECG shows normal regular R-R interval, normal P wave and PR interval and narrow complex QRS [6].

Treatment: Treatment is based on identifying and addressing the underlying physiologic disturbance.

Inappropriate Sinus Tachycardia (IST): IST is associated with a persistently exaggerated increase in sinus rate out of proportion to the level of physiologic and/or metabolic stress [7]. The enhanced automaticity and/or abnormal autonomic regulation of the sinus node plays role in genesis of IST [8]. Proposed mechanisms include increased sympathetic tone, increased sympathetic receptor sensitivity, blunted parasympathetic tone, and sympathovagal imbalance and most common in females of age in between 20 - 45 years. Palpitations are the predominant symptom. The diagnostic criteria's are listed in table 4. Treatment includes use of beta blockers or calcium channel blockers [9].

1.	Heart rate 100 bpm at rest or with minimal stress, as confirmed with 24-hour Holter monitoring.
2.	Reduction in the average heart rate during night time hours.
3.	P-wave morphology and endocardial activation same as during sinus rhythm.
4.	Duration of tachycardia and symptoms are chronic and nonparoxysmal.
5.	Cannot be induced or terminated in the EP laboratory using standard programmed stimulation.
6.	Exclusion of systemic causes of rhythm abnormality.

Table 4: *Diagnostic criteria for inappropriate sinus tachycardia.*

Atrial Tachycardia

AT originates outside the sinus node and atrial rate is more than 100 bpm/min. Atrial tachycardia focus may be located either in left atrium or in right atrium. It is a microfocus and mechanisms responsible for generation of atrial tachycardias are enhanced automaticity, triggered activity or microreentry [11]. On right side, sites from where tachycardia arises are crista terminalis, tricuspid valve annulus and perinodal area. On left side originating sites are pulmonary veins (most common), mitral annulus, coronary sinus musculature or appendage [10,11].

In the adult population, AT is a relatively rare arrhythmia, comprising 10% of SVTs encountered in most EP laboratories [10]. In children, AT accounts for a greater percentage of SVTs (up to 14 - 23%) [3,12].

Conditions associated with AT have been reported and they include congenital heart disease (pre- and post-surgical repair), cardiomyopathy, severe pulmonary disease and associated cor pulmonale, mitral stenosis, post AP ablation, post cardiac transplantation, theophylline toxicity, digoxin toxicity, atrial tumors and pregnancy.

ECG of atrial tachycardia

1. It is long RP-short PR tachycardia (Figure 4,5).
2. Atrial rate is 100 - 250 bpm, ventricular rate varies.
3. As origin of P wave is ectopic rather than sinus, so P waves are inverted in inverted in the inferior leads II, III and aVF.
4. At least three consecutive identical ectopic p waves.
5. QRS complexes usually narrow and regular and may be irregular.
6. Isoelectric baseline (unlike atrial flutter).
7. AV block may be present - this is generally a physiological response to the rapid atrial rate, except in the case of digoxin toxicity where there is actually AV node suppression due to the vagotonic effects of digoxin, resulting in a slow ventricular rate ("PAT with block")

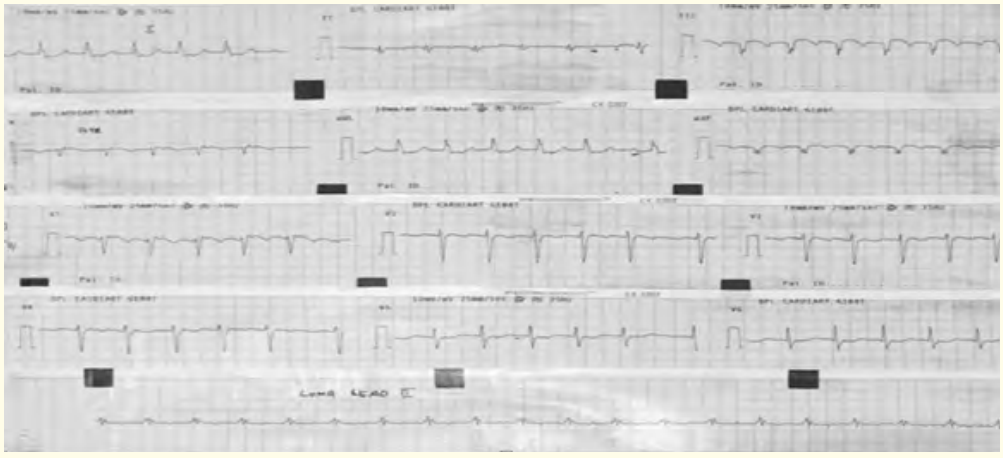


Figure 4: *Ectopic atrial tachycardia, retrograde P wave in lead inferior leads II, III and aVF and RP interval is more than PR interval.*

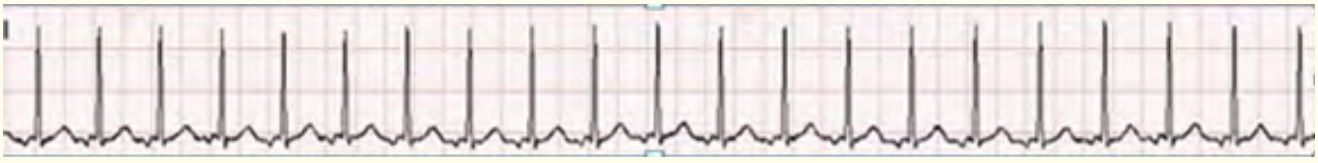


Figure 5: *Atrial tachycardia, long RP tachycardia with inverted P waves.*

Treatment: Depending on the clinical situation, a beta blocker or a calcium channel blocker can be administered to slow the ventricular rate; if atrial tachycardia is still present, class IA, IC, or III drugs can be added. Catheter ablation procedures are generally effective in eliminating the atrial tachycardia, depending on the mechanism and underlying heart disease.

Multifocal atrial tachycardia

This SVT is characterized by irregular P-waves with several different morphologies [13]. It is commonly associated with pulmonary disease as at least 60% of patients with this arrhythmia have pulmonary disease [14,15]. Specific pulmonary diseases include chronic obstructive pulmonary disease (COPD), bronchitis, pneumonitis, and pulmonary embolism. Electrolyte abnormalities like hypokalemia and hypomagnesaemia are also common causes.

ECG of MAT

1. Atrial rate between 100 - 130/min (Figure 6).
2. Multiform P waves with at least three different P waves with three different PR intervals [16]. The P waves can be positive, negative, peaked, or bifid and are most clear in leads II, III, and V1
3. The baseline between PP interval is isoelectric and PP, PR and RR intervals are irregular [16].

Treatment: Included treatment of underlying cause.

Atrial Flutter

Atrial flutter is the prototypic macroreentrant atrial rhythm. The macroreentrant circuit dependent on cavotricuspid isthmus and is further divided into clockwise typical flutter and counter clockwise typical flutter. To identify the location of flutter is important prior to radiofrequency ablation. Non cavotricuspid isthmus (CTI), dependent atrial macro reentry, the atypical flutter can occur in either the right

or left atrium (Table 5). The presence of surgical atrial scars after correction of congenital heart disease or acquired lesions is responsible for genesis of macro reentry. The substrate underlying typical AFL (either counter clockwise or clockwise) has been well-defined and demonstrated to be amenable to curative radiofrequency catheter ablation with a high degree of safety and long-term success. In atypical AFL, the location of the circuit is variable and may involve dual-loop re-entry or complex re-entrant mechanisms [17].

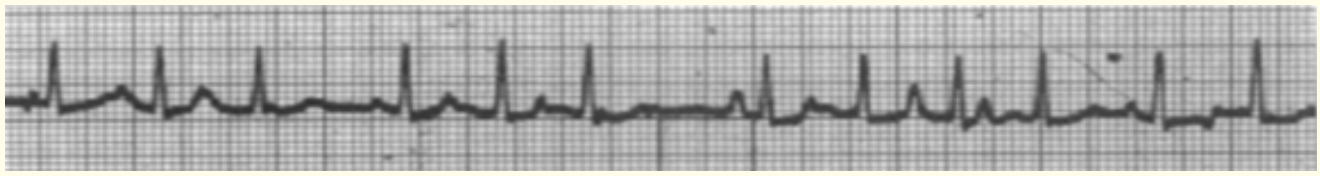


Figure 6: *Multifocal atrial tachycardia, three different P waves with three different PR interval with irregular rhythm.*

Typical counter clockwise	Tricuspid annulus dependent on the CTI	Sawtooth flutter wave; negative in II, III, and aVF	Positive V_1 Negative V_6
Typical clockwise	Tricuspid annulus dependent on the CTI	"Inverse sawtooth"; positive and often notched in II, III, and aVF	Broad and negative in V_1 (often notched) Positive in V_6
Lower loop reentry	CTI	Usually similar to typical counter clockwise CTI flutter except subtle loss of terminal positive deflection in leads II, III, and aVF	Usually similar to typical counter clockwise
Upper loop reentry	SVC and upper crista terminalis	Similar to typical clockwise flutter	Similar to typical clockwise flutter
Right atrial free wall	Around areas of scar in the lateral or posterior right atrium (caused by previous atrial surgery or spontaneously)	Variable	Typically negative or biphasic with terminal negative deflection in V_1
Septal atrial flutter	Atrial septum, typically after previous surgery	Variable	Usually biphasic or isoelectric in V_1

Table 5: *Characteristics of Different Types of Atrial Flutter and Distinguishing Features on Scalar Electrocardiography.*

ECG of Atrial Flutter

1. Narrow complex tachycardia with atrial rate (250 - 350) bpm (Figure 7-9)
2. Saw tooth pattern of flutter waves is seen in leads II, III, aVF
3. Anticlockwise re-entry [18] is the commonest form of atrial flutter (90% of cases). Retrograde atrial conduction produces:
    a. Inverted flutter waves in leads II,III, aVF
    b. Positive flutter waves in V1
4. Clockwise re-entry [18] is uncommon variant and produces the opposite pattern:
    a. Positive flutter waves in leads II, III, aVF
    b. Broad and inverted flutter waves in V1
5. Ventricular rate is fixed and is fraction of atrial rate. 2:1 block = 150 bpm, 3:1 block = 100 bpm, 4:1 block = 75 bpm

Treatment: Cardioversion by synchronous low energy (50 joule) restores sinus rhythm quickly. The ibutilide can also be given intravenously to convert atrial flutter into normal sinus rhythm. Ibutilide appears to successfully cardiovert approximately 60% to 90% of episodes of atrial flutter. For rate control beta blockers or calcium channel blockers can be given. For prevention of clot formation, anticoagulation is necessary [18]. In case of recurrent drug refractory episodes of atrial flutter, ablation of macroreentrant circuit is required [19].

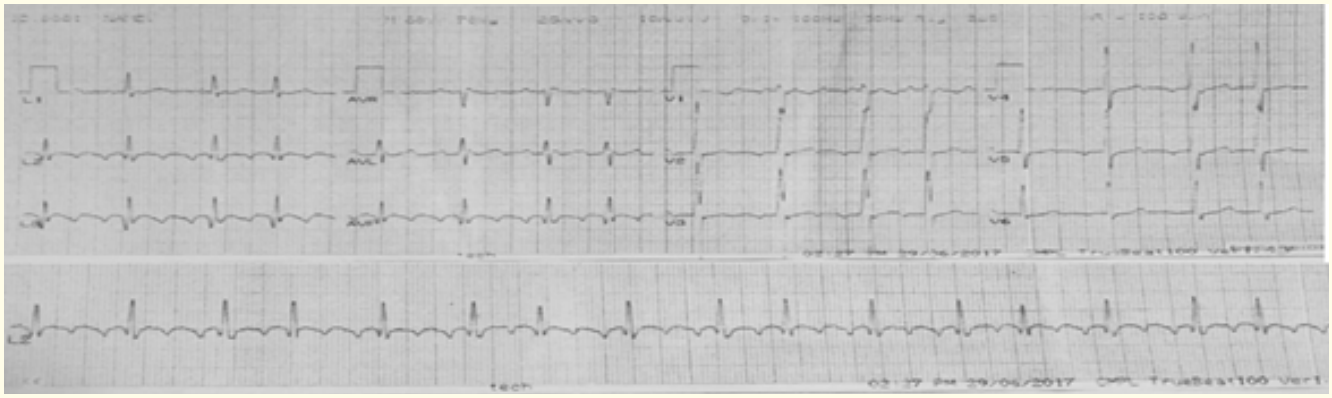


Figure 7: *Type 1 typical Atrial Flutter. Notice inverted saw tooth P waves in leads leads II, III, aVF at rate 300bpm and ventricular rate varies between 100bpm to 150bpm as seen on rhythm strip i.e. 3:1 block and 2:1 block.*

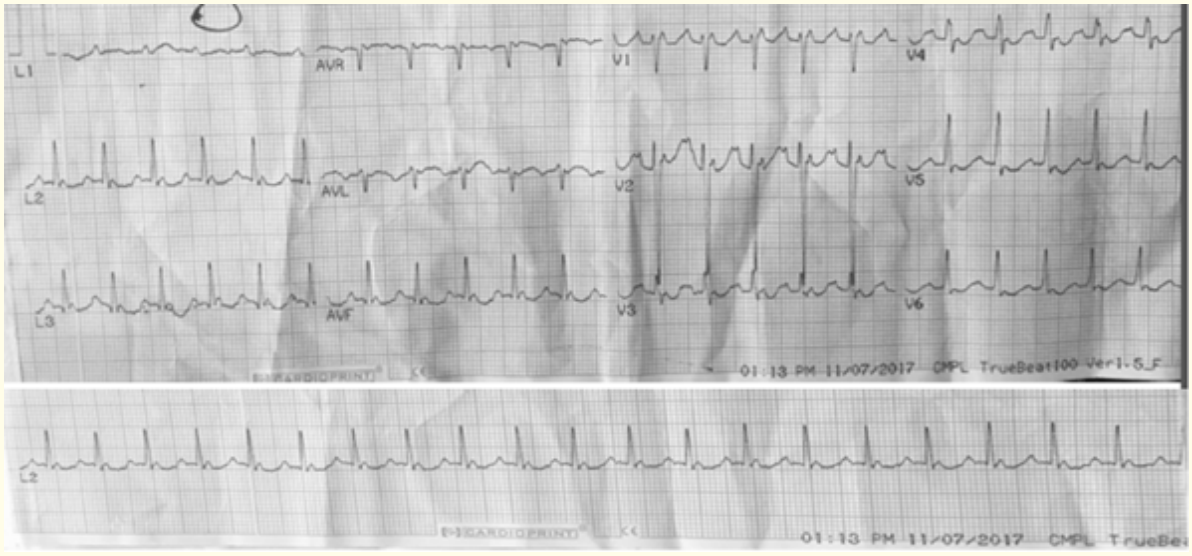


Figure 8: *Atrial flutter. Notice flutter waves in leads II, III, aVF at rate 300bpm and ventricular rate at 150/min i.e. 2:1 block. Flutter waves are positive in lead leads II, III, aVF suggestive of atypical flutter.*

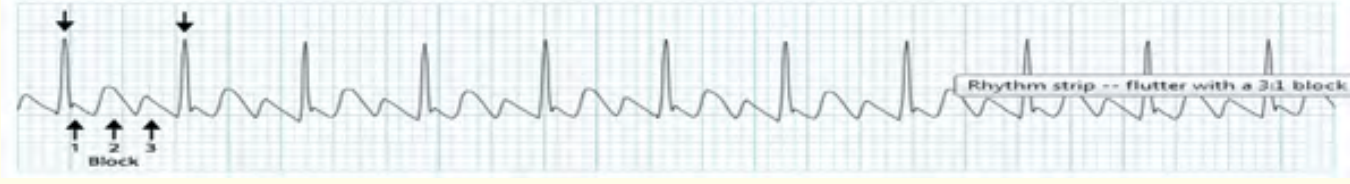


Figure 9: *Atrial flutter with 3:1 block.*

Atrial fibrillation

Atrial fibrillation (AF) is a supraventricular arrhythmia characterized electrocardiographically by low-amplitude baseline oscillations (fibrillatory or f waves) and an irregularly irregular ventricular rhythm. The f waves have a rate of 300 to 600 beats/min and are variable in amplitude, shape, and timing. The ventricular rate during atrial fibrillation in the absence of AV node blocking agents is typically 100 to 160 beats/min [20].

Types of AF depending upon the duration [21]

1. Paroxysmal atrial fibrillation terminates spontaneously within 7 days.
2. Persistent atrial fibrillation present continuously for more than 7 days.
3. Longstanding atrial fibrillation is persistent for longer than 1 year.
4. The longstanding atrial fibrillation refractory to cardioversion is termed permanent AF. However the permanent atrial fibrillation is not necessarily permanent in the literal sense because it may be eliminated successfully by surgical or catheter ablation.
5. Lone atrial fibrillation occurs in patients younger than 60 years who do not have hypertension or any evidence of structural heart disease [21].

The incidence of AF is age and sex related and ranges from 0.1% per year before the age of 40 years to higher than 1.5% per year in women and higher than 2% per year in men older than 80 years [20].

Mechanism involves initiation of automatic, triggered, or microreentrant foci, so-called drivers from pulmonary veins and perpetuation by multiple reentrant circuits meandering throughout the atria that annihilate and reform wavelets.

Causes include valvular heart disease most commonly mitral valve disease, HTN with HHD, HOCM, DCM, pericardial diseases, hyperthyroidism, alcohol (holiday heart syndrome) and OSA [17,18].

ECG of Atrial fibrillation

1. Irregularly irregular rhythm (Figure 10).
2. P waves are not seen. The fibrillatory F waves having amplitude < 0.5 mm) at rate of 300 to 600 beats/min are there [20].
3. Absence of an isoelectric baseline.
4. Variable ventricular rate 100 - 160/min.
5. QRS complexes are usually narrow < 120 ms except in cases having pre-existing bundle branch block, accessory pathway, or rate related aberrant conduction [1].
6. The ventricular rate can be < 60bpm, called slow AF. It can be seen with digoxin toxicity.

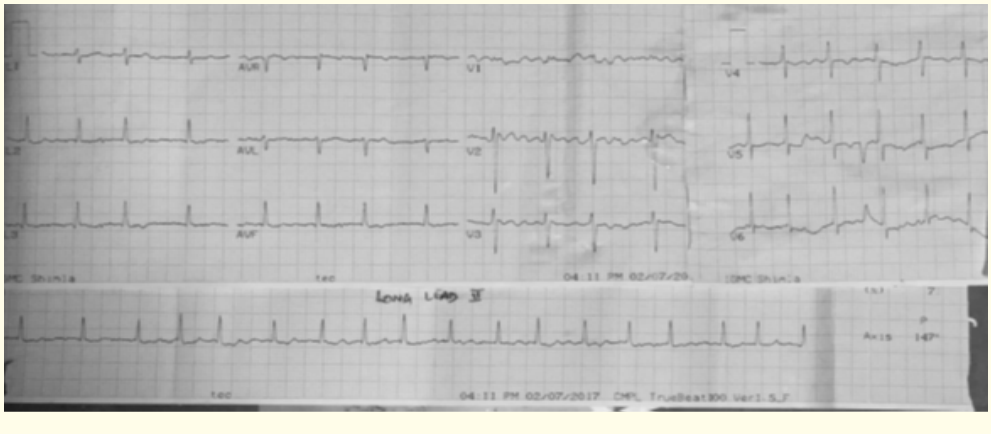


Figure 10: *Atrial fibrillation, note Irregularly irregular rhythm, No P waves, Absence of an isoelectric baseline, Variable ventricular rate, QRS complexes usually < 120 ms and fibrillatory waves in long lead 2.*

Treatment: Treatment [20,22] algorithm is explained in figure 11.

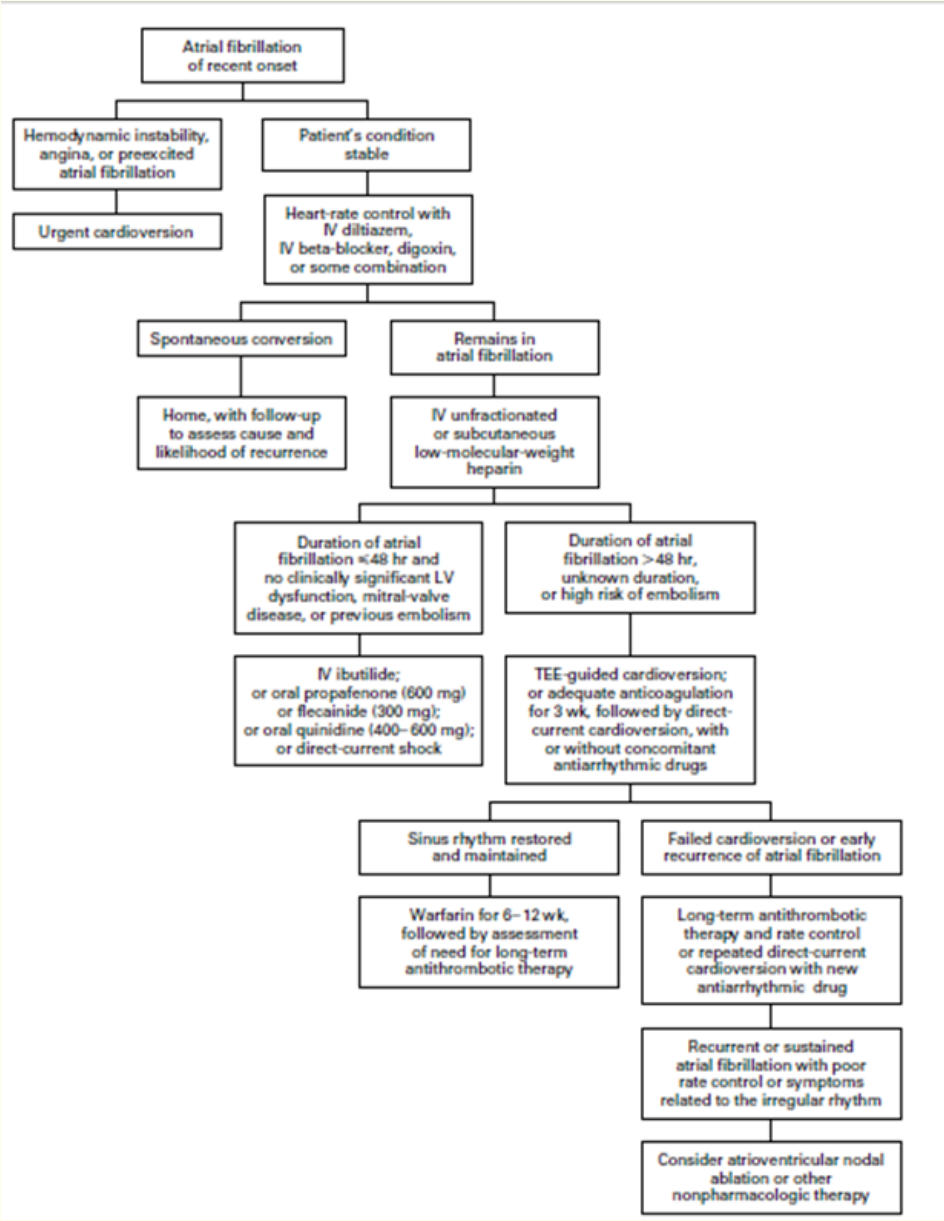


Figure 11: *Management of atrial fibrillation.*

Atrioventricular nodal reentrant tachycardia (AVNRT)

It is common type of SVT, seen in structurally normal heart, in 3rd - 4th decade. The coexistence of slow and fast pathways in atrioventricular nodal tissue is the basis of aberrant substrate for reentrant tachyarrhythmias [23].

Dual AV nodal physiology may be distinct anatomic structures, or may be functionally separate which results in reentry (Figure 12)

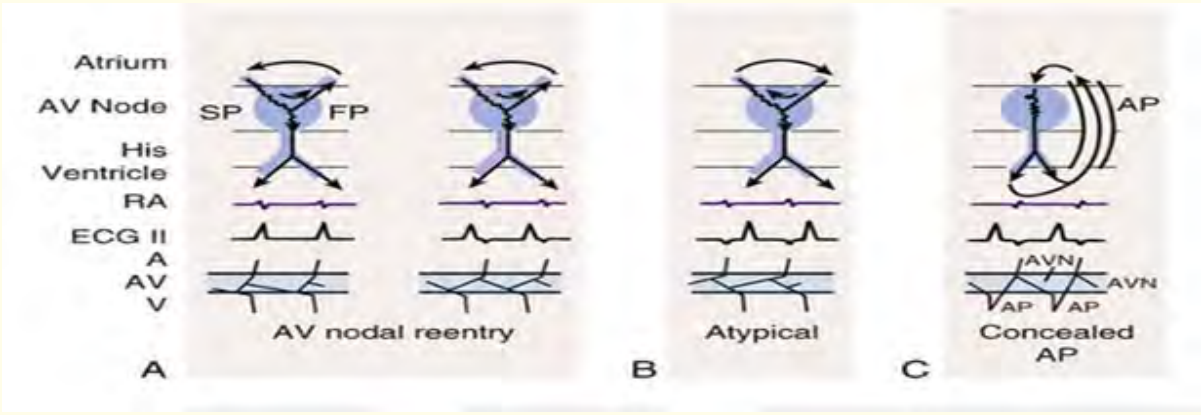


Figure 12: *A) Typical AVNRT showing dual pathways in AV node typical AVRT occurs when antegrade conduction occurs through slow pathway and when impulse reaches junction, the refractory period of fast pathway completed, so retrograde conduction towards atria resulting in hidden P waves in QRS or psedo R or pseudo S patter B) Atypical AVNRT through fast slow conduction, delayed retrograde activation of atria and long RP interval than PR interval C) In presence of concealed accessory pathway as in AVRT retrograde activation of atria through this pathway resulting in p wave falls on ST segment RP > 70 msec but less than PR.*

1. Fast or beta pathway- conducts rapidly and has a relatively long refractory period.
2. Slow or alpha pathway- conducts slowly and has a shorter refractory period.

Typical AVNRT: Most common type of conduction is slow- fast conduction i.e. antegrade conduction towards ventricle through slow pathway and retrograde conduction towards atria through fast pathway, resulting in simultaneous activation of both atria and ventricle. This sets microrentry circuit in AV node which results in tachycardia. ECG changes of retrograde P wave buried in QRS or seen just before or just after QRS as pseudo r or pseudo s pattern and short RP < 70 msec. On EP study AH/HA > 1, VA interval < 60 msec and earliest retrograde atrial activation is in right septum his bundle (R-HIS) electrogram [22].

Atypical AVNRT- Type of conduction is fast- slow conduction i.e. conduction towards ventricle through fast pathway and retrograde conduction towards atria through slow pathway resulting in delayed atrial activation. So in ECG p waves fall on ST segment after QRS, resulting in long RP interval. On EP study AH/HA < 1, VA interval > 60 msec and earliest retrograde atrial activation is in coronary sinus [24].

It presents as palpitations, nervousness, anxiety and syncope. Syncope is due to rapid rate results in low cardiac output or because of asystole when tachycardia terminates as a result of tachycardia induced depression of SA node automaticity.Prognosis of patients without heart disease is good.

ECG of AVNRT

1. Narrow complex tachycardia (Figure 13,14)
2. Sudden onset and termination generally at rates of between 150 and 250 beats/minute (usually 180 to 200 beats/minute in adults), and a regular rhythm
3. Absent P waves
4. In typical AVNRT, frequently, P waves are buried in the QRS complexes because of simultaneous activation of atria and ventricles and it is most common presentation of AVNRT seen in 66% cases. If not synchronous then pseudo s wave in inferior leads, pseudo r' wave in lead V1 seen in 30% cases, resulting in subtle alteration of QRS complex (Figure 13, 14)
5. In Atypical AVNRT, retrograde atrial activation through slow pathway resulting in long RP tachycardia.

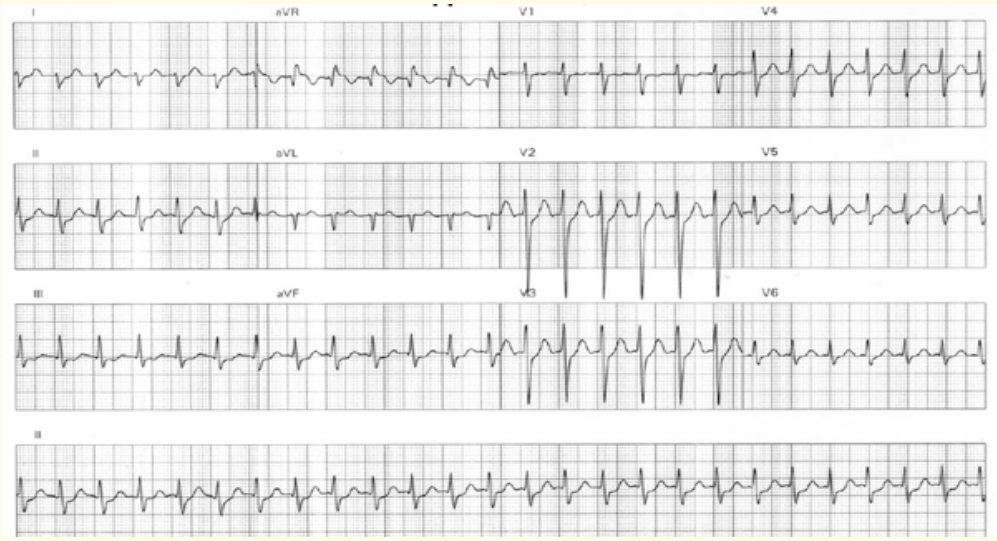


Figure 13: *Typical AVNRT, narrow complex tachycardia, absent P waves with regular R-R interval.*

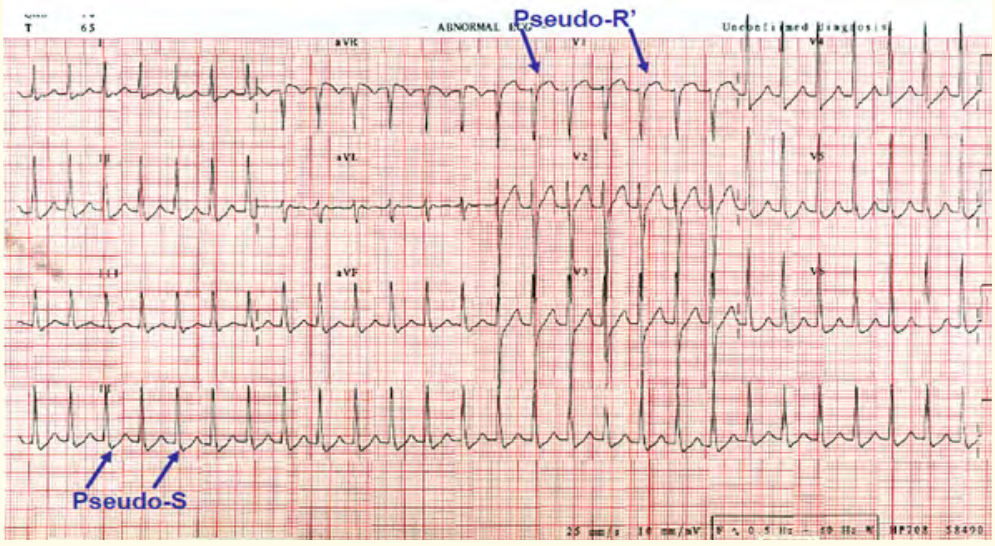


Figure 14: *Typical AVNRT showing pseudo R in V1 and pseudo S in inferior leads.*

Treatment- Acute attack requires valsalva manoeuvres, i.e. push bolus of 6 mg adenosine (half life 6sec) that reverts 90% of AVNRT, if not reverted adenosine can be repeated at 12 mg dose and if still not then Synchronized DC shock of 25 - 50J can be given. Avoid giving adenosine if patient is having bronchospasm or patient is on theophylline.

In HD unstable patient- Give synchronized DC shock 10 - 50J. Be careful in giving DC shock to patients receiving digitalis as it can lead to ventricular fibrillation [25].

Long term management depends on frequency of attacks. If attacks are less frequent then treatment is not required. In case of frequent attacks of AVNRT, treatment with beta blockers or calcium channel blocker should be started [23]. In of drug refractory cases, RFA

is good option. The slow pathway ablation preferred over fast pathway ablation (fast pathway ablation causes 1st degree AV block 10 - 15% and CHB 2 - 5%). RFA has 95% success rate, reoccurrence rate < 2% and occurrence of CHB is < 1% [22-24].

Atrioventricular reentrant tachycardia (AVRT)

Accessory pathways are fibers that connect the atrium or AV node to the ventricle outside the normal AV nodal–His-Purkinje conduction system. These pathways can conduct impulses in the forward (anterograde from the atrium to the ventricle) or reverse (retrograde from the ventricle to the atrium) direction and are potential substrates for reentrant tachycardias (AV reciprocating tachycardia). When the pathway is capable of anterograde conduction, the ventricle can be depolarized in part by the accessory pathway (outside the normal His-Purkinje system) and produces a QRS complex that is preexcited. In some cases the accessory pathways are able to conduct only in the retrograde direction, so in resting electrocardiogram they do not produce any ventricular preexcitation and these pathways are known as concealed pathways [23,24].

Reentry over a Concealed (Retrograde-Only) Accessory Pathway

1. The accessory pathway that conducts unidirectionally from the ventricle to the atrium but not in the reverse direction (Figure 12.C)
2. Resting ECG normal and there was no preexcitation features in resting ECG.
3. Macroreentry results in AVRT. This type is known as Orthodromic AVRT.
4. The tachycardia resulting from this mechanism can be suspected when the QRS complex is normal and the retrograde P wave occurs after completion of the QRS complex, in the ST segment, or early in the T wave
5. Sometimes the P wave is not clearly visible and can result in depression of the ST segment; when this is seen during tachycardia, the mechanism of the arrhythmia is most often reentry involving an accessory pathway (AV reentrant tachycardia)

Clinical features are same as that described in AVNRT

ECG of AVRT

1. Narrow complex tachycardia
2. Sudden onset and termination generally at rates of between 150 and 250 beats/minute (usually 200 to 250 beats/minute in adults), and a regular rhythm (Figure 15)
3. When P wave is not visible as it falls on ST segment resulting in depression of ST segment.
4. If P is visible than RP interval is more than 70msec, but it is less than PR interval.

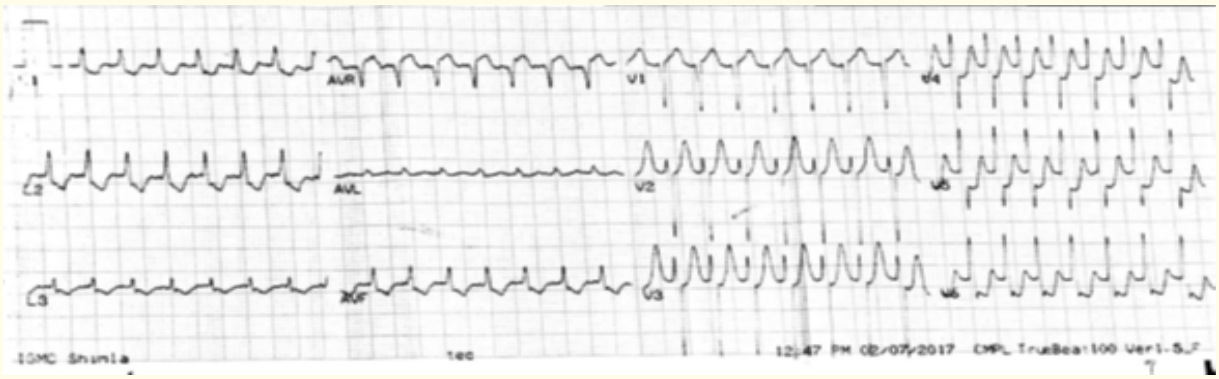


Figure 15: *AVRT showing narrow complex tachycardia with HR of 214/min, absent P waves, absent pseudo R and pseudo S pattern and characteristic ST segment depression in multiple leads.*

When ventricular activation occurs through accessory pathway and retrograde atrial activation occurs through AV node although less common (< 5% cases of AVRT), it is called as antidromic AVRT. This produces ECG of wide complex tachycardia. This is SVT with aberrancy, which should be differentiated from VT by different criteria's (Table 1-3) as management differs in both of these entities.

Treatment is same as of AVNRT. Table 6 differentiates features of AVNRT and AVRT.

	AVNRT	AVRT
Rate	168 ± 25	179 ± 26
RP interval in msec	< 70	> 70
Pseudo R or Pseudo S (most specific)	Yes	No
ST depression	No	Yes
ST elevation avR	No	Yes
Retrograde P waves on ST segment or beginning of T wave	No	Yes
QRS alterans	No	Yes
Retrograde activation on electrophysiological testing	Atria and ventricles activated simultaneously, retrograde atrial activation starts in low right atrium in typical AVNRT and in proximal coronary sinus in atypical AVNRT	Eccentric atrial activation, starts most commonly in left sided as left lateral pathway is most common.

Table 6: *Differentiating features of AVNRT and AVRT.*

WPW Syndrome

The incidence is 0.1 - 3/1000 persons and seen in all age groups. The left lateral accessory pathways are the most common pathways (left lateral > posteroseptal > anteroseptal). The congenital heart diseases associated with accessory pathways are ebstein anomaly and MVP. Approximately 80% of these patients with tachycardia have a reciprocating tachycardia, 15% to 30% have atrial fibrillation, and 5% have atrial flutter. VT occurs uncommonly. If patient is having atrial fibrillations with accessory pathway, avoid giving AV blocking agents like verapamil and digitalis and give agents that block both accessory pathway and AV node. As blocking of only AV node fastens the conduction through accessory pathway resulting in ventricular fibrillation and death [26].

Normal ECG of these type of patients show features of preexcitation and entity is known as WPW syndrome (Figure 16).

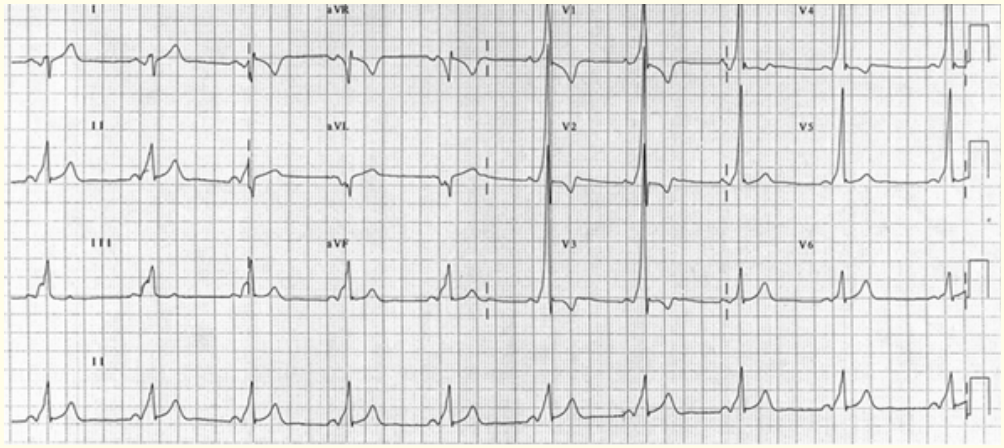


Figure 16: *Type A WPW syndrome, with short PR interval, broad QRS with slurred upstroke of QRS the delta wave and dominant R wave in lead V1 mimicking RVH suggestive of left sided accessory pathway.*

1. PR interval less than 120 milliseconds during sinus rhythm;
2. QRS complex duration exceeding 120 milliseconds with a slurred, slowly rising onset of the QRS in some leads (delta wave) and usually a normal terminal QRS portion; and
3. Secondary ST-T wave changes that are generally directed in an opposite direction to the major delta and QRS vectors.

Permanent junctional reciprocating tachycardia (PJRT)

Permanent junctional reciprocating tachycardia (PJRT) is a rare form of nearly incessant supraventricular tachycardia occurring predominantly in infants and children and characterized by a long RP interval and, in the typical form, by negative P-waves in leads II, III, and aVF on the surface ECG (Figure 17). During sinus rhythm, the surface ECG is normal, without manifest pre-excitation. PJRT is caused by an atrioventricular (AV) re-entry using the AV node as the ante- grade limb and a slowly conducting accessory pathway (AP) as the retrograde limb. The location of the AP is commonly right posteroseptal with an atrial insertion close to the ostium of the coronary sinus, but other locations have been reported. Chronic uncontrolled tachycardia has been reported to result in tachycardia-induced cardiomyopathy (TIC), which usually recovers with adequate ventricular rate control. As PJRT is most of the time refractory to drug therapy, radiofrequency catheter ablation of the AP has become the treatment of first choice [27].

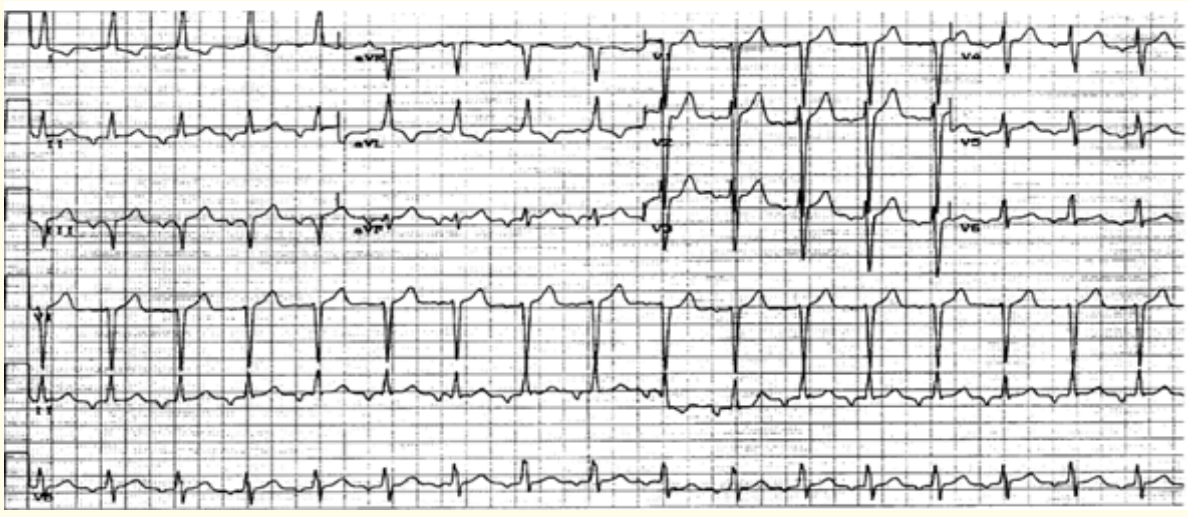


Figure 17: *ECG of PJRT showing a narrow QRS complex tachycardia at a rate of 110 b.p.m., with long RP interval, a negative P-wave in leads II, III, and aVF, and a positive P-wave in lead aVL.*

Conclusion

Supraventricular tachycardia requires careful interpretation of ECG, determining its underlying cause, mechanism and management depending upon the type of tachycardia. Every post graduate medical student must be familiar with different types of SVT, which would be helpful in taking decisions regarding the management of the patient.

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