UNIT 7 ARRHYTHMIA

Structure

7.0 OBJECTIVES

After reading this unit, you should be able to:

- enlist the causes as a diagnostic criteria of premature beats and atrial flutter and atrial fibrillation;
- describe the mechanism of supraventricular tachycardia;
- know the causes and ECG change in ventricular tachycardia and fibrillation; and
- describe the various degree of heart block and their ECG changes.

7.1 INTRODUCTION

Arrhythmias (or dysrhythmias) are problems that affect the electrical system of the heart muscle, producing abnormal heart rhythms. They can cause the heart to pump less effectively. ECG is the best tool for diagnosis of arrhyhmias. Arrhythmias may be seen on 12-lead ECGs or on strips of one or more leads. Some arrhythmias are obvious at first glance and don't require intense analysis; others require systematic analysis of surface ECG followed invasive evaluation in the form of eletrophysiologic study for diagnosis.

Many arrhythmias have no known underlying cause. However, a number of factors can contribute to arrhythmias. They include coronary artery disease, high blood pressure, diabetes, smoking, excessive use of alcohol, drug abuse and stress. Certain substances, including some over-the-counter and prescription medications, dietary supplements and herbal remedies are known to cause arrhythmias in some people.

Bradyarrhythmia refers to any rhythm with rate less than 60 beats/minute. Tachyarrhythmia means any rhythm with rate more than 100 beats/minute. Two important issues that are basic to the understanding the rhythm disturbances are:

- Mechanism of the rhythm abnormality
- Site of origin of the arrhythmia

There are broadly two major mechanisms of that produce arrhythmias:

- Disorders of impulse of formation (automaticity) and
- Disorders of impulse conduction (block or re-entry)

7.2 PREMATURE BEATS

Pacemaker stimuli can also arise from other parts of the heart – the atria, the AV junction, or the ventricles. The terms "**ectopy**" or "**ectopic beat**" are used to describe these non-sinus beats. Ectopic beats are often premature; i.e. they come in early or before the next sinus beat is due.

The premature beat by itself does not cause any symptom; the next normal beat following the premature may cause palpitation. The consequences of a premature beat are:

1) PB occurs earlier than expected beat, hence cannot pump a significant amount of blood.
2) PB prevents the occurrence of subsequent normal beat; and
3) The PB is usually followed by a pause.

There are two types of premature beats:

1) Supraventricular
2) Ventricular

The supraventricular premature beats originate in the atria or the atrioventricular junction and hence produce a narrow QRS complex unless they are conducted aberrantly in which case, there is QRS widening. Ventricular prematures originate beyond the brancing of the conduction tissue and hence prodce an abnormally prolonged QRS complex.

Atrial Premature Beats APB (Premature Atrial Complexes)

Description

A premature atrial contraction results from an ectopic stimulus that arises from somewhere in either the left or the right atrium, but not in the sinus node. The atria are depolarized from the ectopic stimulus, but the remainder of the conduction is typically normal through the AV Node-Junction and downward into the bundle branches (i.e. normal PR and QRS morphology and intervals). APBs occur as single or repetitive events and have unifocal or multifocal origins.

Possible Causes

APBs are very common and may occur in persons with a normal heart or in persons with virtually any type of organic heart disease. APBs do not imply that a person has cardiac disease and may be seen with caffeine intake and with emotional stress. Other causes include:

- Administration of sympathomimetic agents (epinephrine, theophylline)
- Electrolyte abnormalities
- Myocardial ischaemia or injury
- Digoxin toxicity
- Hyperthyroidism

ECG Criteria

1) Heart Rate: Typically normal.
2) Rhythm: Underlying rhythm is typically regular with early premature beats.
3) P-waves: Atrial depolarization is premature, occurring before the next normal P-wave: Since the impulse originates outside the SA node, the P-wave may have a different shape-often notched, peaked or buried in the proceeding T-wave.
4) PR Interval: Maybe normal, shorter or longer than normal PR interval, depending on origin of the APB.
5) QRS Width: Typically normal but may be prolonged if the PAC is aberrantly conducted through the ventricles.

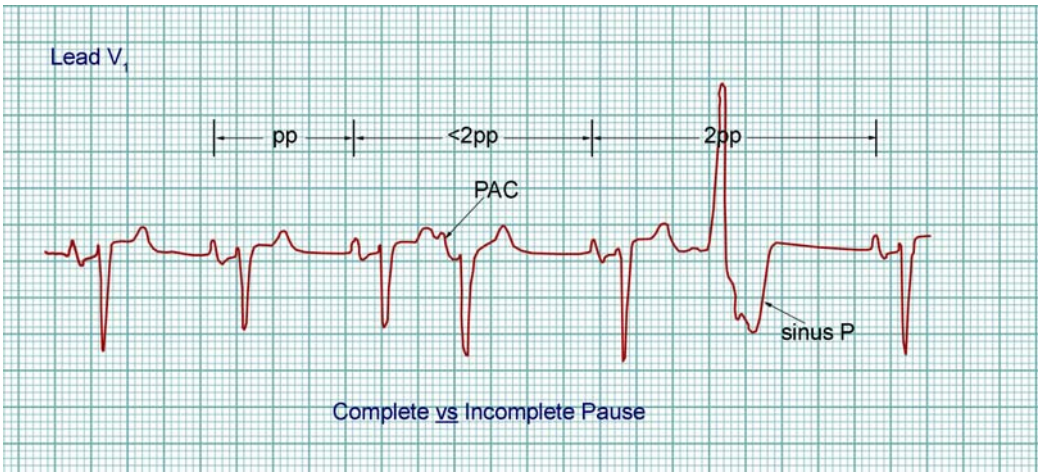


Fig. 7.1: Complete VS Incomplete Pause

APBs can have three different outcomes depending on the degree of prematurity (i.e., coupling interval from previous P-wave), and the preceding cycle length.

1) *Nonconducted (blocked)*, i.e., no QRS complex because the APB finds AV node still refractory. This manifests on surface ECG as a pause. The most common cause of an unexplained pause on ECG is non-conducted APB,

2) *Conducted with aberration,* i.e., APB makes it into the ventricles but finds one or more of the conducting fascicles or bundle branches refractory. The resulting QRS is usually wide, and is sometimes called an Ashman beat,

3) *Normal conduction,* i.e., similar to other QRS complexes in the ECG.

The fate of a PAC depends on:

1) coupling interval from the last P-wave and

2) preceding cycle length or heart rate.

The pause after a APB is usually incomplete; i.e., the APB usually enters the sinus node and resets its timing, causing the next sinus P to appear earlier than expected. (PVCs, on the other hand, are usually followed by a complete pause because the PVC does not usually perturb the sinus node).

Premature Junctional Complexes (PJC)

Similar to APBs in clinical implications, but occur less frequently.

The PJC focus, located in the AV junction, captures the atria (retrograde) and the ventricles (antegrade). The retrograde P-wave may appear before, during, or after the QRS complex; if before, the PR interval is usually short (i.e., <0.12s).

Ventricular Premature Beats or Premature Ventricular Complexes (VPBs)

VPBs may be *unifocal, multifocal* or *multiformed.* Multifocal VPBs have different sites of origin, which means their coupling intervals (measured from the previous QRS complexes) are usually different. Multiformed VPBs usually have the same coupling intervals (because they originate in the same ectopic site but their conduction through the ventricles differ. Multiformed VPBs are common in digitalis intoxication.

VPBs may occur as isolated single events or as couplets, triplets, and salvos (4-6 VPBs in a row), also called brief ventricular tachycardias. VPBs may occur early in the cycle (R-on-T phenomenon), after the T-wave (as seen above), or late in the cycle—often fusing with the next QRS (fusion beat). R-on-T VPBs may be especially dangerous in an acute ischaemic situation, because the ventricles may be more vulnerable to ventricular tachycardia or fibrillation.

For fusion to occur the sinus P-wave must have made it to the ventricles to start the activation sequence, but before ventricular activation is completed the “late” PVC occurs. The resultant QRS looks a bit like the normal QRS, and a bit like the PVC; i.e., a fusion QRS. The events following a VPB are of interest. Usually a VPB is followed by a complete compensatory pause because the sinus node timing is not interrupted; one sinus P-wave isn’t able to reach the ventricles because they are still refractory from the VPB; the following sinus impulse occurs on time based on the sinus rate. In contrast, APBs are usually followed by an incomplete pause because the APB usually enters the sinus node and resets its timing; this enables the following sinus P-wave to appear earlier than expected. Not all VPBs are followed by a pause. If a VPB occurs early enough (especially if the heart rate is slow), it may appear sandwiched in between two normal beats. This is called an interpolated VPB. The sinus impulse following the VPB may be conducted with a longer PR interval because of retrograde concealed conduction by the VPB into the AV junction slowing subsequent conduction of the sinus impulse. Finally a VPB may retrogradely capture the atrium, reset the sinus node, and be followed by an incomplete pause. Often the retrograde P-wave can be seen on the ECG, hiding in the ST-T-wave of the PVC.

The most unusual post-PVC event is when retrograde activation of the AV junction re-enters the ventricles as a ventricular echo.

VPBs usually stick out like “sore thumbs”, because they are bizarre in appearance compared to the normal complexes. However, not all premature sore thumbs are PVCs.

Check Your Progress 1

1) What is meant by the terms bradyarrhythmia and tachyarrhythmia?

.....

.....

2) What are the 2 main mechanisms of production of arrhythmias?

.....

.....

7.3 ACCELERATED AUTOMATICITY

As the name suggests this arrhythmia results from accelerated automaticity in the cells of pacemaker and conduction system. Arrhythmias included in the this category are:

1) Sinus tachycardia
2) Atrial tachycardia
3) Accelerated junctional rhythm
4) Accelerated ventricular rhythm

Sinus Tachycardia

Sinus tachycardia is characterized by a rapid (> 100 bpm) rate of discharge of the SA node. The sinus node is discharging at a rate > 100 and the remainder of the conduction follows the normal pathway. The following ECG shows sinus tachycardia.

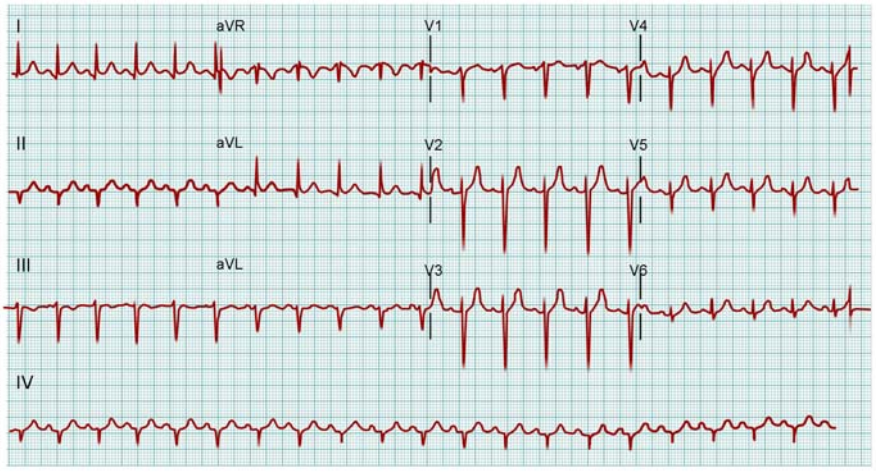


Fig. 7.2: Sinus Tachycardia

Possible Causes

- Normal cardiac response to demands for increased oxygen need during pain, fever, stress, dehydration and exercise
- Caffeine, nicotine ingestion
- Hyperthyroidism
- Post MI or early sign of heart failure

ECG Criteria

1) Heart Rate: ≥ 100 bpm to 160 bpm,in children can go upto 220 bpm
2) Rhythm: Regular
3) P-waves: Upright and normal. One P precedes every QRS
4) PR Interval: .08 -.20 seconds
5) QRS Width: ≤ 0.12 seconds

Junctional Tachycardia

Junctional rhythm greater than 100 bpm is called **Junctional Tachycardia**. The SA node is not working and the junction has taken over as the pacer, only a bit faster than its normal intrinsic rate of 40-60 bpm.

Possible Causes of Accelerated Junctional Rhythm

- Digoxin toxicity (most common cause)
- Hypoxia
- Cardiomyopathy
- MI
- Valve replacement surgery

ECG Criteria of Accelerated Junctional Rhythm

1) Heart Rate: > 100 bpm.
2) Rhythm: Ventricular rhythm is regular.
3) P-waves: may be absent or may occur before, during or after the QRS (due to retrograde conduction).
4) PR Interval: None (impulses are originating from the junction, not the SA node).
5) QRS Width: ≤ 0.12 seconds (the impulse is traveling down the normal pathways of the right and left bundles.

The following picture shows accelerated junctional rhythm.

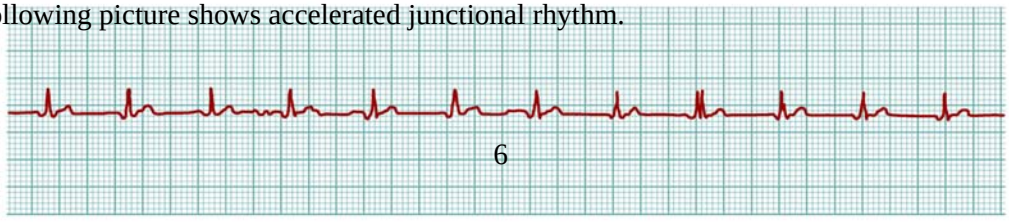


Fig. 7.3: Accelerated Junctional Rhythm

Paroxysmal Atrial Tachycardia

Ectopic, discrete looking, unifocal P'-waves with atrial rate <250/minute (not to be confused with slow atrial flutter). Ectopic P'-waves usually precede QRS complexes with P'R interval < RP' interval (i.e., not to be confused with paroxysmal supraventricular tachycardia with retrograde P-waves appearing shortly after the QRS complexes). Ventricular response may be 1:1 or with varying degrees of AV block (especially in digitalis toxicity).

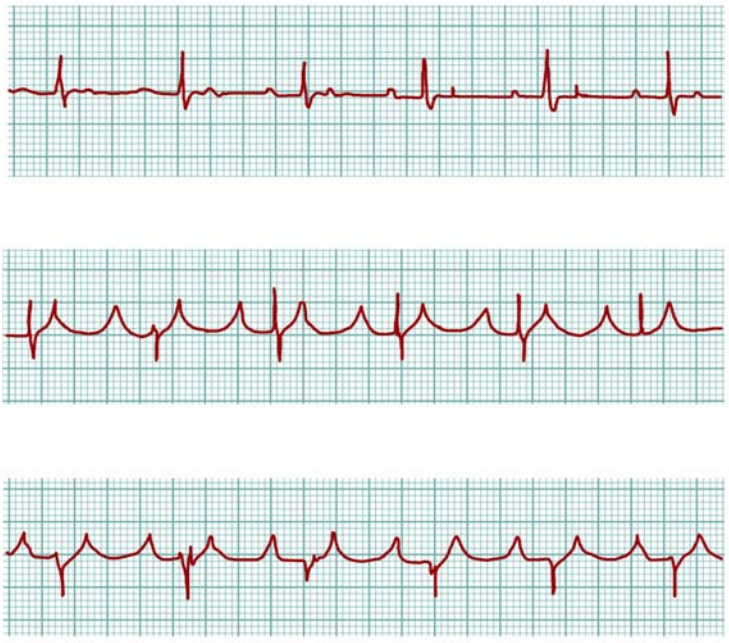


Fig. 7.4: Paroxysmal Atrial Tachycardia

Multifocal Atrial Tachycardia (MAT) and Rhythm

Discrete, multifocal P'-waves occurring at rates of 100-250/min and with varying P'R intervals (should see at least 3 different P-wave morphologies in a given lead). Ventricular response is irregularly irregular (i.e., often confused with A-fib). May be intermittent, alternating with periods of normal sinus rhythm. Seen most often in elderly patients with chronic or acute medical problems such as exacerbation of chronic obstructive pulmonary disease.

Accelerated Ventricular Rhythms

An "active" ventricular rhythm due to enhanced automaticity of a ventricular pacemaker (reperfusion after thrombolytic therapy is a common causal factor). Ventricular rate 60-100 bpm (anything faster would be ventricular tachycardia). Sometimes called ***isochronic ventricular rhythm*** because the ventricular rate is close to underlying sinus rate. May begin and end with

fusion beats (ventricular activation partly due to the normal sinus activation of the ventricles and partly from the ectopic focus). It is usually benign, short lasting, and not requiring any treatment.

Check Your Progress 2

What are the ECG features of multifocal atrial tachycardia?

.....

.....

7.4 ATRIAL FLUTTER AND ARIAL FIBRILLATION

Atrial Flutter

Atrial Flutter is a dysrhythmia, which is the result of a flawed reentry circuit within the atria. It is often described as resembling a sawtooth or picket fence. These **flutter waves** should not be confused for P-waves. The AV node is a wonderful protective mechanism. Imagine the atria depolarizing at a rate of 250 to 350 bpm. If all of these atrial depolarizations were conducted down into the ventricle, the patient's ventricles would likely begin to fibrillate. Think of the AV node as the central train station where numerous train tracks merge. The central station only lets some of the trains through to avoid congestion. The AV node helps to protect the ventricles by only allowing some of the atrial depolarizations to conduct down through the bundle of His into the bundle branches and on to the ventricles. When the ventricular rate is < 100 bpm, we call this "controlled atrial flutter". If the ventricular rate is > 100 bpm, it is labeled "uncontrolled atrial flutter". Since the ventricles always have more time to fill during diastole when the HR is <100, our goal is to have controlled atrial flutter. This can often be accomplished with drug therapy. In the setting of atrial flutter, coordinated contraction of the atria is absent. The patient has therefore, lost their atrial kick with potential loss of cardiac output and lower blood pressure.

Causes

- Acute or chronic cardiac disorder, mitral or tricuspid valve disorder, cor pulmonale, pericarditis
- Post MI complication (usually transient)
- Hyperthyroidism
- Alcoholism
- Post cardiac surgery (usually transient)

ECG Criteria

1) Heart Rate: Atrial rate is 250-350 bpm. Ventricular rate varies according to AV node conduction.
2) Rhythm: Atrial regular; ventricular may be regular or irregular (again, depending on AV node conduction).
3) P-waves: Absent. Only flutter or saw tooth looking wave forms.
4) PR Interval: Not applicable.

5) QRS Width: $\leq$ 0.12 seconds.

Atrial Fibrillation

Atrial fibrillation (often called “a. fib” or “atrial fib”) may result from multiple areas of re-entry within the atria or from multiple ectopic foci. The atrial electrical activity is very rapid (approximately 400 bpm), but each electrical impulse results in the depolarization of only a small islet of atrial myocardium rather than the whole atrium. As a result, there is no contraction of the atria as a whole. Since there is no uniform atrial depolarization, there is no P-wave. The chaotic electrical activity does produce a deflection on the ECG, referred to as a fibrillatory wave. Fibrillatory waves vary in size and shape and are irregular in rhythm. Fibrillatory waves look different from the sawtooth waves of atrial flutter. Transmission of these multiple atrial impulses into the AV node is thought to occur at random, resulting in an irregular rhythm. Some impulses are conducted into but not through the AV node (they are blocked within the AV node). Remember that the ventricular rhythm is always irregular in atrial fibrillation.When the ventricular rate is < 100 bpm, we call this “controlled atrial fibrillation”. If the ventricular rate is >100 bpm, it is labeled “uncontrolled atrial fibrillation”. Since diastolic filling is enhanced when the HR is <100, our goal is to have controlled atrial fibrillation. This can often be accomplished with drug therapy.

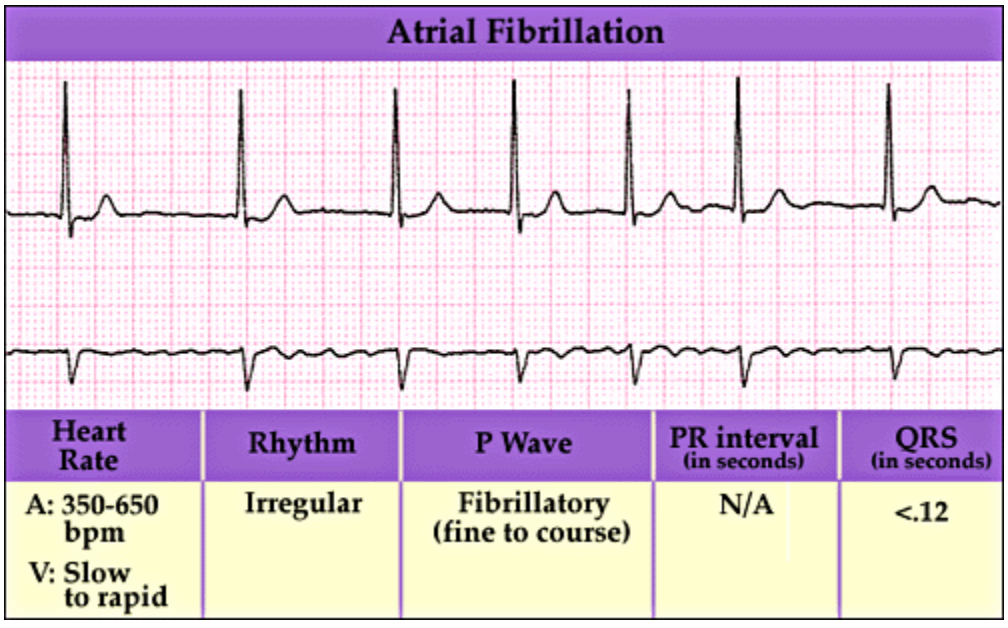


Fig. 7.5: Atrial Fibrillation

Possible Causes

- Mitral valve disorders
- Rheumatic heart disease, MI, hypertension, coronary artery disease (CAD), heart failure, pericarditis.
- Chronic obstructive pulmonary disease (COPD)
- Digoxin toxicity

- Post cardiac surgery (usually transient)

ECG Criteria

1) Heart Rate: Atrial rate 350-400 bpm. Ventricular rate is variable

2) Rhythm: Ventricular rate is irregular (one of the hallmark signs of atrial fibrillation)

3) P-waves: Absent. Only atrial fibrillatory waves (or small looking bumps) are seen

4) PR Interval: Not applicable

5) QRS Width: $\leq$ 0.12 seconds

On the ECG, AF is described by the replacement of consistent P-waves by rapid oscillations or fibrillatory waves that vary in size, shape, and timing, associated with an irregular, frequently rapid ventricular response when atrioventricular (AV) conduction is intact. The ventricular response to AF depends on electrophysiological properties of the AV node, the level of vagal and sympathetic tone, and the action of drugs. Regular RR intervals are possible in the presence of AV block or interference due to ventricular or junctional tachycardia. In patients with electronic pacemakers, diagnosis of AF may require temporary inhibition of the pacemaker to expose atrial fibrillatory activity. A rapid, irregular, sustained, wide-QRS-complex tachycardia strongly suggests AF with conduction over an accessory pathway or AF with underlying bundle-branch block. Extremely rapid rates (over 200 bpm) suggest the presence of an accessory pathway. The picture below shows AF with fast ventricular rate.

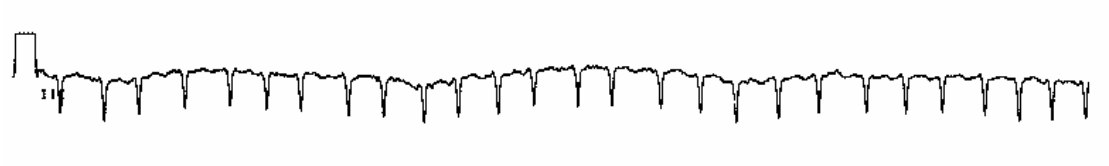


Fig. 7.6: Atrial Fibrillation with Fast Ventricular Rate

7.5 SUPRAVENTRICULAR TACHYCARDIA

The term “supraventricular arrhythmia” refers to a diverse group of abnormal rhythms ranging from chronic atrial fibrillation to paroxysmal sinus tachycardia due to reentry within the sinus node. Supraventricular tachycardia on the other hand can be broadly defined as any tachycardia requiring the atrium or the atrioventricular (AV) node, either in whole or in part, for its perpetuation. The atrial arrhythmias vary considerably in their rate and regularity, their clinical manifestations and the setting in which they occur. These rhythms are characteristically abrupt in onset and termination and are often seen in patients who does not have evidence of organic heart disease. Although these disturbances in rhythm are generally benign, in patients with organic heart disease a rapid supraventricular rhythm may produce significant hemodynamic complications. In some patients with pre-excitation syndromes and antegrade conduction down an accessory pathway, there is a risk of sudden death.

According to recent AHA statement, the term supraventricular tachycardia includes atrioventricular nodal reciprocating tachycardia (AVNRT), atrioventricular reciprocating tachycardia (AVRT), and atrial tachycardia (AT). In this section we would be discussing the details of ECG recognition of AVNRT and AVRT. Atrial tachycardia has been discussed earlier. This section would deal with AVRT and AVNRT.

All narrow QRS (duration<120msec) tachycardias are invariably supraventricular tachycardias.

The following flow chart helps in understanding the differential diagnosis of narrow QRS tachycardia.

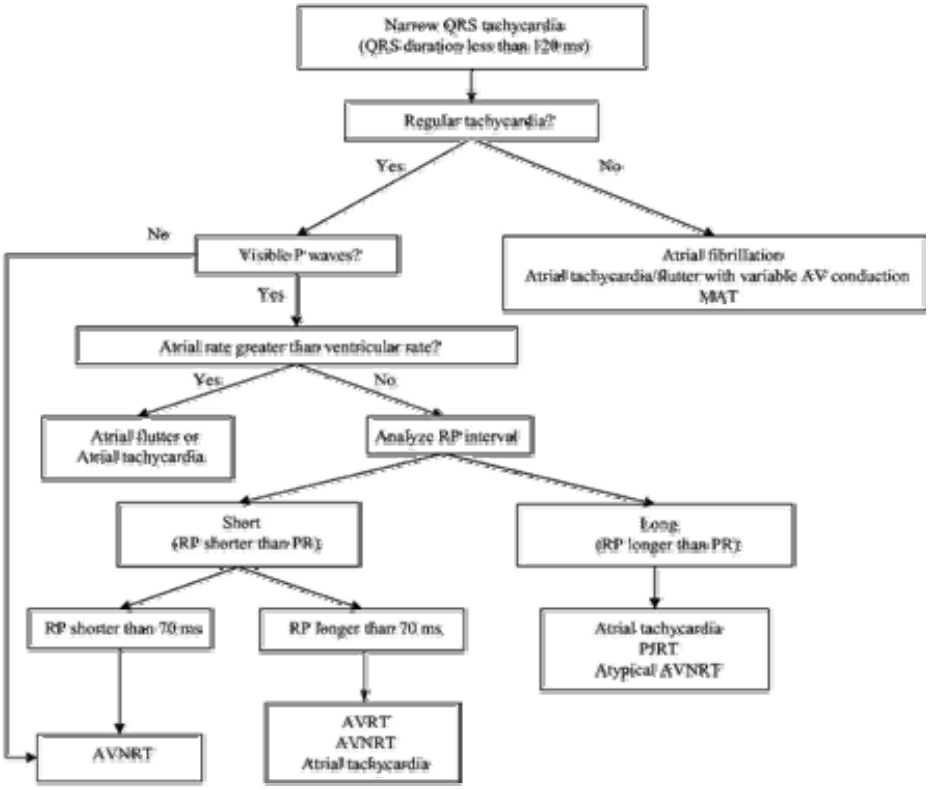


Fig. 7.7: Differential Diagnosis of Narrow QRS Tachycardia

If the RR intervals are irregular then arrhythmia could be one of the following:

1) Atrial fibrillation;

2) Multifocal atrial tachycrdia; and

3) Paroxysmal atrial tachycardia or atrial flutter with varying block.

All regular narrow QRS tachycardias are broadly classified into two groups based on the relationship of RP and PR interval as short and long RP tachycardias. Short RP tachycardias include AVNRT, AVRT and atrialtachycardias. If no P-waves or evidence of atrial activity is apparent and the RR interval is regular, then AVNRT is most commonly the mechanism. P-wave activity in AVNRT may be only partially hidden within the QRS complex and may deform the QRS to give a pseudo–R-wave in lead V1 and/or a pseudo–S wave in inferior leads. If a P-wave is present in the ST-segment and separated from the QRS by 70 ms, then AVRT is most likely. The long RP tachycardias are typical AVNRT, permanent form of junctional reciprocating tachycardia (PJRT) and atrial tachycardias. Responses of narrow QRS-complex tachycardias to adenosine or carotid massage may aid in the differential diagnosis . A 12-lead ECG recording is desirable during use of adenosine or carotid massage. If P waves are not visible, then the use of esophageal pill electrodes can also be helpful. Very rarely what appears to be a narrow RQS tachycardia is a fascicular VT or high septal origin VT.

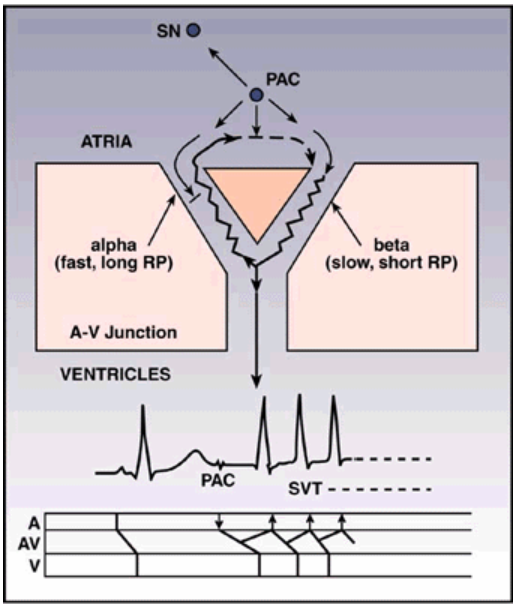


Fig. 7.8: Mechanisms of AVNRT

The cartoon above shows the mechanism of typical AVNRT.

Slow-fast form: In this common form of AV nodal reentry tachycardia, a reentrant circuit is composed of a slow pathway with a short refractory period (RP) and a fast pathway with a long RP. A premature beat is required to initiate tachycardia, and the tachycardia uses the slow pathway for antegrade conduction and the fast pathway for retrograde conduction.

Fast-slow form: In this unusual form of AV nodal reentrant tachycardia, sometimes referred to as "incessant tachycardia", the slow pathway has a long RP and the fast pathway has a short RP. A premature beat is not necessary to initiate tachycardia; a normally timed sinus beat may initiate it.

Summary of ECG criteria

- 150 to 250 beats/minute.
- QRS: normal duration unless bundle branch block is present.
- P-waves: When P-waves are identifiable, the P-wave morphology is often different from sinus P-wave morphology, and the P-wave may precede, coincide with or follow the QRS complex.

AVRT

AV Reciprocating Tachycardia (Extranodal Bypass Pathway): This is the second most common form of PSVT and is seen in patients with WPW syndrome. The WPW ECG, shows a short PR, ***delta*** wave, and somewhat widened QRS.

This type of PSVT can also occur in the absence of manifest WPW on a preceding ECG if the accessory pathway only allows conduction in the retrograde direction (i.e., ***concealed*** WPW). Like AVNRT, a PAC that finds the bypass track temporarily refractory usually initiates the onset of PSVT. The PAC conducts down the normal AV pathway to the ventricles, and reenters the atria retrogradely through the bypass track. In this type of PSVT retrograde P-waves appear shortly after the QRS in the ST-segment (i.e., RP' < 1/2 RR interval). Rarely the antegrade limb for PSVT uses the bypass track and the retrograde limb uses the AV junction; the PSVT then resembles a wide QRS tachycardia and must be differentiated from ventricular tachycardia.

Preexcitation

This condition causes widening of QRS complex. QRS complex represents a ***fusion*** between ***two*** ventricular activation fronts.

Early ventricular activation in region of the accessory AV pathway (***Bundle of Kent***) and ventricular activation through the normal AV junction, bundle branch system. ECG criteria include all of the following:

1) Short PR interval (<0.12s);

2) Initial slurring of QRS complex (***delta wave***) representing early ventricular activation through normal ventricular muscle in region of the accessory pathway;

3) Prolonged QRS duration (usually >0.10s); and

4) Secondary ST-T changes due to the altered ventricular activation sequence.

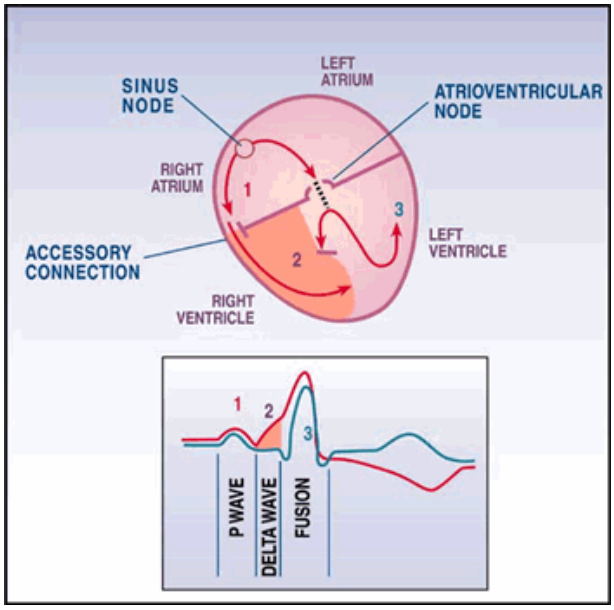


Fig. 7.9: ECG Changes in WPW Syndrome

The cartoon above shows the cardiac abnormality and consequent ECG changes in WPW syndrome. QRS morphology, including polarity of delta wave depends on the particular location of the accessory pathway as well as on the relative proportion of the QRS complex i.e. due to early ventricular activation (i.e., degree of fusion). Delta waves, if negative in polarity, may mimic infarct Q-waves and result in false positive diagnosis of myocardial infarction.

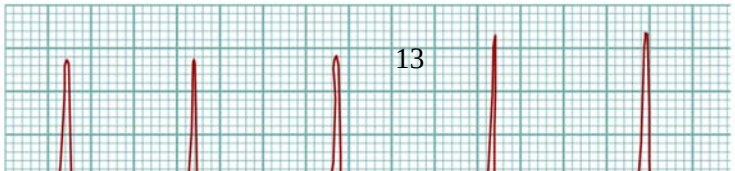


Fig. 7.10: Short PR interval and delta Wave

Typical accessory pathways are extra nodal pathways that connect the myocardium of the atrium and the ventricle across the AV groove. Delta waves detectable on an ECG have been reported to be present in 0.15 per cent to 0.25 per cent of thegeneral population. Pathway conduction may be intermittent.

A higher prevalence of 0.55 per cent has been reported in first degree relatives of patients with accessory pathways. Accessory pathways can be classified on the basis of their location along the mitral or tricuspid annulus; type of conduction (decremental [i.e., progressive delay in accessory pathway conduction in response to increased paced rates] or nondecremental); and whether they are capable of anterograde conduction, retrograde conduction, or both. Accessory pathways usually exhibit rapid, nondecremental conduction, similar to that present in normal His-Purkinje tissue and atrial or ventricular myocardium. Approximately 8 per cent of accessory pathways display decremental anterograde or retrograde conduction.

The term “permanent form of junctional reciprocating tachycardia” is used to refer to a rare clinical syndrome involving a slowly conducting, concealed, usually posteroseptal (inferoseptal) accessory pathway. This syndrome is characterized by an incessant SVT, usually with negative P-waves in leads II, III, and aVF and a long RP interval (RP more than PR). Accessory pathways that are capable of only retrograde conduction are referred to as “concealed,” whereas those capable of anterograde conduction are “manifest,” demonstrating pre-excitation on a standard ECG. The degree of pre-excitation is determined by the relative conduction to the ventricle over the AV node. His bundle axis versus the accessory pathway. In some patients, anterograde conduction is apparent only with pacing close to the atrial insertion site, as, for example, for left-lateral–located pathways. Manifest accessory pathways usually conduct in both anterograde and retrograde directions. Those that conduct in the anterograde direction only are uncommon, whereas those that conduct in the retrograde direction are common. The diagnosis of WPW syndrome is reserved for patients who have both pre-excitation and tachyarrhythmias. Among patients with WPW syndrome, AVRT is the most common arrhythmia, accounting for 95 per cent of re-entrant tachycardias that occur in patients with an accessory pathway. Atrioventricular re-entry tachycardia is further subclassified into orthodromic and antidromic AVRT. During orthodromic AVRT, the re-entrant impulse conducts over the AV node and the specialized conduction system from the atrium to the ventricle and utilizes the accessory pathway for conduction from the ventricle to the atrium.

During antidromic AVRT, the re-entrant impulse travels in the reverse direction, with anterograde conduction from the atrium to the ventricle occurring via the accessory pathway and retrograde conduction over the AV node or a second accessory pathway. Antidromic AVRT occurs in only 5 per cent to 10 per cent of patients with WPW syndrome. Pre-excited tachycardias can also occur in patients with AT, atrial flutter, AF, or AVNRT, with the accessory pathway acting as a bystander (i.e., not a critical part of the tachycardia circuit). Atrial fibrillation is a potentially life-threatening arrhythmia in patients with WPW syndrome. If an accessory pathway has a short anterograde refractory period, then rapid repetitive conduction to the ventricles during AF can result in a rapid ventricular response with subsequent degeneration to VF. It has been estimated that one-third of patients with WPW syndrome also have AF. Accessory pathways appear to play a pathophysiological role in the development of AF in these patients, as most are young and do not have structural heart disease. Rapid AVRT may play a role in initiating AF in these patients. Surgical or catheter ablation of accessory pathways usually eliminates AF as well as AVRT.

The incidence of sudden cardiac death in patients with the WPW syndrome has been estimated to range from 0.15 per cent to 0.39 per cent over 3 to 10 year follow-up. It is unusual for cardiac arrest to be the first symptomatic manifestation of WPW syndrome. Conversely, in about half of the cardiac arrest cases in WPW patients, it is the first manifestation of WPW. Given the potential for AF among patients with WPW syndrome and the concern about sudden cardiac death resulting from rapid pre-excited AF, even the low annual incidence of sudden death among patients with the WPW syndrome is of note and supports the concept of liberal indications for catheter ablation. Studies of WPW syndrome patients who have experienced a cardiac arrest have retrospectively identified a number of markers that identify patients at increased risk. These include 1) a shortest pre-excited R-R interval less than 250 ms during spontaneous or induced AF, 2) a history of symptomatic tachycardia, 3) multiple accessory pathways, and 4) Ebstein's anomaly. A high incidence of sudden death has been reported in familial WPW. This familial presentation is, however, exceedingly rare.

Check Your Progress 3

1) What is the difference in the appearance of the QRS complex between supraventricular and ventricular premature (ectopic) beats?

   .....

   .....

2) What is meant by the term R on T ventricular premature beats and what is the significance?

   .....

   .....

3) What are the features of the Wolff Parkinson white (WPW) syndrome?

   .....

   .....

   .....

.....

.....

.....

.....

7.6 VENTRICULAR TACHYCARDIA AND VENTRICULAR FIBRILLATION

Ventricular Tachycardia (VT) is defined as three or more consecutive PVCs in a row at a rate greater than 100 beats per minute. VT is generally caused by single foci in either ventricle that fire at a rapid rate to override the SA node and thereby take control of the heart's rhythm. A short run of consecutive PVCs is often called a "burst" of VT. ECG characteristics include a rapid, regular rhythm with a wide QRS. The QRS is wide since the origin of the rhythm is outside the bundle branches, thereby taking a longer time to conduct cell to cell within the ventricle. Definitions used to classify ventricular arrhythmias:

- sustained: > 30 second duration or requiring intervention for termination
- non-sustained: 6 beats – 29 second
- monomorphic: regular rate and consistent beat-to-beat QRS morphology
- polymorphic: frequent changes of QRS morphology and/or axis. If sustained changes must occur at least every 1–2 second
- repetitive ventricular beats: 1–5 ventricular beats

Atrioventricular dissociation is present, but not always noticeable. This means that the sinus node is depolarizing the atria in a normal manner at a rate either equal to or slower than the ventricular rate. Thus sinus P-waves sometimes can be recognized between QRS complexes but do not conduct down into the ventricles. AV Dissociation strongly suggests ventricular tachycardia. Unfortunately AV dissociation only occurs in approximately 50 per cent of ventricular tachycardias (the other 50 per cent have retrograde atrial capture or "VA association"). Of the patients with AV dissociation, it is only easily recognized if the rate of tachycardia is <150 bpm. Faster heart rates make it difficult to visualize dissociated P-waves. This arrhythmia may be either well tolerated or associated with life-threatening hemodynamic compromise. The hemodynamic consequences of VT depend largely on the presence or absence of myocardial dysfunction (such as might result from ischaemia or infarction) and on the rate of VT (the faster the rate, the less well tolerated).

Possible Causes

- Myocardial irritability
- Acute MI
- CAD
- Drug toxicity
- Electrolyte imbalance

- Heart failure

ECG Criteria

1) Heart Rate: 100-250 bpm.

2) Rhythm: Ventricular rhythm regular.

3) P-waves: P-waves may or may not be seen. If present, they are not associated with the QRS complex. (AV disassociation occurs with this rhythm, but P-waves are not always seen).

4) PR Interval: Not applicable.

5) QRS Width: > 0.12 seconds, wide and bizarre in appearance.

6) Fusion beats or captures often occur when there is AV dissociation and this also strongly suggests a ventricular origin for the wide QRS tachycardia.

7) QRS morphology in V1 to V6: consider a few other morphology clues:

   - Bizarre frontal plane QRS axis (i.e. from +150 degrees to –90 degrees or NW quadrant) suggests ventricular tachycardia.

   - QRS morphology similar to previously seen PVCs suggests ventricular tachycardia.

   - If all the QRS complexes from V1 to V6 are in the same direction (positive or negative), ventricular tachycardia is likely.

   - Especially wide QRS complexes (>0.16s) suggests ventricular tachycardia.

   - Also consider the following **Four-step Algorithm** reported by Brugada et al., *Circulation 1991;* 83:1649.

   Step 1: Absence of RS complex in *all* leads V1-V6?

   Yes: Diagnosis is ventricular tachycardia!

   Step 2: **No**: Is interval from beginning of R-wave to nadir of S-wave > 0.1s in any RS lead?

   Yes: Diagnosis is ventricular tachycardia!

   Step 3: **No**: Are AV dissociation, fusions, or captures seen?

   Yes: Diagnosis is ventricular tachycardia!

   Step 4: **No**: Are there morphologic criteria for VT present both in leads V1 and V6?

   Yes: Diagnosis is ventricular tachycardia!

8) Pre-existing complete BBB, if present is very helpful in diagnosis. For example, in a patient with complete RBBB during sinus rhythm, it is highly likely that wide complex tachycardia with LBBB pattern is VT.

9) Narrow complex tachycardia. Very rarely VT may be narrow complex. This may be the case, for example, if, in a patient with an anterior wall aneurysm the site of origin of the VT is in the basal portion of the intraventricular septum. Arrhythmia may then

spread over both ventricles in a similar fashion to the spread of intraventricular beats thereby causing a narrow QRS VT.

10) If in doubt, in patients with structural heart disease it is safer to diagnose VT and this usually proves to be correct. The ECG below shows a regular wide QRS tachycardia which was diagnosed as VT. Two examples of VT.

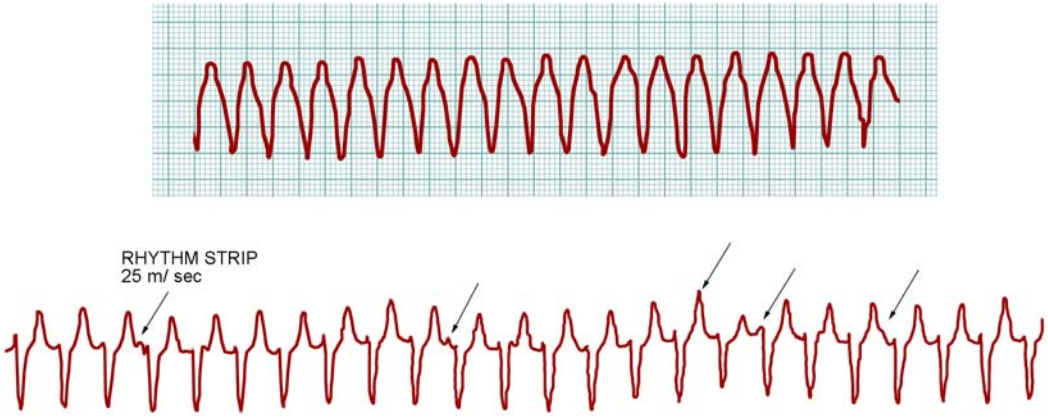


Fig. 7.11: Ventricular Tachycardia

Ventricular Fibrillation (VF)

Ventricular Fibrillation (VF) is the result of highly irritable ventricle(s), which begin to send out rapid electrical stimuli. The stimuli are chaotic resulting in no organized ventricular depolarization. The ventricles do not contract because they never depolarize. Because the ventricles are fibrillating and never contracting, the patient does not have a pulse, cardiacoutput, or blood pressure. The terms **coarse** and **fine** have been used to describe the amplitude of the waveforms in VF. With **Coarse** VF, the fibrillatory waves are more easily seen and are usually greater than 3mm in height (3 small boxes tall). **Coarse VF** usually indicates a more recent onset of VF, which could be more easily converted by prompt defibrillation. The presence of **fine VF** (which looks a bit like asystole and is less than 3mm in height) often means there has been a considerable delay since collapse, and successful resuscitation is more difficult.

Possible Causes

- Acute MI
- Untreated ventricular tachycardia
- Underlying heart disease
- Acid-base imbalance
- Electrolyte imbalances such as hypokalemia, hyperkalemia, and hypercalcemia

ECG Criteria

1) Heart Rate: None. No discernable P-waves or QRS complexes

2) Rhythm: Chaotic wavy recording. No discernable rhythm.

3) P-waves: None

4) PR Interval: Not applicable

5) QRS Width: Not applicable

ECG strip shows ventricular fibrillation.

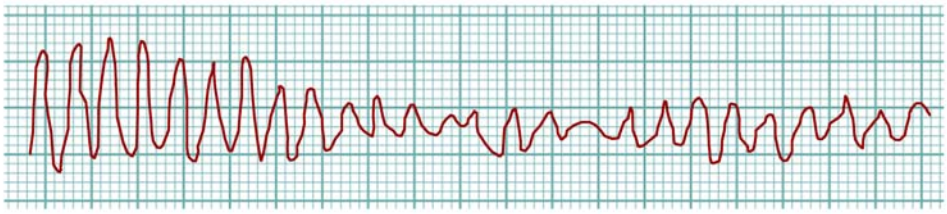


Fig. 7.12: Ventricular Fibrillation

Torsades de Pointes

Definition

- VT characterized by QRS complexes of progressively changing amplitude and contour that seem to revolve about the isoelectric line.

- diagnosis based on characteristic VT and prolonged ventricular repolarisation time with QT intervals usually > 500 msec.

- additional ECG features that may be present include prominent U-wave, abnormal contour of T or TU-waves, T-wave alternans, subnormal spontaneous sinus rate (especially in children) and sinus pauses.

- Standard ECG recordings and analysis of QTc duration and T-wave morphology are the most useful tests in diagnosing LQTS. The prolongation of the QTc interval is defined based on age-specific and sex-specific criteria. The QTc, corrected for heart rate, is calculated by dividing the measured QT by the square root of the R-R interval, both of which are measured in seconds. QTc prolongation greater than 0.46 seconds indicates a high likelihood of LQTS diagnosis. However, approximately 10-15 per cent of gene-positive patients with LQTS present with QTc duration within the reference range.

Definition of the QTc Interval Based on Age-Specific and Sex-Specific Criteria.

Age and Sex	Prolonged QTc (Seconds)	Borderline QTc (Seconds)	Reference Range (Seconds)
Children (<15 y)	>0.46	0.44-0.46	<0.44
Adult males	>0.45	0.43-0.45	<0.43

Adult females	>0.46	0.45-0.46	<0.45

- In patients with suggested LQTS with borderline QTc values (or even values within the reference range) in standard ECG, the analysis of dynamic behavior of QTc duration during exercise ECG testing or long-term Holter monitoring may reveal maladaptation of the QT interval duration to the changing heart rate, with evident QTc prolongation at a faster heart rate. Ventricular arrhythmias rarely are observed during exercise testing or Holter recordings in patients with LQTS.

- Invasive electrophysiology testing with attempts to induce ventricular tachycardia do not facilitates diagnosis.

- Detection of visible T-wave alternans in patients with LQTS indicates increased risk of cardiac arrhythmias (i.e, torsade de pointes and ventricular fibrillation).

- Detection of microvolt T-wave alternans has low sensitivity and high specificity in diagnosing LQTS. The prognostic value of microvolt T-wave alternans has not been studied systematically.

The following picture shows torsades.

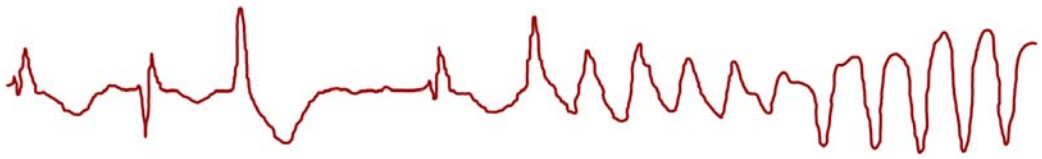


Fig. 7.13: Torsades de Points

Causes of Long QT

Congenital

- Jervell-Lange-Nielsen syndrome. Autosomal recessive. Long QT and neural deafness
- Romano-Ward. Autosomal dominant. Normal hearing
- Sporadic form. Normal hearing
- Acquired: largely iatrogenic disease with most cases being due to drugs
- Antiarrhythmics
  - — type 1a and type 3 drugs associated with torsades
  - — of the type 1a drugs quinidine appears to have the greatest potential for causing it and is estimated to cause syncope in 0.5-4 per cent of patients as a result of this tachyarrythmia. Prolongs QT interval in most patients, whether or not ventricular arrhythmias occur, but significant QT prolongation (500-600 ms) more often a characteristic of patients with quinidine syncope
  - — class Ic drugs may also prolong QT interval

- Non-cardiac drugs. Following have been associated with long QT and torsades:
  - — phenothiazines especially thioridazine
  - — tricyclic and occasionally tetracyclic antidepressants
  - — H1 blockers (e.g. terfenadine and astemizole; former particularly in association with erythromycin)
- Metabolic and electrolyte disorders
  - — hypokalaemia
  - — hypomagnesemia
  - — appears to be synergistic effect between these electrolyte disorders and type 1a drugs
- Bradycardia. VT is a complication of severe bradycardia
- CNS lesions
  - — intracranial disease, especially subarachnoid haemorrhage, occasionally produces torsades. Probably due to the influence of autonomic nervous system on ventricular repolarisation
- Cardiac lesions
  - — mitral valve prolapse and cardiac ganglionitis may be associated with long QT

Clinical Features

Congenital long QT syndrome

- may present in childhood with syncope due to torsades. Often precipitated by heightened sympathetic tone
- sudden death may occur but frequency of this complication seems to vary considerably from family to family (< 5% to 80 per cent)
- VT may evolve into VF but unlike VF associated with coronary or structural heart disease often resolves spontaneously to sinus rhythm

Acquired long QT

- principal clinical features are syncope and pre-syncope, often accompanied by palpitations
- sudden death may occur

Check Your Progress 5

1) What is the definition for ventricular tachycardia?

.....

.....

2) What is meant by the terms monomorphic and polymorphic ventricular tachycardia?

.....

.....

3) What is the Brugada algorhythm for the evaluation of a broad QRS tachycardia?

.....

.....

4) What are the hemodynamic consequences of ventricular fibrillation?

.....

.....

5) What is meant by the term Torsade de Pointes?

.....

.....

6) What are the features of Torsade?

.....

.....

7) What is meant by the long QT syndrome? (LQTS).

.....

.....

7.7 SINOATRIAL AND ATRIOVENTRICULAR BLOCKS

This section considers all the important disorders of impulse conduction that may occur within the cardiac conduction system. Heart block can occur anywhere in the specialized conduction system beginning with the sino-atrial connections, the AV junction, the bundle branches and their fascicles, and ending in the distal ventricular Purkinje fibers. Disorders of conduction may manifest as slowed conduction (1st degree)*, intermittent conduction failure* (2nd degree)*, or complete conduction failure* (3rd degree)*. In addition, 2nd degree heart block occurs in two varieties – Type I* (Wenckebach) *and Type II* (Mobitz)*. In Type I block there is* decremental conduction *which means that conduction velocity progressively slows down until failure of conduction occurs. Type II block is all or none. The term* exit block *is used to identify conduction delay or failure immediately distal to a pacemaker site. Sino-atrial (SA) block is an exit block. This section considers conduction disorders in the anatomical sequence that defines the cardiac conduction system.*

Sino-Atrial Exit Block (SA Block)

Second Degree SA Block: This is the only degree of SA block that can be recognized on the surface ECG (i.e., intermittent conduction failure between the sinus node and the right atrium). There are two types, although because of sinus arrhythmia they may be

hard to differentiate. Furthermore, the differentiation is electrocardiographically interesting but not clinically important.

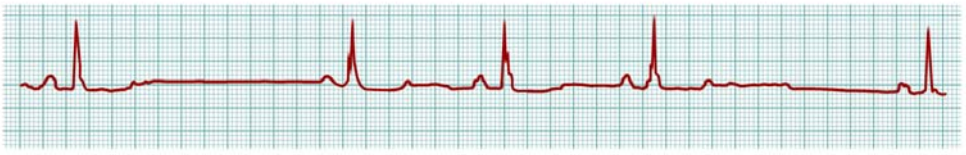
Type I (SA Wenckebach): the following three rules represent the classic rules of Wenckebach, which were originally described for **Type I *AV* block**. The rules are the result of decremental conduction where the ***increment*** in conduction delay for each subsequent impulse gets smaller until conduction failure finally occurs. This declining increment results in the following findings:

1) PP intervals gradually shorten until a pause occurs (i.e., the blocked sinus impulse fails to reach the atria);

2) The pause duration is ***less than*** the two preceding PP intervals; and

3) The PP interval following the pause is ***greater than*** the PP interval just before the pause.

Differential Diagnosis: sinus arrhythmia without SA block.



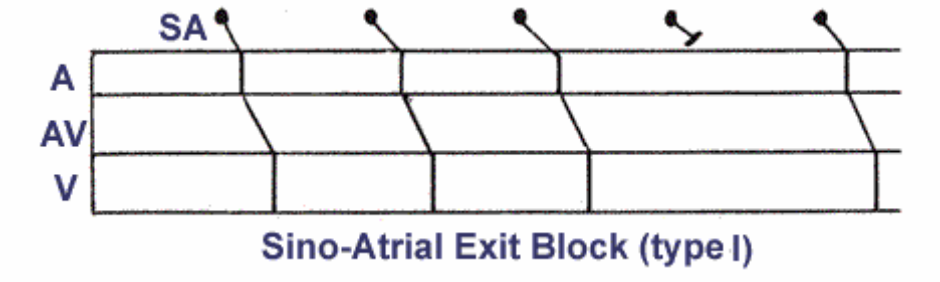


Fig. 7.14: Sino-Atrial Exit Block (Type I)

Type II SA Block: 1) PP intervals are fairly constant (unless sinus arrhythmia present) until conduction failure occurs. 2) The pause is approximately ***twice*** the basic PP interval.

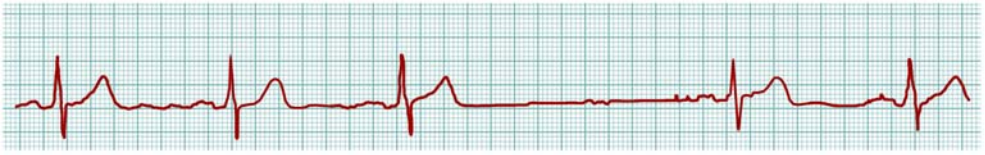


Fig. 7.15: Sino-Atrial Exit Block (Type II)

Atrio-Ventricular (AV) Block

Possible sites of AV block**:**

AV node (most common)

His bundle (uncommon)

Bundle branch and fascicular divisions (in presence of already existing complete bundle branch block)

1st Degree AV Block: The following are the ECG criteria:

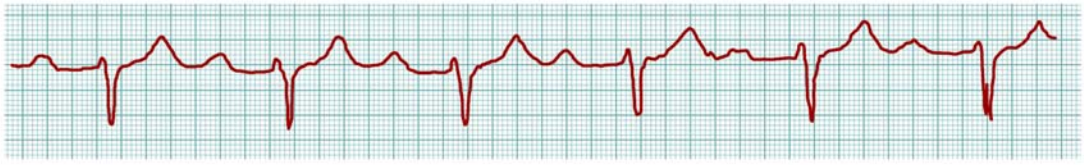


Fig. 7.16: 1st Degree AV Block (PR = 280 ms)

1) PR interval > 0.20 sec,

2) All P-waves conduct to the ventricles.

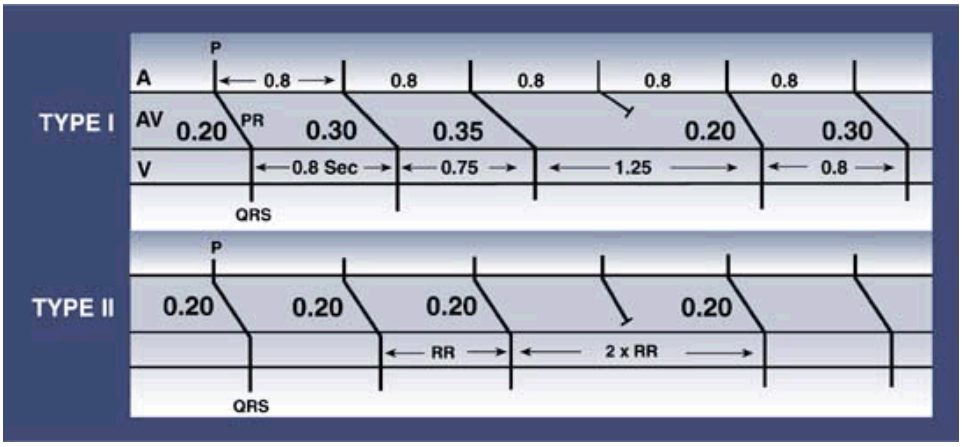


Fig. 7.17: 2nd Degree AV Block

In "classic" Type I ***(Wenckebach)*** AV block, the PR interval gets longer ***(by shorter increments)*** until a nonconducted P-wave occurs. The RR interval of the pause is ***less than*** the two preceding RR intervals, and the RR interval after the pause is ***greater than*** the RR interval before the pause. These are the ***classic*** rules of ***Wenckebach*** (atypical forms can occur).

In Type II ***(Mobitz)*** AV block the PR intervals are constant until a nonconducted P-wave occurs. There must be ***two consecutive constant*** PR intervals to diagnose Type II AV block (i.e., if there is 2:1 AV block we can't be sure if its type I or II). The RR interval of the pause is ***equal to*** the two preceding RR intervals. Type I AV block is ***almost always*** located in the AV node, which means that the QRS duration is usually narrow, unless there is preexisting bundle branch disease. Type II AV block is ***almost always*** located in the bundle branches, which means that the QRS

duration is wide indicating complete block of one bundle; the nonconducted P-wave is blocked in the other bundle.

Fig. 7.18: 2nd Degree AV Block (Type II) with LBBB

Complete (3rd Degree) AV Block

Usually see ***complete AV dissociation*** because the atria and ventricles are each controlled by separate pacemakers. Narrow QRS rhythm suggests a junctional escape focus for the ventricles with block above the pacemaker focus, usually in the AV node.

Wide QRS rhythm suggests a ventricular escape focus (i.e., ***idioventricular rhythm***). The location of the block may be in the AV junction or bilaterally in the bundle branches. The ECG below shows complete heart block with narrow QRS escape.

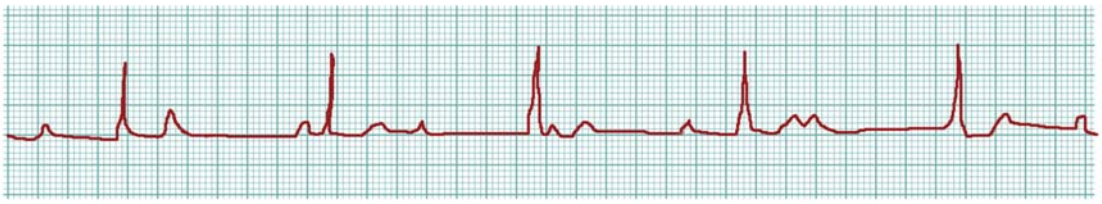


Fig. 7.19: Complete Heart Block with Narrow QRS escape

AV Dissociation (independent rhythms in atria and ventricles):

This is *not* synonymous with 3rd degree AV block, although AV block is *one* of the causes. May be ***complete*** or ***incomplete.*** In *complete* AV dissociation the atria and ventricles are always independent of each other. In *incomplete* AV dissociation there is either intermittent atrial *capture* from the ventricular focus or ventricular *capture* from the atrial focus.

There are ***three categories*** of AV dissociation (categories 1 and 2 are always ***incomplete*** AV dissociation).

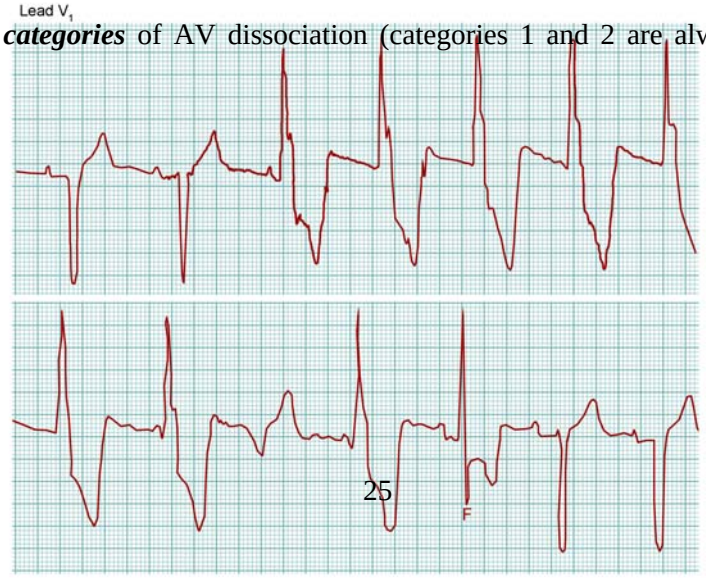


Fig. 7.20: Incomplete AV dissociation (usurpation) due to accelerated ventricular rhythm
F = fusion beat

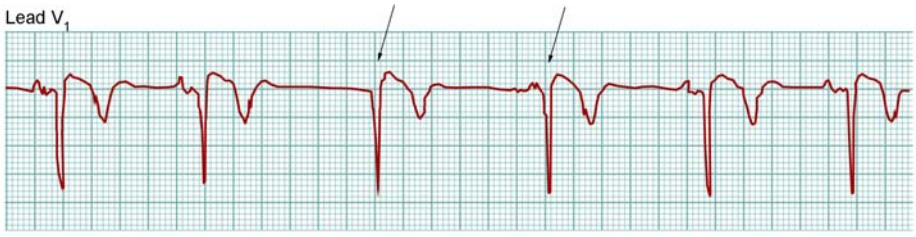


Fig. 7.21: Incomplete AV dissociation due to sinus slowing (default) with junctional escapes (arrows)

Intraventricular Blocks

Right Bundle Branch Block (RBBB)*:*

"Complete" RBBB has a QRS duration $\geq$ 0.12s. Close examination of QRS complex in various leads reveals that the terminal forces (i.e., 2nd half of QRS) are oriented ***rightward*** and ***anteriorly*** because the right ventricle is depolarized ***after*** the left ventricle. This means the following:

1) Terminal R'-wave in lead V1 (usually see rSR' complex) indicating late ***anterior*** forces;

2) Terminal S-waves in leads I, aVL, V6 indicating late ***rightward*** forces; and

3) Terminal R-wave in lead aVR indicating late ***rightward*** forces.

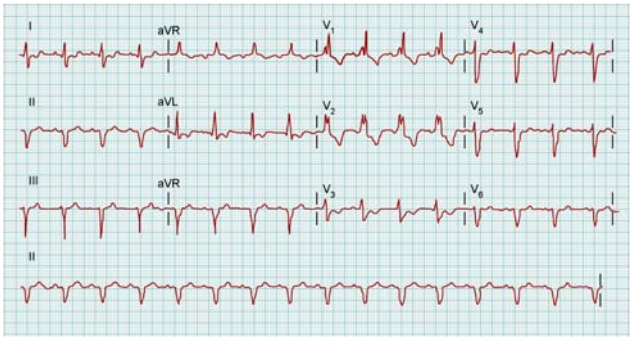


Fig. 7.22: ECG Showing RBBB

The frontal plane QRS axis in RBBB should be in the normal range (i.e., - 30 to + 90 degrees). If left axis deviation is pre cent, think about left anterior fascicular block, and if right axis deviation is present, think about left posterior block in addition to the RBBB. "Incomplete" RBBB has QRS duration of 0.10 - 0.12second with the same terminal QRS features. This is often a normal variant. The "normal" ST-T-waves in RBBB should be oriented opposite to the direction of the terminal QRS forces; i.e., in leads with terminal R or R' forces the ST-T should be negative or downwards; in leads with terminal S forces the ST-T should be positive or upwards. If the ST-T-

waves are in the ***same direction*** as the terminal QRS forces, they should be labeled ***primary ST-T wave abnormalities.***

Left Bundle Branch Block (LBBB)

"Complete LBBB" has a QRS duration $\geq$ 0.12second. Close examination of QRS complex in various leads reveals that the terminal forces (i.e., 2nd half of QRS) are oriented ***leftward*** and ***posteriorly*** because the left ventricle is depolarized ***after*** the right ventricle.

1) Terminal S-waves in lead V1 indicating late ***posterior*** forces.
2) Terminal R-waves in lead I, aVL, V6 indicating late ***leftward*** forces; usually broad, monophasic R-waves are seen in these leads.
3) The "normal" ST-T-waves in LBBB should be oriented opposite to the direction of the terminal QRS forces; i.e., in leads with terminal R or R' forces the ST-T should be downwards; in leads with terminal S forces the ST-T should be upwards. If the ST-T-waves are in the ***same direction*** as the terminal QRS forces, they should be labelled ***primary ST-T wave abnormalities.***

"Incomplete" LBBB looks like LBBB but QRS duration = 0.10 to 0.12second, with less ST-T change. This is often a progression of LVH.

Left Anterior Fascicular Block (LAFB) is the most common intraventricular conduction defect. The following ECG features are seen:

1) Left axis deviation in frontal plane, usually -45 to -90 degrees;
2) rS complexes in leads II, III, aVF;
3) Small q-wave in leads I *and/or* aVL;
4) R-peak time in lead aVL > 0.04second, often with slurred R-wave downstroke;
5) QRS duration usually < 0.12second unless coexisting RBBB;
6) Usually see poor R progression in leads V1-V3 and deeper S-waves in leads V5 and V6; and
7) May ***mimic*** LVH voltage in lead aVL, and ***mask*** LVH voltage in leads V5 and V6.

Left Posterior Fascicular Block (LPFB) is a very rare intraventricular defect. The following ECG features are usually seen:

1) Right axis deviation in the frontal plane (usually > +100 degrees)
2) rS complex in lead I
3) qR complexes in leads II, III, aVF, with R in lead III > R in lead II
4) QRS duration usually < 0.12second unless there is coexisting RBBB

Must first exclude (on clinical grounds) other causes of right axis deviation such as cor pulmonale, pulmonary heart disease, pulmonary hypertension, etc., because these conditions can result in the identical ECG picture.

Bifascicular Blocks

RBBB plus either LAFB (common) ***or*** LPFB (uncommon) Features of RBBB plus frontal plane features of the fascicular block (axis deviation, etc.).

The picture below shows bifascicular block:

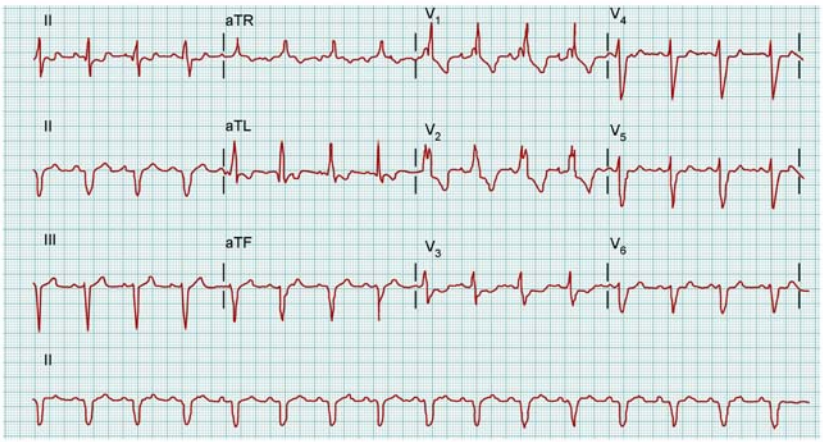


Fig. 7.23: Bifascicular Block

Nonspecific Intraventricular Conduction Defects (IVCD)

QRS duration > 0.10s indicating slowed conduction in the ventricles Criteria for specific bundle branch or fascicular blocks not met Causes of nonspecific IVCD's include:

1) Ventricular hypertrophy (especially LVH)
2) Myocardial infarction (so called *periinfarction blocks*)
3) Drugs, especially class IA and IC antiarrhythmics (e.g., quinidine, flecainide)
4) Hyperkalemia
5) Myocarditis/dilated cardiomyopathy.

Check Your Progress 6

1) What are some of the causes of LQTS?

.....

.....

.....

.....

.....

.....

.....

.....

.....

2) What is 1st degree AV Block?

.....

.....

3) What is 2nd degree AVBlock-Wenckebach type?

.....

.....

4) What is Mobitz AVB lock?

.....

.....

5) What is 3rd degree(complete) AV Block?

.....

.....

.....

.....

6) What are the criteria for right bundle branch block?

.....

.....

.....

.....

7) What are the criteria for left bundle branch block?

.....

.....

.....

.....

7.8 LET US SUM UP

In this unit, you have learnt the various causes of premature beats and its ECG changes. Rapid (more than 100 bpm) rate of discharge of SA node is following the normal conduction pathway then it can cause sinus tachycardia. The causes of ECG changes of atrial flutter and fibrillation. You have also learnt that three or more consecutive premature ventricular contractions in a row at a rate grater than 100 beats per minute in known as ventricular tachycardia. Heart block is one of the most important conduction defect and in this unit, you have already learnt about the various degree of heart block. Slowed conduction in the heart will result Ist degree heart block,

intermittent conduction failure will result IInd degree heart block and complete conduction heart failure is know as IIIrd degree heart block.

7.9 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

1) Bradyarrhythmia refers to any rhythm with rate less than 60 beats/minute.
   Tachyarrhythmia means any rhythm with rate more than 100 beats/minute.

2) There are broadly 2 major mechanisms that produce arrhythmias:

   - Disorders of impulse of formation (automaticity) &
   - Disorders of impulse conduction (block or re-entry)

Check Your Progress 2

Discrete, multifocal P' waves occurring at rates of 100-250/min and with varying P'R intervals (should see at least 3 different P wave morphologies in a given lead). Ventricular response is irregularly irregular (i.e., often confused with A-fib). May be intermittent, alternating with periods of normal sinus rhythm.

Check Your Progress 3

1) The supraventricular premature beats produce a narrow QRS.

   Ventricular premature beats produce an abnormally wide and bizarre QRS complex.

2) Here the R wave of the VPB falls on the preceding T wave.R-on-T VPBs may be especially dangerous in an acute ischemic situation, because the ventricles may be more vulnerable to ventricular tachycardia or fibrillation.

3) This is due to preexcitation through an accessory pathway.The condition causes widening of QRS complex. QRS complex represents a ***fusion*** between ***two*** ventricular activation fronts: Early ventricular activation in region of the accessory AV pathway (***Bundle of Kent***) & ventricular activation through the normal AV junction, bundle branch system. ECG criteria include all of the following:

   1) Short PR interval (<0.12s),
   2) Initial slurring of QRS complex (***delta wave***) representing early ventricular activation through normal ventricular muscle in region of the accessory pathway.
   3) Prolonged QRS duration (usually >0.10s) and
   4) Secondary ST-T changes due to the altered ventricular activation sequence.

Check Your Progress 4

1) Ventricular Tachycardia is defined as three or more consecutive PVCs in a row at a rate greater than 100 beats per minute.

2) Monomorphic: regular rate and consistent beat-to-beat QRS morphology

   Polymorphic: frequent changes of QRS morphology and/or axis. If sustained changes must occur at least every 1-2 second.

3) **Step 1:** Absence of RS complex in all leads V1-V6?

Yes: Diagnosis is ventricular tachycardia!

Step 2: Is interval from beginning of R wave to nadir of S wave >0.1s in any RS lead?

Yes: Diagnosis is ventricular tachycardia!

Step 3: Are AV dissociation, fusions, or captures seen?

Yes: Diagnosis is ventricular tachycardia!

Step 4 Are there morphologic criteria for VT present both in leads V1 and V6?

Yes: Diagnosis is ventricular tachycardia!

4) The ventricles do not contract because they never depolarize. Because the ventricles are fibrillating and never contracting, the patient does not have a pulse, cardiac output, or blood pressure.

5) It is a form of ventricular tachycardia (VT) characterized by QRS complexes of progressively changing amplitude and contour that seem to revolve about the isoelectric line .

6) VT diagnosis based on characteristic VT and prolonged ventricular repolarisation time with QT intervals usually > 500 msec .

Additional ECG features that may be present include prominent U wave, abnormal contour of T or TU waves, T wave alternans, subnormal spontaneous sinus rate (especially in children) and sinus pauses .

7) QTc prolongation greater than 0.46 seconds indicates a high likelihood of LQTS diagnosis.

Check Your Progress 5

1) **Congenital**

- Jervell-Lange-Nielsen syndrome. Autosomal recessive. Long QT and neural deafness
- Romano-Ward. Autosomal dominant. Normal hearing
- **Acquired:** largely iatrogenic disease with most cases being due to drugs
- Antiarrhythmics
  - — type 1a and type 3 drugs associated with torsades
  - — class Ic drugs may also prolong QT interval
- Non-cardiac drugs. Following have been associated with long QT and torsades:
  - — phenothiazines especially thioridazine
  - — tricyclic and occasionally tetracyclic antidepressants

— H1 blockers (eg terfenadine and astemizole; former particularly in association with erythromycin)

Electrolyte changes

- hypokalemia
- hypomagnesemia.

2) The following are the ECG criteria:

   1) PR interval > 0.20 sec,

   2) All P waves conduct to the ventricles.

3) In "classic" Type I ***(Wenckebach)*** AV block, the PR interval progressively gets longer until a nonconducted P wave occurs.

4) In Type II ***(Mobitz)*** AV block the PR intervals are constant until a nonconducted P wave occurs.

5) **Complete (3rd Degree) AV Block**

   Usually there is ***complete AV dissociation*** because the atria and ventricles are each controlled by separate pacemakers.No sequential atrio ventricular activity. Narrow QRS rhythm suggests a junctional escape focus for the ventricles with block above the pacemaker focus, usually in the AV node.

   Wide QRS rhythm suggests a ventricular escape focus (i.e., ***idioventricular rhythm***).

6) "Complete" RBBB has a QRS duration $\geq$0.12s.The terminal forces (i.e., 2nd half of QRS) are oriented rightward and anteriorly because the right ventricle is depolarized ***after*** the left ventricle. This means the following:

   1) Terminal R' wave in lead V1 (usually see rSR' complex) indicating late ***anterior*** forces,

   2) Terminal S waves in leads I, aVL, V6 indicating late ***rightward*** forces

      Terminal R wave in lead aVR indicating late ***rightward*** forces.

7) "Complete" LBBB" has a QRS duration $\geq$0.12s. The terminal forces (i.e., 2nd half of QRS) are oriented leftward and posteriorly because the left ventricle is depolarized ***after*** the right ventricle.

   1) Terminal S waves in lead V1 indicating late ***posterior*** forces

   2) Terminal R waves in lead I, aVL, V6 indicating late ***leftward*** forces; usually broad, monophasic R waves are seen in these leads.

   3) The "normal" ST-T waves in LBBB should be oriented opposite to the direction of the terminal QRS forces; i.e., in leads with terminal R or R' forces the ST-T should be downwards; in leads with terminal S forces the ST-T should be upwards.