Modern approach to the management of arrhythmias

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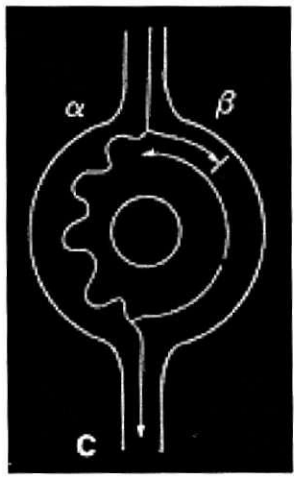
1. Supraventricular tachycardia (SVT)

Introduction

The era of catheter ablation for the treatment of arrhythmias began in 1981 when Dr. Schieman performed the first AV node ablation through direct current energy on a patient with resistant atrial fibrillation[1]. Although the AV node could be ablated with this method, severe baro trauma was a major problem and permanent pacing was also necessary. Therefore this was quickly abandoned. In 1985 trans catheter Ablation with RF energy was introduced by Stephen Huang[2]. Since then there has been a dramatic expansion of electrophysiology as a therapeutic field. Today RF ablation is the most widely used source of energy for selective ablations of different pathways with great success. More recently other sources of energy such as cryo have also been introduced.



α – antegrade pathway
β – retrograde pathway
c – final common pathway to ventricle

Figure 1. *Re-entry mechanism in AVJRT.*

Mechanisms of tachycardia

Three mechanisms have been identified[3].

1. Re-entry

Most of the tachycardias (70%) are due to re-entry. The re-entry loop consists of antegrade and retrograde pathways. One pathway is generally inactive whilst one is in sinus rhythm. Tachycardia initiates when this closed pathway opens up due to early extra beats and changing refractory periods. During tachycardia a loop forms, which continues to activate the atrium and the ventricle rapidly until it is interrupted by therapeutic manoeuvres or drugs.

ie: AVRT, AVJRT

(Figure 1)

2. Increased automaticity

Abnormal automaticity generates rapidly firing foci
ie: Atrial tachycardia.

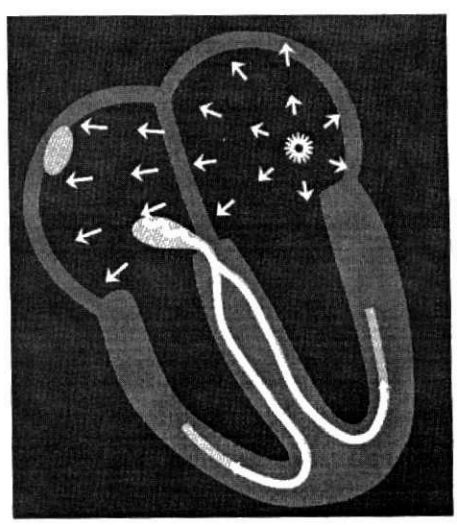


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Figure 2. *Rapidly firing foci.*

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3. Triggered activity

Rapid depolarization during the re-polarization phase can generate early (EAD) or delayed after depolarization (DAD). This can generate tachycardia.

Pacing manoeuvres and response to cardiac stimulants can differentiate the mechanisms during electrophysiological testing.

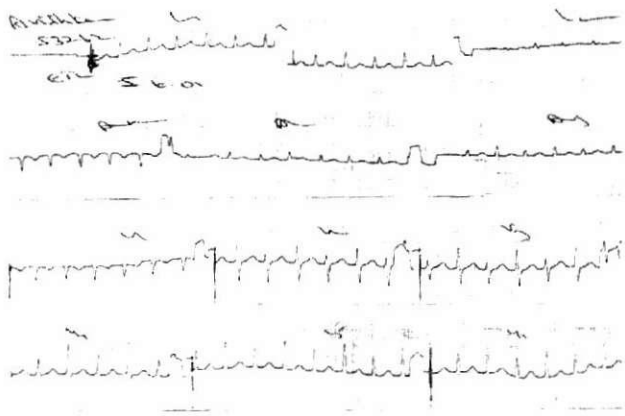
Types of SVT

Tachycardias originating in and above AV node are termed SVT.

- AV Junctional Re entry Tachycardia (AVJRT)
- Accessory Pathway Related Tachycardia
  - o Manifest – WPW
  - o Concealed – AV Re entrant Tachycardia (AVRT)
- Atrial Tachycardia
  - o Intra – Atrial Re entry Tachycardia (IART)
  - o Automatic Atrial Tachycardia
- Sinus Node related
  - o Inappropriate Sinus Tachycardia (IST)
- Atrial Flutter
  - o Typical – isthmus dependant
  - o Atypical – scar related
- Atrial Fibrillation (AF)

AV Junctional Re-entrant Tachycardia (AVJRT)

This is the commonest (50%) of regular SVT and predominantly seen between 20 – 60 years. The mechanism is a re-entrant circuit in the AV junction which consists of a slow conducting (alpha) pathway and a fast conducting (beta) pathway. Normal conduction is only through beta pathway and alpha pathway opens up only in tachycardia. Tachycardia rates are generally between 160 – 200 bpm. Clinically the patient presents with palpitations or syncope. ECG typically shows absent P wave tachycardia. However more commonly short RP tachycardia is seen.



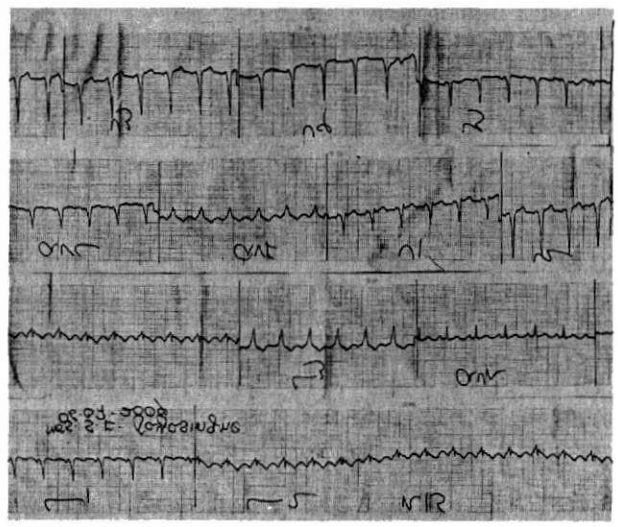
ECG 1 – Absent P wave tachycardia

Accessory pathway related tachycardia

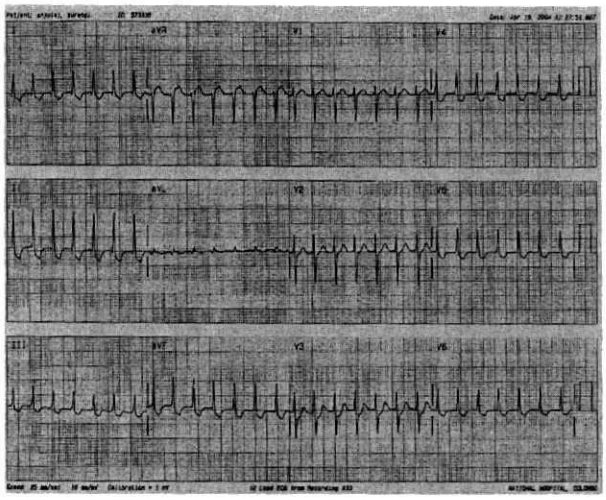
This is the second commonest (40%) cause of regular SVT and predominantly seen in older children and young adults. Accessory pathways are muscle strands connecting the atrium and the ventricle which traverse mitral or tricuspid valve. Anatomical sites of accessory pathways could be categorized as left (60%), septal (30%), and right (10%). Clinically, patients present with palpitations. Shortness of breath or chest pain can occur due to rapid heart rates. Syncope is unlikely.

ECG in sinus rhythm may show pre excitation which is called manifest pathway, commonly known as Wolf – Parkinson – White (WPW) syndrome[4]. The concealed accessory pathways present as tachycardia. The circuit is macro re-entry involving AV node, ventricular muscle and accessory pathway. ECGs during tachycardia show one of three types,

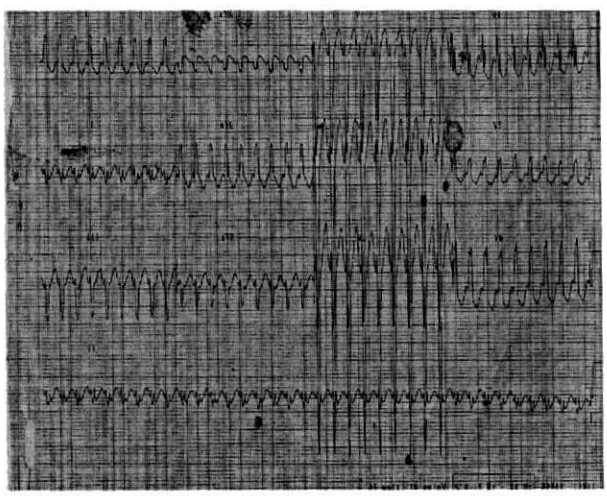
- Regular narrow complex SVT – Short RP tachycardia
- Pre excited wide complex SVT
- Pre excited atrial fibrillation



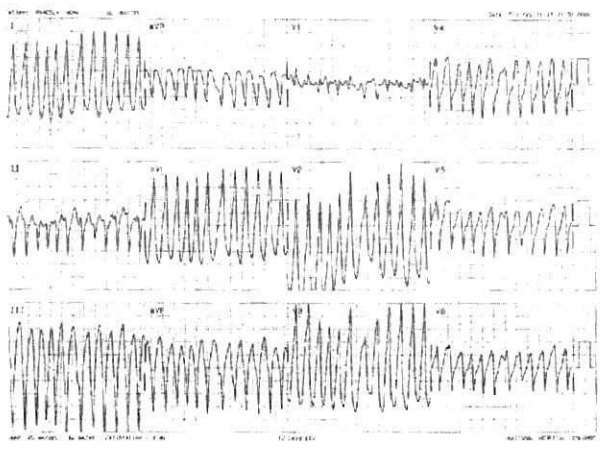
ECG 2 – WPW syndrome



ECG 3 – Short RP tachycardia



ECG 4 – Pre excited wide complex SVT

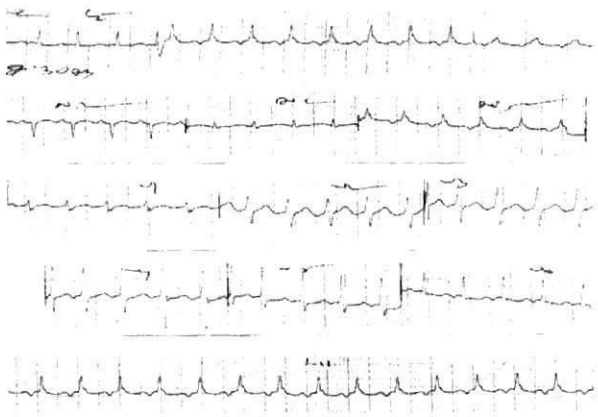


ECG 5 – Pre excited AF

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

Atrial tachycardia

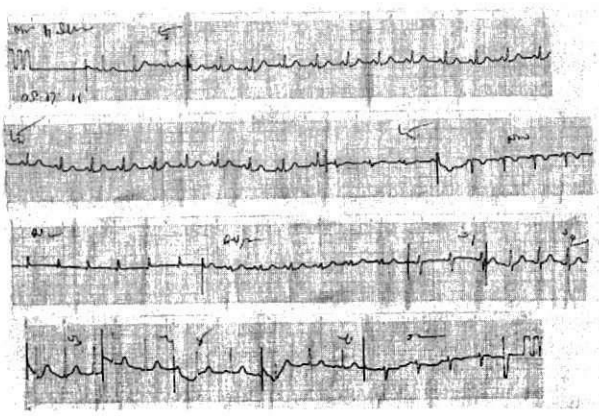
This is less common and (5-10%) predominantly seen in those under 10 and over 60 years of age. The mechanism could be automatic or re-entrant. Origin can be from different places in the atria. Common sites are crista terminalis, pulmonary veins, annulus of valves and triangle of Koch's. These clinically present with recurrent palpitations. On occasions patient present with incessant tachycardia. ECG during tachycardia typically shows long RP. The P wave could be upright or inverted depending on the focus.



ECG 6 – Long RP tachycardia

Inappropriate Sinus Tachycardia (IST)

This is a common variant specially seen in young females. Origin is within the SA node due to automaticity or re-entry. Resting heart rate is over 100 bpm with very frequent episodes of rates between 100-150 bpm. Clinical presentation could vary from accidental detection to disabling palpitations. ECG during tachycardia shows similar features to sinus tachycardia, P followed by QRS. P wave morphology during tachycardia is identical to that of sinus rhythm.

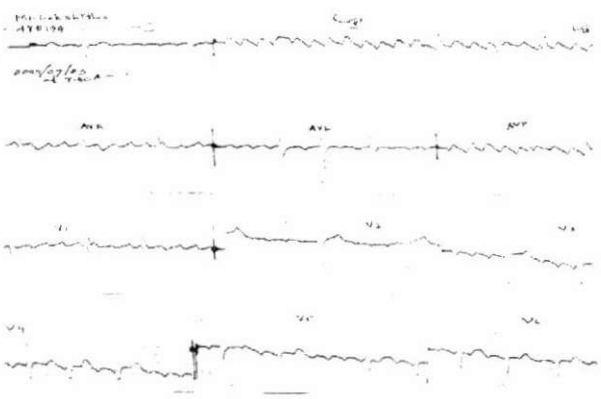


ECG 7 – Inappropriate sinus tachycardia

Atrial flutter

This is not a very common tachycardia. The mechanism is due to macro re-entrant circuit. It could be categorized as typical or atypical. Typical atrial flutter is due to a macro re-entrant loop consisting of medial wall (crista terminalis), lateral walls of RA, and isthmus between tricuspid valve and IVC (cavo-tricuspid isthmus). The direction could be clockwise or anticlockwise. Atypical flutters are scar related or incisional and are more like atrial tachycardias. Clinical presentation varies from asymptomatic to very rapid tachycardia.

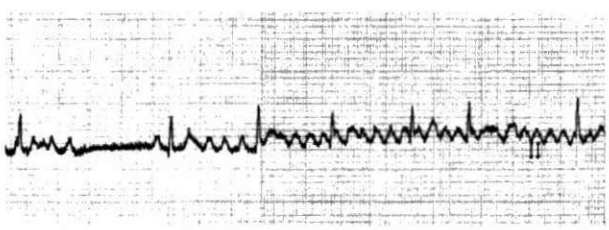
ECG during typical anticlockwise atrial flutter shows, negative flutter waves in inferior leads whereas positive flutter waves are seen in clockwise flutter. Ventricular rate during tachycardia is commonly 100 bpm to 150 bpm but occasionally it can go up to 300 bpm.



ECG 8 – Typical atrial flutter

Atrial Fibrillation

This is the commonest arrhythmia. It has a complex aetiology leading to electrical instability of the left atrium. Rapidly firing foci from pulmonary veins have been identified as the primary cause. AF can be categorized as paroxysmal or persistent. Paroxysmal group switches in and out without the assistance of drugs. The persistant group needs drugs or cardio-versions to get a similar effect. Clinically presentations are palpitations, syncope, or embolic strokes.



ECG 9 – Sustained AF due to focal pulmonary vein firing

Management of SVT

This can be divided into acute and long term management.

Acute management

This generally happens in an ICU setting. Here focus is on immediate termination of tachycardia. In haemodynamically stable patients this is achieved by intravenous injection of AV nodal blocking drugs such as adenosine, or verapamil. Adenosine is considered better than verapamil in faster heart rates over 175 bpm. In certain situations (ie: fast AF) other agents such as flecanide, or amiadarone could be used. In haemodynamically unstable patients electrical cardioversions with 50 -100 J should be done.

Long term management

With introduction of Radio Frequency Ablation (RFA) focus was shifted dramatically from control to cure. RFA is a high frequency (300-750 kHz) continuous alternating current. At the electrode-tissue surface current is converted to heat, causing thermal injury. Heat penetrates to deeper tissues by acceleration of ions (resistive heating), thereby breaking the circuit to effect a cure. Discrete lesions are produced to interfere with the circuit when temperature is 60° C for 40 seconds.

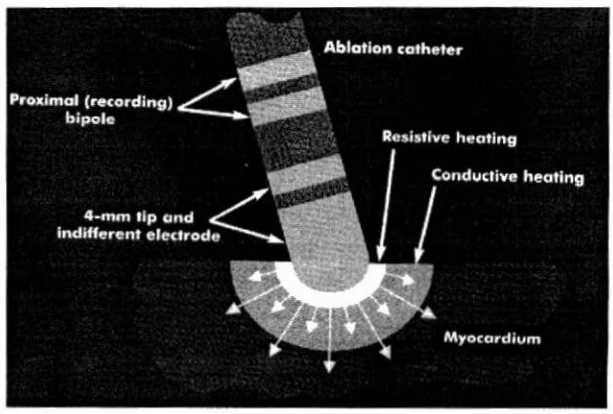


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Figure 3. *Effect of RF ablation – indications for RF ablation in SVT,*

Indications for RF ablation in SVT

- Recurrent supraventricular tachycardia (≥ 2 attacks, drug resistant)
- Symptomatic WPW syndrome
- Focal atrial tachycardia
- Recurrent Atrial Flutter
- Drug resistant (more than 2 drugs) atrial fibrillation

Effects of RF ablation on sub groups of SVT

AVJRT

The strategy is to ablate slow pathway leaving the fast pathway intact. Ablation success for cure is 90-95%[6]. This is equally effective in elderly[7]. Risk of heart block needing permanent pacing in 1-3%.

Accessory pathway related tachycardia

The strategy is to ablate the accessory pathway. This can be achieved by several methods. In WPW syndrome, ablation can be performed in sinus rhythm looking for early electrical signals in the ablation catheter. In concealed accessory pathways, this is achieved by ablation during tachycardia or retrograde pacing. RF ablation is 90-95% effective in obtaining a cure[8]. Left sided pathways have the highest success rate. Risks are few and these depend on the location and access.

Asymptomatic WPW is only ablated if tachycardia is induced or if found to have antegrade refractory period less than 250msec[9].

Atrial tachycardia

The strategy is to ablate the responsible focus / foci. Success depends on the location and is generally around 60-70%. Certain variations such as crystal atrial tachycardia, and post-operative atrial tachycardia need 3D mapping systems to improve the success of ablation.

Inappropriate sinus tachycardia

The strategy is to identify and ablate focus / foci in and around the sinus node (sinus node modification). Results are not promising as immediate success is generally followed by early recurrence. Particular precautions should be taken not to damage the phrenic nerve in the vicinity of sinus node.

Atrial flutter

Atypical atrial flutter (isthmus dependent) the ablation strategy is to interfere with the circuit at its narrowest point. This is in the area between IVC and tricuspid valve septal leaflet (cavo-tricuspid isthmus). Success rate for a cure is 80-90%[10].

Atypical atrial flutters are more like atrial tachycardias. They will need, mapping with a 3D system for successful ablations[11].

Atrial fibrillation

Ablation strategy depends on two goals.

Rate control – This is achieved by complete AV node ablation, and a permanent pacemaker in the ventricle. This gives a regular ventricular rhythm and generally performed either in older patients or patients who are already on pacemakers for sick sinus syndrome.

Rhythm control – This is based on an ablation strategy, which isolates the pulmonary veins (PVI), or which involves left atrial circumferential ablation (LACA). Recently more combined strategy has also been described[12]. The optimum method is yet to be achieved. The degree of success depends on the stage of atrial fibrillation. It is highly successful in paroxysmal atrial fibrillation (70-85%) and less successful in persistent AF (40-80%). While higher percentage of success was reported by pioneering centres, other centres struggle to keep up with them. 3D mapping systems are an integral part of these complex procedures.

Complictions RF ablations

RF ablations are comparatively safe procedures. However, major and minor complications can occur.

Major complications

Death – approximately 1: 1000. This is due to thrombosis, thromboembolic phenomena or hazards such as pericardial tamponade.

Heart block needing permanent pacing – There is 2-3% risk depending on the type of circuit subjected to ablation. Highest risks are in slow pathway ablation for AVJRT and accessory pathway location in right antero-septal region.

Strokes – related to left sided ablations particularly in atrial fibrillation patients.

Pericardial tamponade – Related to extensive ablations, and trans-septal procedures.

Minor complications

Puncture site haematomas and local wound infections can occur. Prophylactic antibiotics are routinely given per operatively to prevent systemic infections.

Conclusions

Management of supra ventricular tachycardia is now focused on cure. RF ablation provides highly successful cure rates with low risks, particularly for common sub types, such as AVJRT and AP related. In complex cases, efficiency can be increased with 3D systems.

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2. Ventricular tachycardia (VT)

Ventricular tachycardias have special importance as they

- are potentially serious
- are likely to be life threatening
- always warrant investigation

Classification

Normal heart VT

This is due to primary electrical disease in the background of structurally normal heart.

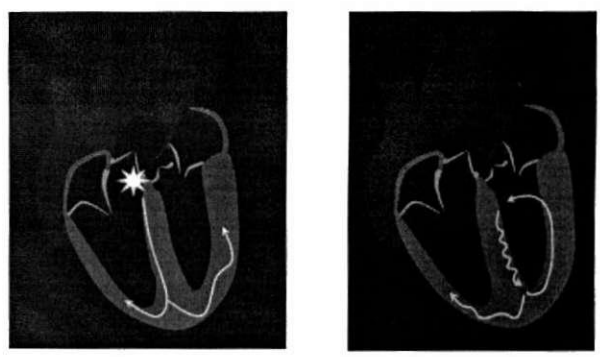
- Outflow Tract VT – origin is from right ventricle (RVOT) or left ventricle (LVOT)
- Idiopathic Left Ventricular VT (ILVT)
- Congenital chanellopathies –
  Brugada syndrome
  Congenital Long QT syndrome
  Short QT syndrome

Abnormal heart VT

This is secondary to structural heart disease.

- Scar Related VTs – post infarct or cardiomyopathy.
- Arrhythmogenic right ventricular dysplasia (ARVD)
- Infiltrative disorders – Sarcoid and Amyloid

Normal heart VTs



C/O – Handbook of cardiac electrophysiology by Murgatroyd FD, Krahn AD, *et al*:

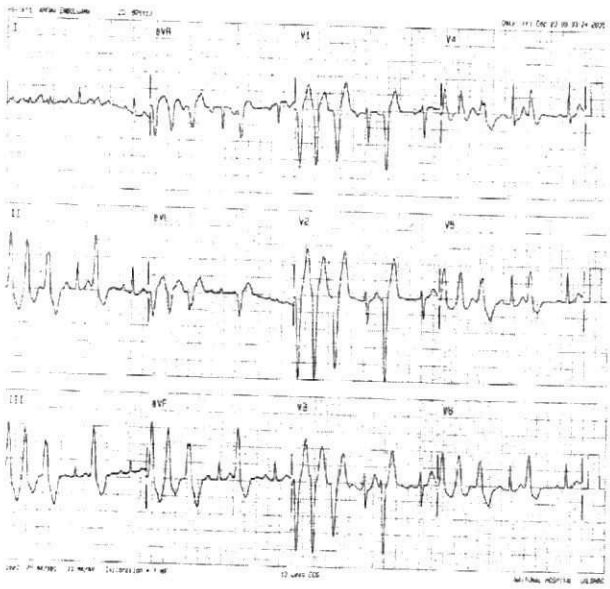
1A **1B**

Diagram shows circuit responsible RVOT (1A) and ILVT (1B)

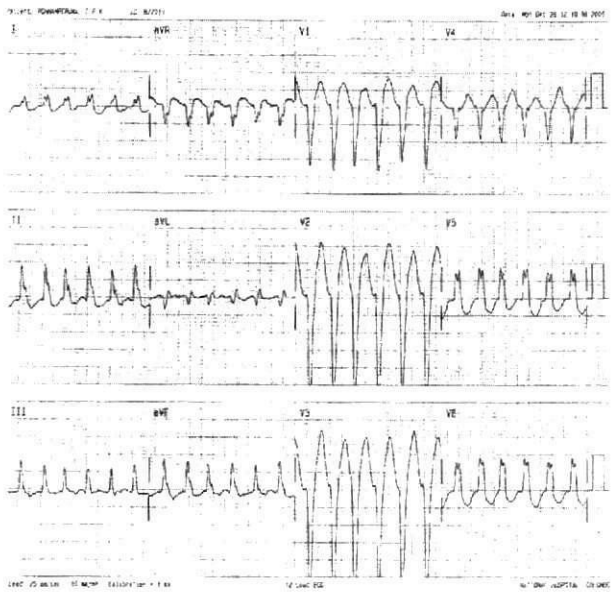
RV outflow tract VT

This is a common arrhythmia in the young. It presents with wide variation of symptoms, from palpitations, chest pain to syncope[1]. Some are accidentally detected on routine ECGs. Most patients have structurally normal hearts but a minority has arrhythmogenic dysplasia.

The mechanism of tachycardia is considered to be triggered activity.



1A



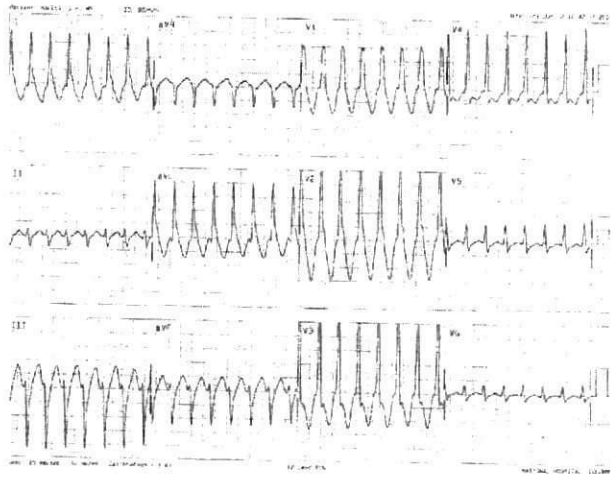
1B

ECG 1 – RVOT ectopy (1A) & RVOT – VT (1B)

Minority has a variant of LVOT – VT originating from aortic root.

Idiopathic left ventricular tachycardia (ILVT)

Other names given are fascicular VT and verapamil sensitive VT. The supposed mechanism is now considered as re-entry in the LV septum. Clinically patients present with palpitations to syncope.



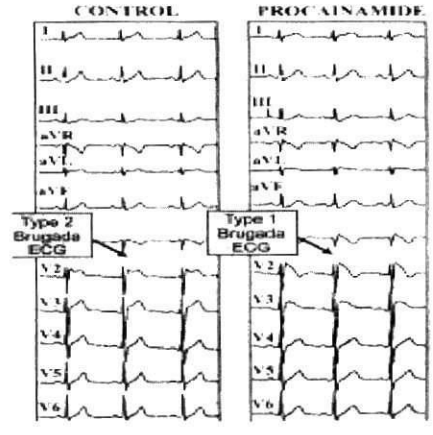
ECG 2 – ILVT

Congenital chanellopathies

Primary electrical diseases such as Brugada, long QT and short QT syndromes present with sudden onset VT or VF. The first attack maybe fatal.

Brugada syndrome

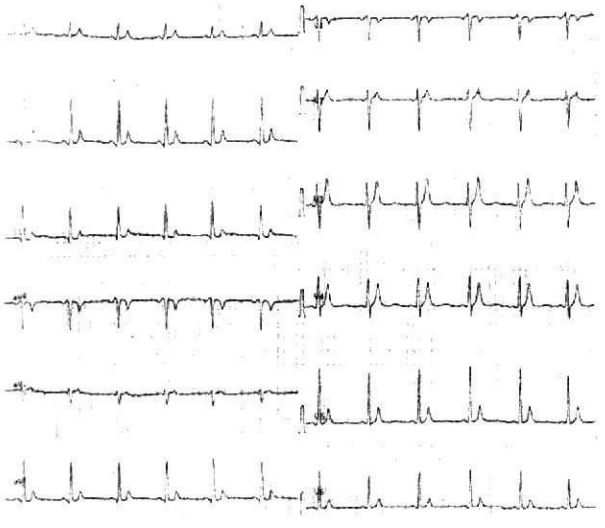
The syndrome was first identified in 1992 by Joseph and Pedro Brugada[2]. This is a sodium (Na) chanello-pathy. Several mutations have been described. The net functional effect is loss of function of the sodium channel. The syndrome is responsible for 20% of sudden cardiac deaths in structurally normal heart, and 4% of all sudden deaths. Cause of death is sudden onset ventricular fibrillation (VF). It predominantly presents at age around 40. Typically, patients have coved type ST elevation ECG (type 1), which itself is diagnostic and the other patients have saddle back type ST changes (type 2) in V1 – V3 leads. The latter group needs procainamide, flecainide or ajmaline challenge test to uncover the changes[3]. The ECG changes are known to be dynamic so that coved type ECG on one day may appear as normal the next day[4].



ECG 3 – Brugada syndrome

Short QT syndrome

This entity has been recently identified as a cause of sudden cardiac death due to VT and VF. The defect is a mutation leading to gain of function in Ikr channel. The main feature is a QT interval at rest of < 320 msec. QRS appears to be merge into T wave, and there is a lack of QT dependence on the heart rate[5].



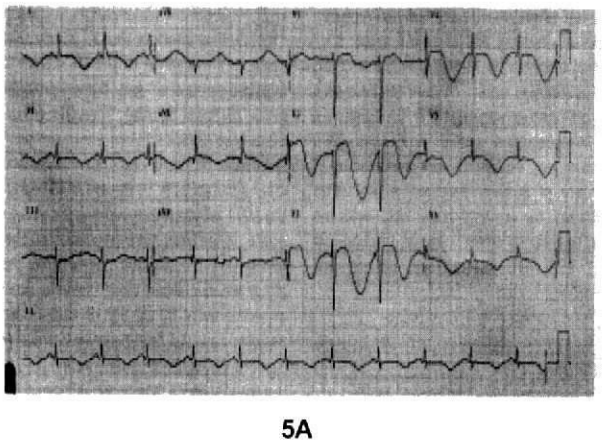
Baseline ECG

Baseline ECG - QT / QTc, 240 ms / 268 ms

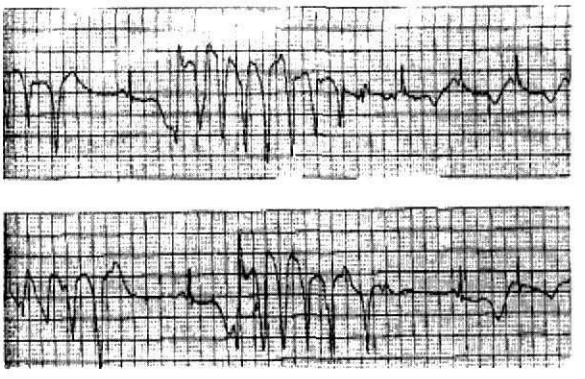
ECG 4 – Short QT syndrome

Long QT syndrome

It is diagnosed when the QTc ($QT/_{RR}$) is more than 0.44 seconds. There are many sub types, but the major ones are LQT1, LQT2 & LQT3.[6] First two types are potassium channellopathies, while LQT3 is a sodium channellopathy. Arrhythmia is typically polymorphic VT, leading to VF & death. The circumstances of arrhythmia differ. LQT 1 generally occurs during exercise, LQT 2 during emotions & LQT 3 during sleep.



5A



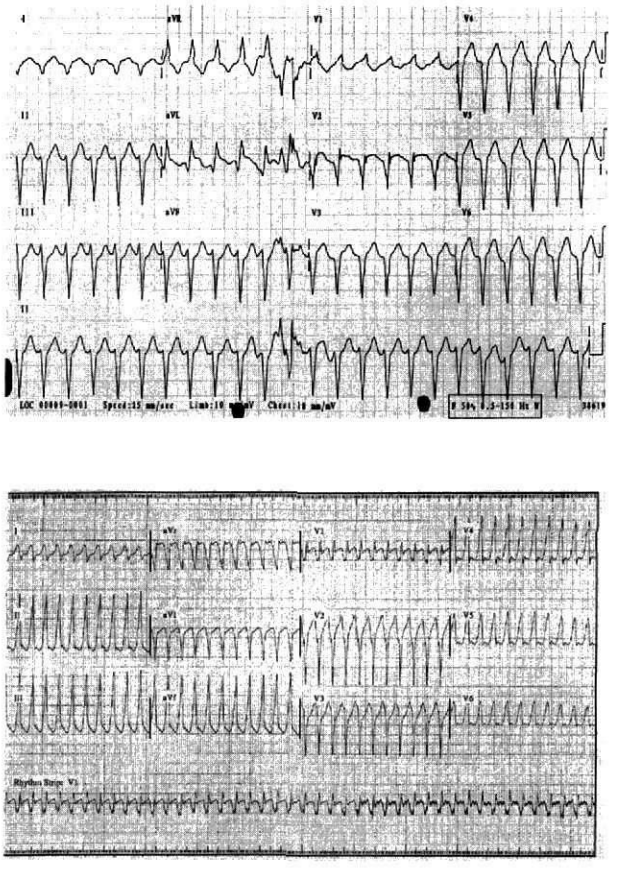
5B

ECG 5 – LQT 1 (A) & polymorphic VT (B)

Abnormal heart VTs

Scar related VTs

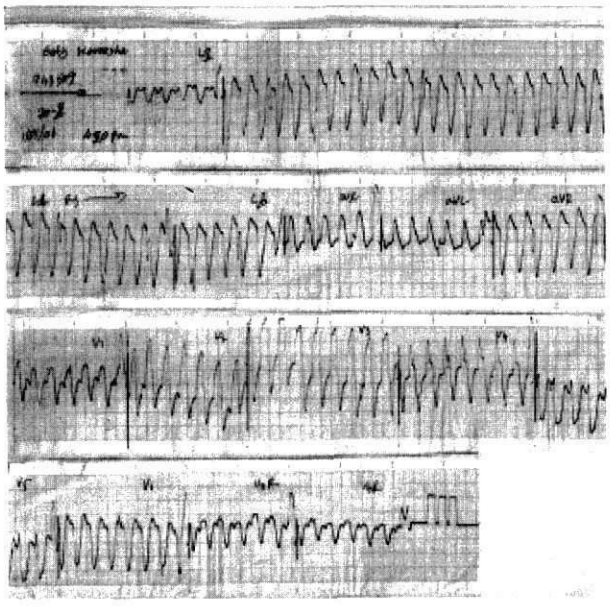
This is due to previous myocardial infarctions or cardiomyopathy, leading to a chronic scar, which acts as a re-entrant circuit. Ischaemic VTs have a high mortality. This can lead to life threatening VTs. One patient may have more than one morphological type of VTs.



ECG 6 – Two morphologies of ischaemic VT in the same patient

Arrhythmogenic right ventricular dysplasia (ARVD)

This is a rare disorder causing fibro fatty degeneration of the RV myocardium. The degenerated sites act as the myocardial scar leading to VT. In early stages it can be diagnosed by cardiac MRI imaging. Severe forms can be identified through echocardiography.



ECG 7 – ARVD – VT (LBBB with superior axis)

Management of VT

Acute Management

The focus is on terminating the tachycardia. These patients commonly need DC cardioversions (50 - 300 J) as they are haemodynamically unstable. In more stable patients, drugs such as, lignocaine, mexiletine, or amiodarone can be given intravenously for medical cardiversion.

In ILVT, verapamil is used for termination of tachycardia.

Long term management

Normal heart VT

RF ablation is the treatment of choice. This gives a realistic chance of cure. Idiopathic RVOT & ILVT have success rates close to 80-85%[7,8]. Ablation with a conventional mapping system may be adequate for most patients. Exceptionally difficult circuits need additional help from 3D mapping.

Congenital chanellopathies need implantable cardioverter defibrillators (ICDs) to prevent sudden cardiac death. However certain drugs such as quinidine in Brugada and short QT syndrome and beta blockers in long QT syndrome have shown to be reasonably effective.

Abnormal heart VT

As many circuits are chronic and complex, ICDs are the treatment of choice. ICDs have clearly been shown to save lives by primary & secondary prevention in different groups of patients.

Indications for ICD are,

- Sustained ventricular tachycardia or ventricular fibrillation
  - o non-recurring or recurring, inducible by EPS
  - o non-inducible by EPS, but recurring
- Patients with primary VF and consecutive cardiac arrest.
- Cardiomyopathy or previous myocardial infarction LVEF ≤ 40%.

Further if these patients have poor LV function (LVEF ≤ 30%) with evidence of electrical or echo dis-synchrony cardiac resynchronization therapy and defibrillator (CRT- D) is the ideal treatment.

Several trials have clearly shown the mortality benefit of ICD. The initial landmark trial was the MADIT – II trial. This was followed by COMPANION and CARE – HF trials. Latter trials also included CRT devices (Bi-Ventricular pacing) for heart failure[9].

Place of RF ablation in abnormal heart VT

There were several studies in highly skilled centres showing RF ablations alone as a method of treatment has success rates up to 75-80% for further prevention of VTs[10]. However realistically RF ablation is used to reduce the arrhythmic burden in the background of already implanted ICDs.

Conclusions

- In normal heart VTs, RF ablation should be considered as first choice.
- In, abnormal heart VTs, and congenital channellopathies, the use of ICDs is the management of choice.
- Use of RF ablation in abnormal heart VT is presently recommended to reduce the tachycardia burden.

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MCQs

Choose the most appropriate answer.

1. Which of the following is correct regarding RF ablation?

A It is the first line treatment for recurrent post MI VT.
B It is mandatory in exercise induced WPW syndrome.
C It is successful for inappropriate sinus tachycardia.
D It is the treatment of choice for a 60-year-old male with the first attack of SVT.
E It has a lower risk profile in LV outflow tract than in RV outflow tract VT.

2. Which of the following is true for recurrent ventricular tachycardia 50 days following an antero septal myocardial infarction?

A It should be treated with long term amiodarone.
B Patient should be subjected to a coronary angiogram.
C Patient should be subjected to ventricular stimulation test to evaluate the inducibility of VT.
D Patient should be subjected to RF ablation as first line treatment.
E ICD implantation is the best method of treatment at present.

3. Which of the following is true?

A Most tachycardias are a result of triggered activity.
B Multifocal atrial tachycardia is seen in young healthy individuals.
C Long term amiodarone is suitable for Brugada syndrome.
D ECG changes in Brugada syndrome are dynamic (could vary in different ECGs).
E Long QT syndrome cannot be drug induced.

4. Which of the following is true of AF?

A AF is mortly due to triggers within left atrium.
B If noticed in young, underlying WPW should be considered.
C Best rate control is achieved by using AV nodal blocking drugs.
D Following the first attack patient should be subjected to RF ablation.
E Anti platelets are the best form of stroke prevention if the age is over 75 years.