# 3

# Cardiac Rhythms and Arrhythmias

Case Example: You have just admitted to the nursery a term infant who was delivered by emergency cesarean section. The obstetrician stated that the infant had experienced intrauterine arrhythmias. The infant's vital signs are stable, he is breathing room air, the chest x-ray is normal, there is no murmur, and the physical assessment examination is normal. The ECG rhythm strip shows a rare beat that is different in appearance from the infant's sinus beats. Could you identify the beat? Would you know if it were something to be concerned about? Would you be able to determine if this infant needed to be kept in the NICU based on his ECG status?

Many nurses believe they need to recognize only the life-threatening arrhythmias and the common ones. However, as the case just cited exemplifies, it is important that NICU nurses be familiar with nonthreatening and less common rhythm disturbances so that they can make thorough assessments. This chapter classifies cardiac rhythms and arrhythmias by the location of the stimulus.

Cardiac rhythms can be classified as five types:

- **Sinus:** Sinus rhythms originate in the SA node. They may be normal, fast, slow, or irregular.
- **Atrial:** Atrial rhythms originate from somewhere in the atria, usually from a focus other than the SA node. They indicate an abnormality in atrial conduction.
- **Junctional:** Also known as nodal rhythms, junctional rhythms originate from a focus in the AV node.
- **Ventricular:** Ventricular rhythms originate from a focus in the ventricles and indicate a problem with ventricular conduction. Any sustained ventricular rhythm can decrease cardiac output, resulting in severely compromised hemodynamics.
- **Heart blocks:** Heart blocks result from an actual blockage to impulse formation or propagation anywhere along the conduction pathway.

Cardiac arrhythmias can also be classified as bradyarrhythmias or tachyarrhythmias. Bradycardia may be caused by depressed function of the SA node or disorders of conduction. Tachycardia may be the result of cellular mechanisms such as re-entry or automaticity. This book discusses bradycardias and tachycardias under each of the pertinent rhythm types.

## SINUS RHYTHMS

Sinus rhythms are so named because they are electrically stimulated by the sinoatrial (SA) node. The SA node is considered to be the heart's pacemaker. Other areas of the heart have the ability to set the pace when normal pacemaking mechanisms fail. However, in sinus rhythms, the SA node normally sets the pace.

## Normal Sinus Rhythm

The rhythm normally seen in neonates and infants is called normal sinus rhythm (Figure 3-1).

### Parameters

- Normal P, QRS, and T waves
- Normal PR interval and QRS interval
- Heart rate of 90–180 bpm
- P wave before every QRS complex
- PP and RR intervals the same throughout the rhythm strip
- No pauses
- No early beats

## Sinus Bradycardia

*Brady-* refers to a slower than normal rhythm. In sinus bradycardia (SB) (Figure 3-2), the rhythm originates in the SA node.

### Parameters

- Normal P, QRS, and T waves
- Normal PR, QRS, and QT interval measurements
- Heart rate less than 90 bpm

When reviewing the ECG, look at the RR intervals. In bradycardia, the intervals lengthen (the R waves occur farther and farther apart) as the heart rate becomes slower and slower (Figure 3-3). Do not rely solely on the digital number on the ECG monitor to diagnose bradycardia. Artifact, such as movement, can give a false reading. Always look at the RR intervals and at the patient before initiating treatment. If the patient is pink and breathing, you may wish to monitor the infant's ability to resume a normal rate.

### Incidence

Sinus bradycardia is the most frequently observed rhythm disturbance in premature infants.[9] It may occur in up to 90 percent of preterm infants on the first day of

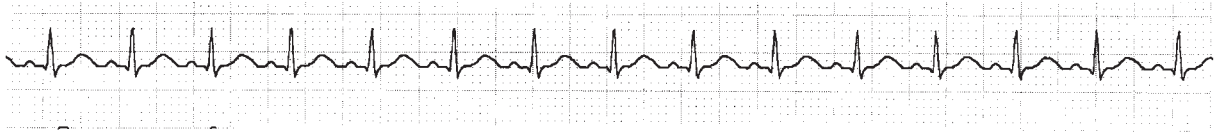

*Figure 3-1 Normal sinus rhythm.*

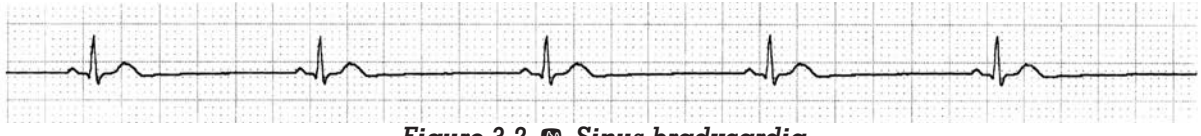

*Figure 3-2 Sinus bradycardia.*

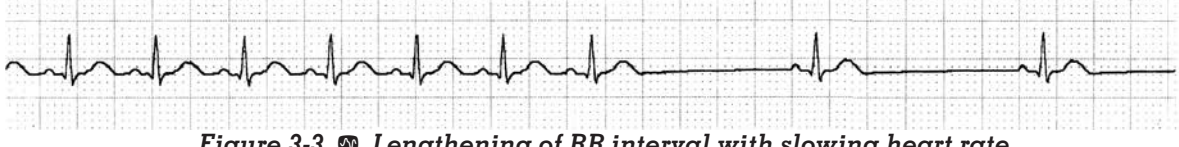

*Figure 3-3 Lengthening of RR interval with slowing heart rate.*

life.[1] It is responsible for up to 35 percent of the arrhythmias in infants.[10]

## Etiology

- Apnea (the most common cause in neonates)
- Blood pressure elevation
- Congenital heart disease
- Electrolyte imbalances
- Hypothermia
- Hypothyroidism
- Immaturity of the central nervous system in the premature infant
- Increased vagal tone, such as with spitting, yawning, distended abdomen, or increased intracranial pressure
- Medications
- Oversedation
- Sinus node disease
- Vagal stimulation

## Treatment

Sinus bradycardia may be transient and asymptomatic. It is important to evaluate the infant's color and respiratory status during any bradycardic episode. If the infant is symptomatic, stimulate or provide respiratory support as indicated. If the slow heart rate persists, begin resuscitation using Neonatal Resuscitation protocol.[11] Treat the cause if the infant is symptomatic or bradycardia recurs.

## Complications

Hypotension and decreased cardiac output can result from sinus bradycardia. If not treated, they can lead to asystole and death.

# Sinus Tachycardia

*Tachy-* refers to a faster than normal rhythm. In sinus tachycardia (also called sinus tach or ST), the SA node stimulates conduction at a faster than normal rate (Figure 3-4).

## Parameters

- Normal P, QRS, and T waves
- Normal PR, QRS, and QT interval measurements
- Heart rate of 180–230 bpm[12]

## Incidence

Brief episodes are common in healthy preterm infants.[12,13]

## Etiology

- Anemia
- Hydrops
- Hyperthermia
- Hyperthyroidism
- Hypovolemia
- Infection
- Pain
- Sympathetic tone changes, increased demands, or changes in the respiratory cycle may cause the heart rate to vary

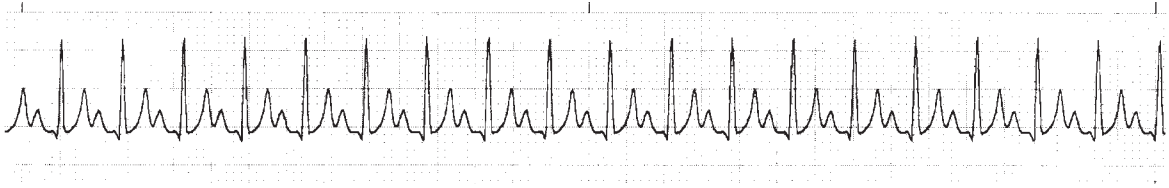

***Figure 3-4 Sinus tachycardia.***

### Treatment

No treatment is indicated if the condition is transient. Remove or treat the cause. With hydrops, ST can be associated with cardiovascular collapse; the infant should be assessed closely and treated as for shock.

### Complications

With any tachycardia, the ventricles do not have sufficient time to fill with blood prior to contraction. This may lead to hypotension and decreased cardiac output.

## Sinus Arrhythmia

As the name implies, sinus arrhythmia (Figure 3-5) is initiated by the SA node. It is a normal variation of cardiac rhythm that is associated with respiration. The heart rate increases during inspiration and decreases during expiration.

### Parameters

- Normal P, QRS, and T waves
- Normal heart rate
- Slight irregularity of RR interval

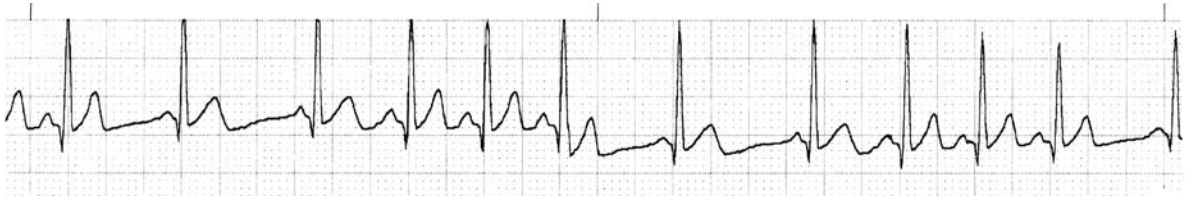

***Figure 3-5 Sinus arrhythmia.***

### Incidence

This arrhythmia is common in the healthy newborn.[13] It usually occurs at lower heart rates, such as when the infant is resting or sleeping.[14]

### Etiology

Sinus arrhythmia is probably the result of intrathoracic pressure changes that occur with respiration.

### Treatment

This is a normal variation and does not require intervention or special monitoring.[14]

### Complications

There are no complications.

## Sinus Arrest

Sinus arrest (Figure 3-6) occurs when the SA node does not send out an impulse or when the impulse is

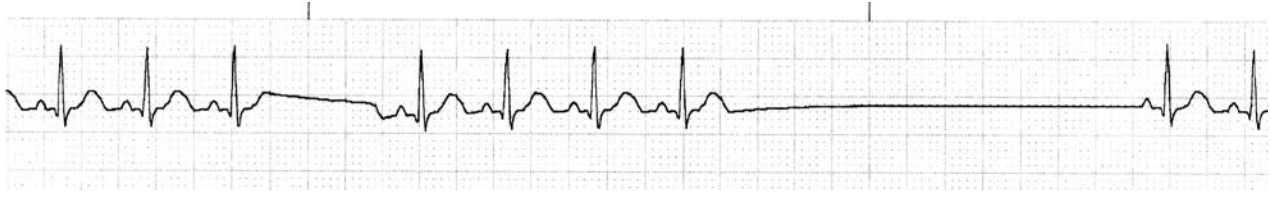

***Figure 3-6 Sinus arrest.***

blocked. After a pause, either the SA node recovers or another pacemaking area takes over. Sinus node dysfunction can be further complicated after cardiac surgery with coexistent atrial arrhythmias. This combination is termed sick sinus syndrome (SSS) or tachy-brady syndrome.[15–17]

## Parameters

- Normal QRS and T waves
- P waves may change in appearance
- Absent or delayed beat, followed by normal conduction
- Heart rate slows during arrest, then may accelerate to normal or tachycardiac arrhythmias

## Incidence

Sick sinus syndrome is common after surgery for congenital heart disease, especially if the surgery is near the sinus node.[15–18]

## Etiology

- Autonomic heart disease associated with sympathetic and parasympathetic responses
- Cardiac surgery
- Digitalis toxicity
- Hyperkalemia
- Increased sensitivity to vagal stimulation
- Increased vagal tone
- Inflammation of the SA node
- Injury to the SA node
- Myopathies

## Treatment

Identify and treat the cause. If bradycardia persists, the infant may require treatment with epinephrine. Isoproterenol has also been used to increase the SA node rate but may result in accelerated rhythms.[15] Atrial pacing may be indicated for known SA node injury and can eliminate the need for drug therapy in some infants.[10]

Pacing may also alleviate signs of fatigue and improve hemodynamic status in SSS.[19]

### Complications

Escape rhythms may follow sinus arrest and compromise cardiac output. Escape rhythms are nonsinus rhythms that occur during or after recovery from another arrhythmia.

## ATRIAL RHYTHMS

Atrial rhythms originate in the atria or the SA node. Because the P wave reflects what is happening in the atria, focusing on what is happening with the P wave helps the clinician determine if atrial rhythm disturbances are present.

### Premature Atrial Contractions

A premature atrial contraction (PAC) (Figure 3-7) is an early beat that originates in a pacemaker

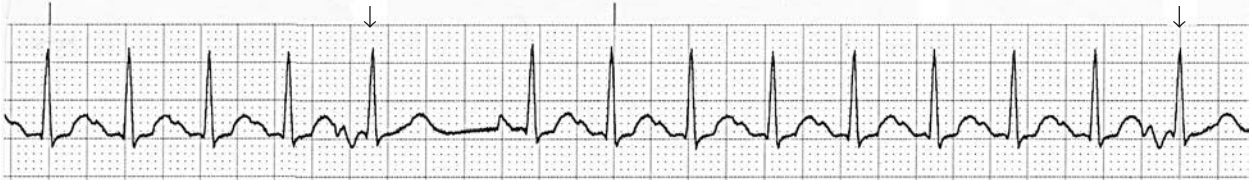

*Figure 3-7 Premature atrial contractions.*

cell of the atria, other than the SA node. The impulse then travels its normal path through the remainder of the conduction system, often resulting in ventricular contraction with a normal QRS complex and T wave. PACs are also known as atrial premature beats (APBs).

## Parameters

- Variation in shape and size of P waves
- Variation in PP interval
- Early beats that otherwise look normal
- Usually normal QRS complexes and T waves

## Incidence

PACs can occur frequently in neonates, yet are rare by one month of age.[12] PACs have been documented in 5 to 30 percent of premature infants.[9]

## Etiology

PACs are almost always benign in the neonate. However, they may not be benign when associated with these factors:

- Atropine, caffeine, theophylline, or inotropic agent administration
- Cardiac tumors, myopathies, or myocarditis
- Digitalis toxicity
- Electrolyte abnormalities
- Hyperthyroidism
- Irritation from a central line
- Sepsis
- Severe respiratory distress or hypoxia
- Structural heart disease

### Treatment

No treatment is necessary for isolated cases. If PACs are associated with symptoms such as hypotension or altered consciousness, treat the cause, and obtain a 12-lead ECG to rule out underlying heart disease.

### Complications

PACs are almost always benign in the asymptomatic infant. Some infants may develop supraventricular tachycardias later. Otherwise, complications are related to the cause.

## Atrial Tachycardia

Atrial tachycardia (atrial tach) (Figure 3-8), is a type of supraventricular tachycardia (SVT) discussed in more detail later in this chapter. There are several forms of atrial tachycardia, including atrial ectopic tachycardia (AET), multifocal atrial tachycardia (MAT), and paroxysmal atrial tachycardia (PAT). In the case of AET, the focus and cause are known. AET and MAT are caused by increased atrial automaticity and can result from repeated firing of a single ectopic focus in the atria, AET, or from multiple ectopic foci in the atria, MAT. Paroxysmal atrial

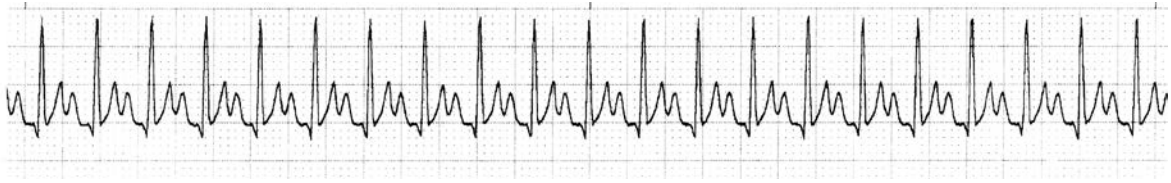

***Figure 3-8 Example of atrial tachycardia (simulation).***

tachycardia is a type of atrial tachycardia that starts and stops suddenly and is characterized by extremely rapid rates (230–300 bpm).

Atrial tachycardia generally has a gradual onset with rates greater than 180 bpm.[6,20] It can be seen in infants with normal hearts, in those with congenital heart disease, and after cardiac surgery because of high levels of circulating catecholamines.[6,15]

## Parameters

- Variation in size and pattern of P waves
- Usually slow to develop (except for PAT)
- Sustained or intermittent rhythm
- Variation in atrial rate
- With AET and MAT, ventricles may respond normally or may require multiple atrial stimuli to evoke a ventricular response[13] (see **Heart Blocks**)

## Incidence

Atrial tachycardia is uncommon but may sometimes be seen after cardiac surgery.[21] AET is probably responsible for 10 percent of all supraventricular tachycardias (SVTs).[18]

## Etiology

- May occur postoperatively because of elevated circulating catecholamines
- May be idiopathic
- Occurs primarily after surgery performed at or near the pulmonary veins[22]
- PAT is associated with cor pulmonale or ischemic heart disease[23]

## Treatment

Adenosine is not effective in treating most forms of atrial tachycardia. Intravenous esmolol can be used acutely.[14] Digoxin may reduce the heart rate by blocking

the AV node.[10,15] Phenytoin may suppress the ectopic focus.[15] Medications such as flecainide, propafenone, atenolol, sotalol, and amiodarone may be used for a refractory rhythm.[14,16,20] Transcatheter or surgical ablation of the focus may be required. Because of the fast rates, PAT is treated with vagal stimulation, adenosine, propranolol, procainamide,[6] cardioversion (Chapter 4), or pacing.

### Complications

Decreased cardiac output.

## Wandering Atrial Pacemaker

The SA node is not always the origin of atrial stimulation. In some instances, the focus (or pacemaker) changes from one area of the atria to another and then another. As the focus changes, or "wanders," the shape of the P wave also changes. This variation in the P wave characterizes the wandering atrial pacemaker rhythm, also known as WAP (Figure 3-9).

### Parameters

- ♥ Variation in shape and size of P wave

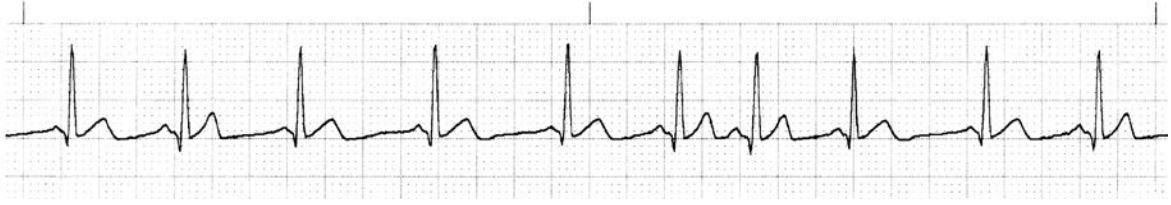

***Figure 3-9 Wandering atrial pacemaker.***

- ♥ Possible variation in PR interval
- ♥ Variation in PP and RR intervals
- ♥ QRS and T waves are normal in shape and measurement

### Incidence

Wandering atrial pacemaker is seen frequently in the newborn period.[22]

### Etiology

Impulses originate from varying atrial foci. Wandering atrial pacemaker is a benign arrhythmia associated with increased vagal tone.[12]

### Treatment

No treatment is necessary for this condition.

### Complications

Wandering atrial pacemaker causes no complications.

## Atrial Flutter

Atrial flutter (Figure 3-10) originates in an ectopic atrial focus. P waves occur in rapid succession. The P waves are all identical, but not all are conducted to the ventricles. The identical, rapidly repeating P waves

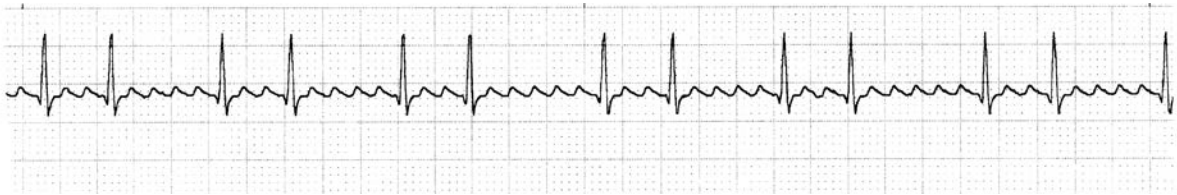

***Figure 3-10 Atrial flutter.***

produce a "sawtooth" pattern on the ECG. Atrial flutter is thought to originate in the right atrium and is sometimes categorized as a type of SVT.[12,24] Atrial flutter can also occur in conjunction with heart block, with a resultant slow ventricular rate.

## Parameters

- Multiple P waves (flutter waves) prior to each QRS complex
- P waves usually have a sawtooth appearance
- Atrial rate of 200–600 bpm[10,15,20,24]
- Slow, normal, or rapid ventricular rate
- A specific number of P waves may occur prior to each QRS complex (ratios such as 2:1, 3:1, or 4:1).

## Incidence

Atrial flutter comprises 9 to 14 percent of neonatal tachyarrhythmias.[25] Arterial catheters are thought to be responsible for three in eight of the episodes noted in infants.[26] Atrial flutter may occur in up to 50 percent of infants after cardiac surgery and is related to atrial scarring and right atrial enlargement.[18,24]

## Etiology

- Atrial septal aneurysms
- Cardiac catheterization
- Congenital heart disease associated with atrial dilation or hydrops
- Costello syndrome
- Damage to the sinus node
- Digoxin toxicity
- Restrictive cardiomyopathy

## Treatment

Treatment goals include slowing the fast rate, preventing recurrence, and decreasing complications and symptoms. Digoxin, diltiazem, and amiodarone may be helpful in controlling rate.[24] Intravenous procainamide

may provide acute conversion, yet should be preceded by digitalization to prevent accelerated AV conduction. Adenosine has been used with varied results.[12] For the hemodynamically unstable infant, cardioversion (Chapter 4) may be indicated. However, cardioversion is contraindicated if the arrhythmia results from digoxin toxicity because electrical stimulation can augment the myocardial excitability in these cases. Radiofrequency catheter ablation can be used for persistent atrial flutter. Digoxin is recommended for long-term suppression.[24] Anticoagulation is indicated for chronic atrial flutter.

### Complications[24]

- Brain and other end organ injury
- Heart failure
- Low cardiac output
- Thrombosis
- Sudden death

## Atrial Fibrillation

Atrial fibrillation (Figure 3-11) is caused by simultaneous firing of multiple foci in the atria (usually originating in the left atrium).[24] These foci fire at different rates, producing an unorganized atrial rhythm. Because only small areas of the atria are depolarized by the chaotic impulses, no real wave of depolarization occurs. As a result, atrial contraction is ineffective, and emptying is minimal. In a normal rhythm, the SA node sends out an impulse that spreads through the atria like the concentric circles caused by tossing a pebble into still water. The erratic depolarization of atrial fibrillation is similar to numerous pebbles being thrown into different areas of the same pool all at once.

### Parameters

- Unidentifiable P waves
- Isoelectric line with many fibrillatory waves that look like artifact

- Normal appearing QRS complexes
- Irregular conduction with variations in RR interval
- Variable ventricular rate of 120–250 bpm

## Incidence

This rhythm is rare in infants; the exact incidence in the newborn remains unknown.[27] Of infants presenting with atrial fibrillation, 77 percent have had intracardiac surgery.[28] Infants with markedly distended atria may also occasionally develop this rhythm.[15]

## Etiology

- Atrial dilation
- Large atrial septal defect (ASD)
- Mitral valve abnormalities

## Treatment

Treatment is similar to that for atrial flutter.[29] If the infant is unstable, cardioversion (Chapter 4) is the primary treatment. If the ventricular rate is fast, digoxin or β-blockers may slow the AV conduction rate. Procainamide,[6] amiodarone, flecainide, propafenone, or

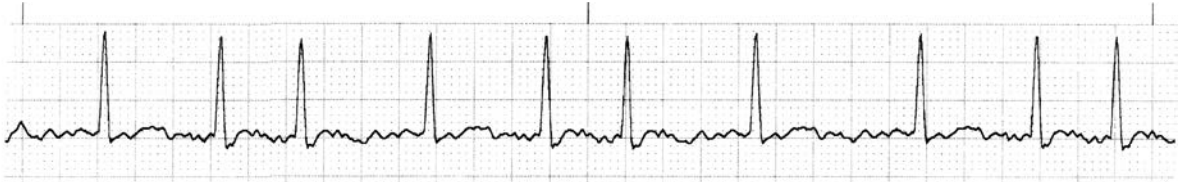

***Figure 3-11 Atrial fibrillation.***

sotalol[15] may be used to prevent recurrence. The long-term treatment is surgical repair of the underlying cardiac defects.

### Complications

Decreased ventricular filling and decreased cardiac output may occur. Inadequate emptying of the atria may cause stagnation and thrombus formation, potentially contributing to cerebral or other organ embolisms.

## Supraventricular Tachycardia

Supraventricular tachycardia (SVT) (Figure 3-12) is a very fast rhythm that originates somewhere above the ventricles. SVT usually originates from an ectopic focus in the atria or from the area around the AV node. SVT is named based on the location of the stimulus if it is known, or based on the cause. Some of the names include atrioventricular tachycardia, re-entrant tachycardia, multifocal atrial tachycardia, pre-excitation tachycardia, paroxysmal atrial tachycardia, and junctional ectopic tachycardia.

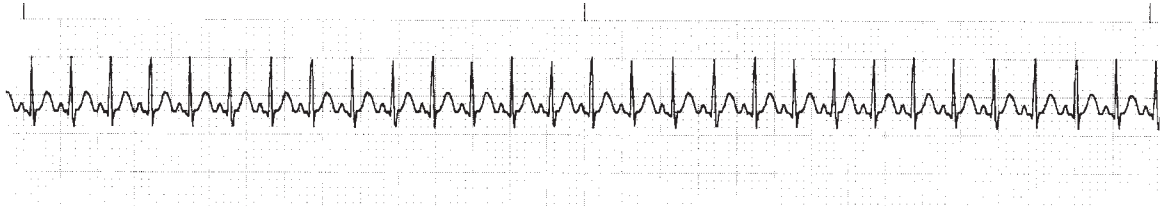

***Figure 3-12 Supraventricular tachycardia.***

## Parameters

- Consistent RR interval
- Heart rate of 230–400 bpm
- Normal or narrow QRS complex
- Relatively fixed heart rate despite changes in activity
- Variation in PR interval[29]
- Visible or invisible P wave (may be lost in previous T wave)

## Incidence

SVT is the most common symptomatic tachydysrhythmia in newborns.[13] It occurs in 1.5–4 of every 1,000 neonates and represents 13 percent of all childhood arrhythmias.[27,30]

## Etiology

- Cardiac defects such as:
  - Ebstein's anomaly
  - Transposition of the great vessels
  - Tricuspid atresia
- Conduction abnormalities such as Wolff-Parkinson-White (WPW) syndrome
- Myocarditis
- Systemic infections
- Thyrotoxicosis

## Treatment

Treatment of SVT is accomplished in three phases: termination, initial therapy, and maintenance.[12] The severity of the symptoms also influences treatment decisions. If the infant does not demonstrate congestive heart failure or has only mild symptoms such as tachypnea, the dysrhythmia may be terminated by placing ice over the infant's face; yet this must be done cautiously to ensure proper ventilation.[9,15,29] Also, it is important to monitor for reflex bradycardia.

Symptomatic infants (shock or congestive heart failure) warrant rapid treatment, including vagal maneuvers and IV adenosine, followed by cardioversion (Chapter 4) or transesophageal pacing.[12,15,16,29,31]

It is important to note that repeated doses of adenosine should be avoided, as they may result in perpetuation of the arrhythmia. As a result, initial therapy may also include intravenous infusion of esmolol, procainamide, or amiodarone.[12] SVT tends to be a recurring problem requiring maintenance (oral) therapy. First-line maintenance therapy may include digoxin or β-blockers such as propranolol or sotalol.[32] Second-line medications for refractory SVT may include amiodarone, flecainide, propranolol, or sotalol. *Note:* Digoxin and calcium channel blockers should be avoided when SVT is caused by WPW syndrome, as they can increase the conduction rate and lead to ventricular fibrillation or sudden death. Surgical ablation, a method used to destroy the arrhythmia's focus, may be necessary in cases where medical therapy is not successful.[15]

### Complications

With sustained SVT, decreased cardiac output and congestive heart failure can occur.[9]

## JUNCTIONAL RHYTHMS

Junctional rhythms, also known as nodal rhythms, are stimulated by the AV node, rather than the SA node. The wave of depolarization travels backward to stimulate the atria and forward to stimulate the ventricles. This backward, or retrograde, atrial depolarization may result in an inverted P wave (P is below the isoelectric line), absent P waves that are hidden in the QRS complex, retrograde P waves (a P wave occurring after the QRS when ventricular conduction occurs before atrial depolarization), or a very short PR interval.

### Nodal Rhythm

A nodal rhythm (Figure 3-13) may be missed because all the waveforms appear normal except for an

absent or abnormal P wave. The bradycardia that occurs with hypoxic episodes is often a junctional bradycardia (no P waves) rather than a sinus bradycardia.

## Parameters

- Absent or inverted P waves, possible retrograde P waves (following QRS complexes), or short PR interval
- Normal, bradycardic, or tachycardic rate
- Normal QRS interval
- Normal PP and RR intervals

## Incidence

Nodal rhythms may be present in 18–70 percent of premature infants, and up to 28 percent of normal newborns.[16]

## Etiology

- May be normal in the newborn
- May be associated with congenital heart disease
- Hypoxia

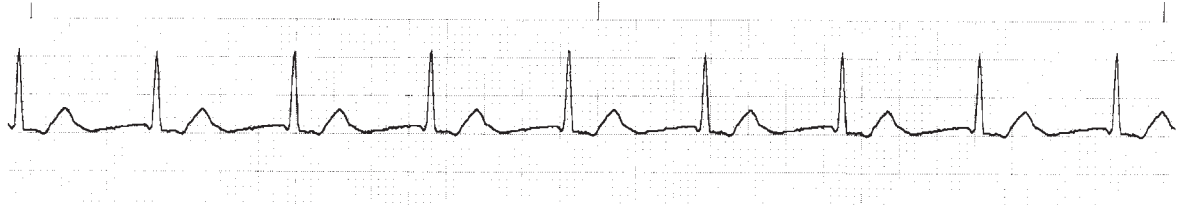

***Figure 3-13 Nodal rhythm.***

### Treatment

Monitor asymptomatic patients. Treat the cause if condition is the result of hypoxia. In some cases of symptomatic junctional bradycardia, atropine or isoproterenol may increase the heart rate and encourage the SA node to take over.[33]

### Complications

There may be decreased cardiac output if the arrhythmia is associated with high or low blood pressure.

## Junctional Ectopic Tachycardia

Junctional ectopic tachycardia (JET) (Figure 3-14) is a type of supraventricular tachycardia that occurs almost exclusively after surgery for congenital heart disease. There is also a rare congenital form of JET. JET is caused by increased automaticity of the AV node and bundle of His, resulting in an increased ventricular rate. The atria are frequently unaffected and continue to function

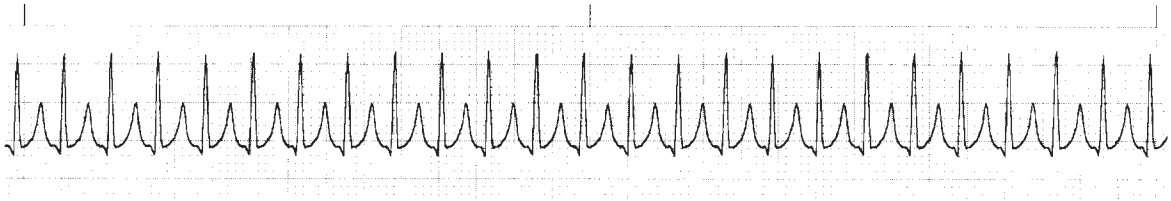

*Figure 3-14 Junctional ectopic tachycardia.*

normally; thus, JET may be the only form of SVT in which the ventricular rate can exceed the atrial rate.

### Parameters

- Gradual increase in heart rate over several hours
- Possibility of hidden P waves or P waves after QRS complexes
- Possibility of inverted waves (when visible)
- Usually transient (lasting a few hours to a few days)
- Variable ventricular rate, with periods where it speeds up and slows down
- Ventricular rate of 100 bpm or greater (up to 370 bpm)

### Incidence

JET is rare, occurring primarily after surgery near the AV junction.[15] The highest postoperative incidence is in the very young neonate who has had long or difficult surgeries and who requires inotropic support after bypass.[21] With congenital JET, 50 percent of patients have a positive family history, and the mortality rate is 35 percent.[21]

### Etiology

- Enhanced automaticity of the AV node or Purkinje system
- Related to surgical irritation
- A rare congenital form may be noted prenatally or in the first few weeks of life and is probably hereditary

### Treatment

If the patient is asymptomatic and the heart rate is less than 170 bpm, no treatment may be required. However, with faster rates, there may be acute hemodynamic consequences requiring treatment. Initial treatment of postoperative JET includes correcting contributing factors such as electrolyte abnormalities and anemia, optimizing sedation, and correcting fever.

Amiodarone is the primary treatment. If the infant does not respond to amiodarone, additional pharmacologic treatment can include dexmedetomidine, magnesium, procainamide, or esmolol. Temporary pacing, cooling, or ablation may also be indicated. The congenital form is treated with amiodarone or β-blockers. Ablation is indicated for refractory JET or heart failure.[34]

### Complications

Complications include severe hemodynamic compromise as a result of the accelerated rate. Congestive heart failure and acute cardiovascular collapse may occur.

## VENTRICULAR RHYTHMS

Ventricular rhythms are associated with problems originating in the ventricles. Because the QRS complex reflects ventricular depolarization and contraction, the QRS complex is abnormal in these dysrhythmias.

### Premature Ventricular Contractions

Premature ventricular contractions (PVCs) (Figure 3-15 a & b) originate from an ectopic focus or foci in the ventricles. They occur early in the cycle, before the SA node can fire. The impulse does not follow the usual conduction system. Thus conduction is slow, resulting in a wide QRS complex.[2,9] PVCs can have the same shape and appearance each time they are present, or they can have different shapes resulting from different morphologies. If they are all the same shape, they are termed unifocal PVCs.[6,17] If they have different shapes, they are known as multifocal PVCs. PVCs may also be labeled on the basis of their frequency: For example, bigeminy denotes a rhythm in which a PVC occurs every other beat; trigeminy, every third beat.

### Parameters

- ♥ Normal baseline rhythm
- ♥ Periodic early beats

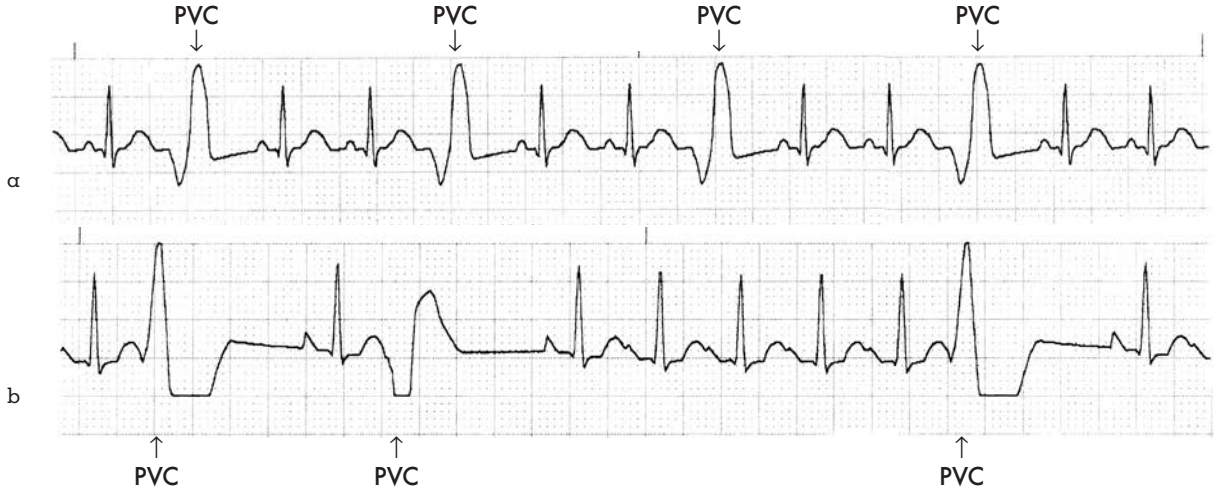


***Figure 3-15 a & b Premature ventricular contractions.***

a. In the first rhythm strip, the PVCs are all the same in appearance; therefore, they probably are being elicited from the same focus (unifocal).

b. The PVCs in the second strip are not alike in appearance and are elicited from more than one focus (multifocal).

- Early beats characterized by wide QRS complexes
- QRS complexes are usually taller and deeper than normal
- Compensatory pause before the next normal beat
- Early beat may not perfuse; thus arterial blood pressure (BP) tracings may be dampened during the PVC

## Incidence

PVCs are reported to occur in as many as 33 percent of newborns, but they disappear within the first four to eight weeks of life.[35]

## Etiology

- May be normal
- Acidosis
- Cardiac tumors[16]
- Decreased cardiac output
- Electrolyte or metabolic disturbances
- Irritation from invasive catheters
- May be associated with caffeine, digoxin, or antiarrhythmic agent administration[14]
- Myocarditis[26]
- Prolonged QT syndrome
- Systemic infection[36]
- Underlying cardiac disease

## Treatment

If PVCs are infrequent or the infant is asymptomatic, treatment may not be required. If the infant is symptomatic, treat the cause and give lidocaine as needed. Lidocaine may also be given if the PVCs are frequent (more than ten per minute or in runs of three or more in a row) or if the R wave occurs very close to or at the same time as the T wave (this R-on-T phenomenon can result in ventricular tachycardia).

## Complications

If frequent, PVCs may compromise cardiac output. They may progress to ventricular tachycardia or ventricular fibrillation.

## Long QT Syndrome

Prolonged QT intervals, also known as Long QT syndrome (LQTS) have a QT interval of >0.44 seconds as the key identifying factor (Figure 3-16).[7] Although this is a syndrome that may be genetic or acquired, it is included in the **Ventricular Rhythms** section because of its likelihood to progress to more serious ventricular arrhythmias and sudden death. The genetic form is associated with alterations in potassium and sodium involved in the control of ventricular repolarization.[37] The acquired form may be associated with reactions to certain medications.

The QT interval is commonly measured from the onset of the QRS to the end of the T wave. The duration of the QT interval varies as the heart rate changes. As a result, Bazett's formula is commonly used to measure the corrected QT interval:[12]

$$QTc = \frac{QT \text{ (in seconds)}}{\sqrt{RR \text{ (preceding RR interval in seconds)}}}$$

### Parameters

- Normal P and QRS waveforms
- Normal rate
- QT interval exceeds the normal range
- T wave may be notched, biphasic, or inverted

### Incidence

A mild, benign prolongation may be present after a stressful birth, and usually resolves within 72 hours.[12]

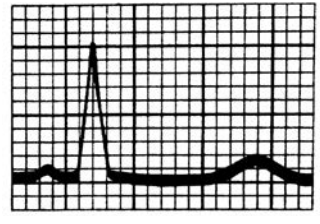

***Figure 3-16 Prolonged QT interval with hypocalcemia.***

Physiologic prolongation of the QT interval may also be present in 2.5 percent of healthy newborns until six months of age. LQTS is reported to be an uncommon rhythm disturbance in infants;[7] the European Society of Cardiology Task Force estimates an incidence of 1:3,000–5,000.[37] Congenital LQTS is associated with sudden infant death syndrome (SIDS), with gene variants found in 9.5 percent of SIDS victims.[12]

## Etiology[7]

- Antiarrhythmic medications such as quinidine and procainamide
- Jervell and Lange-Nielson syndrome
- Romano-Ward syndrome

## Treatment

Typical presenting symptoms in neonates include syncope, seizures, and sudden death, thus early diagnosis is essential, especially in cases with a known family history of prolonged QT syndrome.[14] β-blockers are the initial treatment of choice. Propranolol and nadolol are most commonly used and are effective in eradicating the arrhythmia in the majority of cases. Compliance must be stressed and dosages adjusted with weight throughout childhood. Patients with persistent syncopal episodes may require internal defibrillator placement. A permanent pacemaker may be indicated in patients who have a prolonged QT interval along with a second degree AV block.[7,19]

## Complications

Complications include seizures, ventricular arrhythmias, and heart blocks. In particular, LQTS is characterized by delayed ventricular repolarization and an increased risk of ventricular arrhythmias, particularly a polymorphic type of ventricular tachycardia known as torsades de pointes (discussed in the next section), which can lead to ventricular fibrillation and sudden death.[12]

## Ventricular Tachycardia

Ventricular tachycardia—also known as V tach or VT—originates from a focus in the ventricle (rather than from the SA node). When the ventricles do not receive an impulse from above, they will fire at their own intrinsic rate (50–60 bpm). As the name implies, V tach is a rhythm that is faster than the normal ventricular rate (Figure 3-17). It is a dangerous rhythm because rapid ventricular contractions prevent the ventricles from completely filling and emptying with each contraction. This rhythm can greatly reduce cardiac output.

Ventricular tachycardia may rapidly progress to more lethal arrhythmias. Always assume that a wide QRS tachycardia is V tach until proven otherwise.

Torsades de pointes is a type of V tach. It's name literally means "twisting of the points." In this type of VT, the QRS complexes gradually change from one ventricular focus to another. There may be a normal-width QRS complex between two episodes of VT marked by wide QRS complexes. The QRS complex may change completely every five to ten beats.

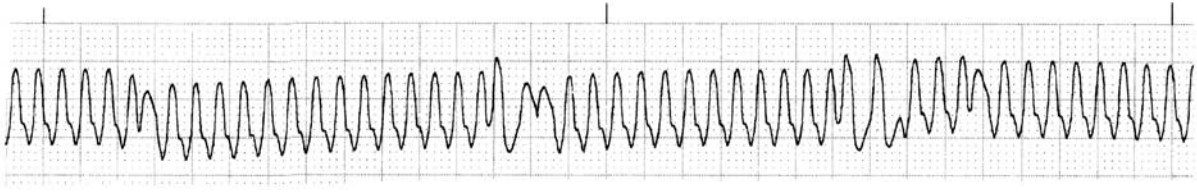

***Figure 3-17 Ventricular tachycardia.***

### Parameters

- Altered or widened QRS complexes without P waves (P waves are hidden in QRS complexes.)
- QRS complex has a sawtooth or flutter appearance
- May look like several PVCs in a row (Three or more PVCs in a row are called a short run of ventricular tachycardia.)
- Heart rate of 120–240 bpm
- RR intervals may be regular
- Inadequate ventricular contraction and cardiac output

### Distinguishing VT from SVT

Those who are just learning to interpret ECGs can sometimes find it difficult to distinguish ventricular tachycardia from supraventricular tachycardia (Figures 3-18 and 3-19). Table 3-1 compares the two dysrhythmias. ***Remember:*** *Always assume that a tachycardia with a wide QRS is ventricular tachycardia. Treat it as an emergency until proven otherwise!*

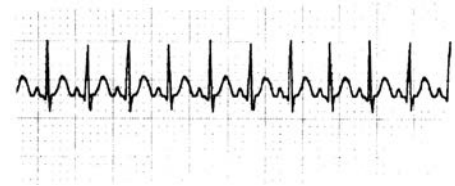

***Figure 3-18 Supraventricular tachycardia.***

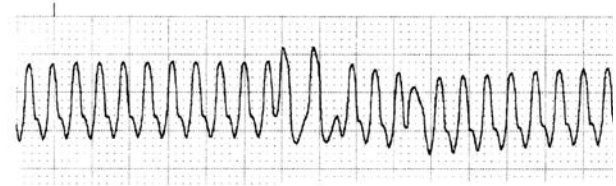

***Figure 3-19 Ventricular tachycardia.***

*Table 3-1 Comparison of Supraventricular Tachycardia and Ventricular Tachycardia*

| Characteristic | SVT | VT |
|---|---|---|
| Occurrence in infants | Frequent | Uncommon |
| P waves | May be abnormal | None present |
| QRS complex deflection* | Usually normal | May be opposite normal |
| QRS complex width | Usually normal or narrow | Wide or different |
| Hemodynamics | Tolerated better initially than VT | Altered |
| Vagal maneuvers as treatment | May slow or stop SVT | Not effective |

* Deflection refers to location of the QRS complex above or below the isoelectric line. Normal deflection will depend on which lead on the cardiac monitor is selected.

## Incidence

Ventricular tachycardia is rare in neonates.[15] Of infants who have episodes of ventricular tachycardia, 85 percent have an abnormal heart. The majority have cardiac tumors, particularly rhabdomyomas, cardiac fibromas, or Purkinje cell tumors.[12] Another 10 percent have myocarditis.[35] After cardiac surgery, ventricular tachycardia may occur in 10 to 15 percent of infants, and 5 to 10 percent of those may experience sudden death.[18]

## Etiology

- Acidosis
- Electrolyte imbalance
- Cardiac anomalies

- Cardiac tumors
- Cardiomyopathy
- Drug toxicity
- Exposure to some antiarrhythmic medications
- Hypoxia
- Mitral valve prolapse
- Myocardial fibrosis
- Myocarditis
- "Tet" spells, which occur with tetralogy of Fallot, may include cyanosis, irritability, pallor, flaccidity, tachypnea, and/or loss of consciousness[13]
- Tumors of the ventricle

### Treatment

Treatment is based on the presence or absence of a pulse: If the infant is symptomatic and has a pulse, lidocaine therapy, sedation, and cardioversion (Chapter 4) are indicated. Ventricular tachycardia without a pulse is treated the same as ventricular fibrillation: with CPR and defibrillation (Chapter 4).[31] Antiarrhythmic drugs such as lidocaine (usually the first drug of choice),[10] flecainide, or amiodarone may be used.[12] Treat the cause. For infants with recurrent torsades de pointes, magnesium sulfate infusion is the drug of choice. Overdrive pacing is also effective.[14]

### Complications

Complications include severely compromised cardiac output and progression to ventricular fibrillation.

## Ventricular Fibrillation

Ventricular fibrillation—also known as V fib or VF—is created by rapid stimuli from many ventricular ectopic foci (Figure 3-20). This stimulation causes a chaotic twitching or quivering of the ventricles. There is no effective cardiac output because the heart muscle is "quivering" rather than pumping.

## Parameters

- Irregular, coarse ECG pattern without identifiable waves
- No pulse or audible heartbeat

## Incidence

Ventricular fibrillation is rare in infants.[15] Most infants with ventricular fibrillation have abnormal hearts.[25]

## Etiology[14]

- Acidosis
- Cardiac anomalies
- Drug toxicity
- Electrolyte imbalance
- Hypoxia
- Myocarditis

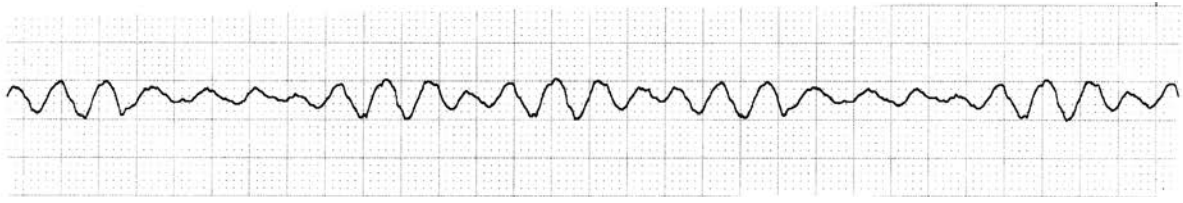

***Figure 3-20 Ventricular fibrillation.***

### Treatment

Immediate resuscitation, including CPR and defibrillation (Chapter 4), is indicated. Lidocaine drug therapy may be indicated. Treat the cause. An implantable or external automated defibrillator can be used for ventricular fibrillation.[38]

### Complications

Ventricular fibrillation is lethal.

## HEART BLOCKS

In a heart block, the electrical impulse is blocked at some point in the cardiac cycle. This may simply prolong the time required for the impulse to pass through the cardiac cycle, or it may prevent the impulse from traveling from the atria to the ventricles.

### Bundle Branch Block

Under normal circumstances, the right and left ventricles are depolarized simultaneously. This results in a single QRS complex. Bundle branch block (BBB) is caused by a slowing of the impulse in either the right or left bundle branch, prolonging conduction through the ventricles. Most situations of BBB cause depolarization of one ventricle before the other. This results in two QRS complexes that are often merged into one QRS with a characteristic "rabbit ear" appearance (Figure 3-21).

Bundle branch blocks may occur in either branch (right or left) and in specific divisions of each bundle. A 12-lead ECG printout (Chapter 5) is needed to determine whether the block is occurring in the left or right bundle branch. Left bundle branch block (LBBB) is diagnosed by assessing the R wave in leads $V_5$ and $V_6$; right bundle branch block (RBBB) is diagnosed by assessing leads $V_1$ and $V_2$.[2,29] Identification of the specific location

of the block is an advanced interpretation technique not discussed in this text because it does not necessarily affect treatment or outcome.

### Parameters

- Notched R waves or R waves with a "rabbit ear" appearance
- Widened QRS complexes

### Incidence

Bundle branch block is relatively common in the pediatric population. It is estimated to occur in 0.2 percent of normal children.[6] LBBB is infrequent and usually occurs only after cardiac surgery of the aortic valve or subvalvular area. RBBB occurs in 32 to 44 percent of children after ventricular septal defect (VSD) repair,[39] and in 59 to 100 percent of children after repair of tetralogy of Fallot.[18]

### Etiology

- Possible association with Ebstein's anomaly, Kearns-Sayre syndrome, and neuromuscular disorders
- Electrolyte imbalance

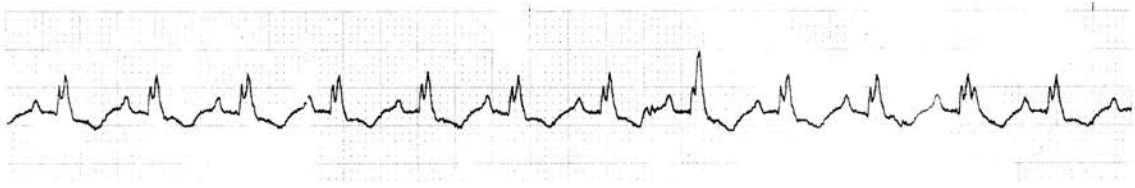

***Figure 3-21 Bundle branch block.***

- Heredity
- Myocarditis
- Post-cardiac surgery or transplantation
- Volume overload

### Treatment

Follow BBB conservatively.

### Complications

Bundle branch block may progress to a higher degree block or to complete heart block.[29]

## First Degree Heart Block

First degree heart block (Figure 3-22) occurs at the AV node and causes a longer than normal delay between atrial and ventricular stimulation. Normally, a short pause follows atrial depolarization to allow the ventricles time to fill with blood before contracting. This pause is reflected in the PR interval. A prolonged PR interval indicates first degree heart block. Diagnosis requires a printout of the rhythm strip and measurement of the PR interval.

### Parameters

- Prolonged PR interval, generally >0.11–0.14 seconds, based on age and HR[6]
- Otherwise normal ECG appearance and measurements

### Incidence

First degree heart block may occur in up to 6 percent of neonates.[40]

### Etiology

- May be normal in the neonate
- Large ASD
- AV canal[10]
- Digitalis effect
- Ebstein's anomaly[6]

- Electrolyte abnormalities
- Hypothermia
- Increased vagal tone
- Infectious or inflammatory conditions
- Patent ductus arteriosus[23]
- Pulmonary stenosis[23]
- Surgical injury to the AV node or Purkinje system
- Valve disorders

## Treatment

Evaluate the hemodynamic status of the infant and treat the cause. Follow first degree heart block conservatively.

## Complications

There usually are no complications for this condition. Complications may be associated with the cause.

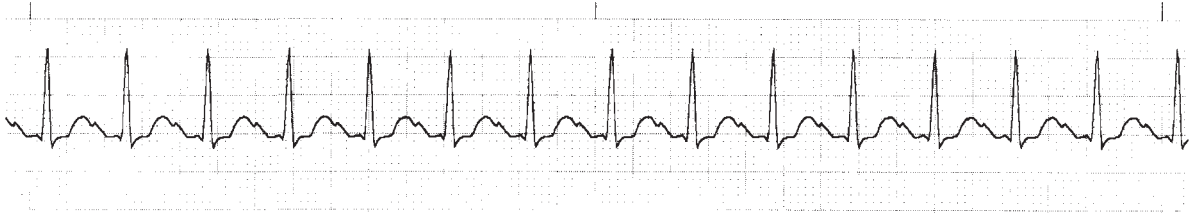

***Figure 3-22 First degree heart block.***

## Second Degree Heart Block

There are two types of second degree heart block: Mobitz I and Mobitz II.

### Mobitz I

Also known as Wenckebach block, Mobitz I (Figure 3-23) may be normal in infants. It is marked by a progressive lengthening of the PR interval until one P wave is not conducted.

#### Parameters

- Progressive lengthening of PR interval, culminating in nonconduction of the beat (P wave is not followed by a QRS complex.)
- Normal P wave and QRS complex in preceding and following cycles
- Otherwise normal rate

#### Incidence

Mobitz I occurs rarely in infants.[40]

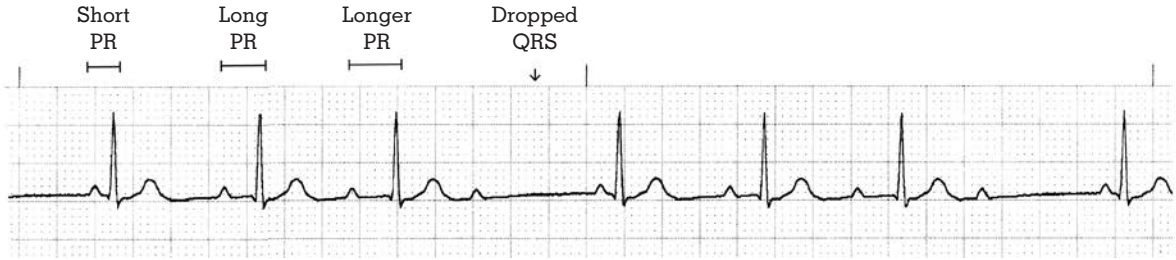


*Figure 3-23 Second degree heart block: Mobitz I.*

### Etiology

- ♥ May be normal in infants, especially during sleep or with increased vagal tone
- ♥ Disease or damage to the AV node or Purkinje system
- ♥ Infectious or inflammatory conditions
- ♥ Structural heart disease

### Treatment

Evaluate the hemodynamic status of the infant, and treat the cause. Follow Mobitz I second degree heart block conservatively.

### Complications

There usually are no complications of Mobitz I.

## Mobitz II

Mobitz II (Figure 3-24) is the more serious form of second degree heart block because it can abruptly

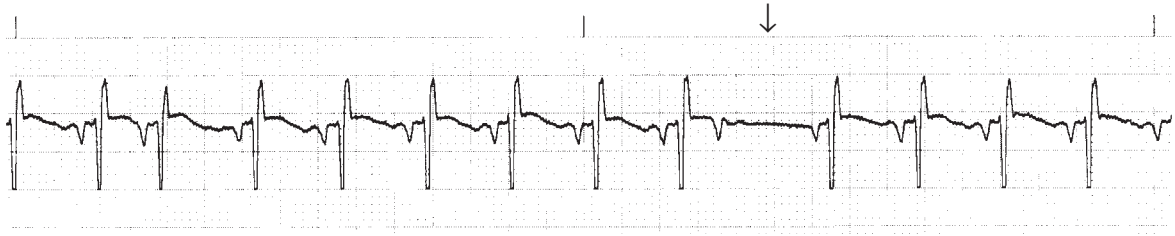

***Figure 3-24 Second degree heart block: Mobitz II.***

progress to complete (third degree) heart block. In Mobitz II, the impulse that triggers ventricular depolarization (shown by the QRS complex) is not conducted for one or two beats after a normal P wave.

### Parameters

- Normal P waves
- Normal PR intervals
- "Dropped" QRS complexes (complex does not occur after all P waves); usually occur in a cyclic fashion (2:1, 3:1, 4:1)
- Varied RR intervals (due to dropped QRS)

### Incidence

Mobitz II is uncommon in neonates.[10,27]

### Etiology[14]

- Associated with prolonged QT syndrome
- Congenital heart disease
- Diffuse disease of the conduction system
- Hypocalcemia
- Infection or inflammation
- Surgical injury
- Systemic disorders
- Dropped QRS

### Treatment

Treat the cause. Mobitz II may require pacing if it is caused by surgical damage or if the infant is having symptoms such as bradycardia, hypotension, or an altered state of consciousness. Symptomatic bradycardias are usually responsive to atropine, epinephrine, or isoproterenol until temporary pacing is available.[12] Treatment should be aggressive if associated with prolonged QT syndrome.[14]

### Complications

Mobitz II often progresses to third degree (complete) heart block.

## Third Degree Heart Block

In third degree heart block (Figure 3-25)—also known as complete heart block or third degree AV block—there is a complete blockage of electrical impulses between the atria and the ventricles. The atria are being stimulated and are contracting at their own rate (90–180 bpm), while the ventricles are being stimulated from a ventricular focus that causes contraction at another rate, usually the ventricular intrinsic rate (50–60 bpm).

### Parameters

- Faster atrial rate than ventricular rate (because of the normal intrinsic rates of each area)
- Slow ventricular rate of 50–80 bpm
- No relationship between P waves and QRS complexes on the ECG
- Possibility of wide QRS complexes

### Incidence

Third degree heart block is reported to occur in 1/15,000–20,000 live births.[12] Most occurrences are

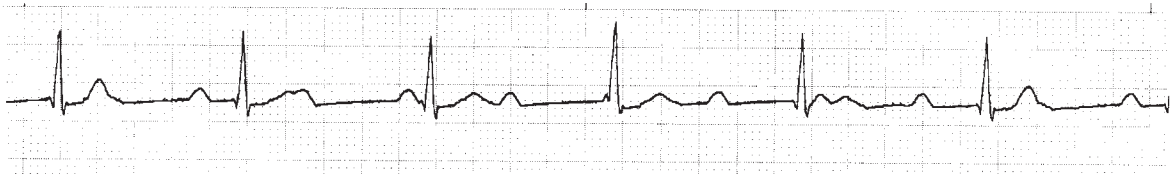

***Figure 3-25 Third degree heart block.***

associated with maternal antibodies that cross the placenta during the critical stage of fetal cardiac tissue development and disrupt normal formation of the conduction system. Other infants (25–35 percent) with complete heart block have structural cardiac anomalies.[27] Third degree heart block is also reported to occur in 1–2 percent of patients after cardiac surgery, especially VSD and tetralogy repairs.[18]

## Etiology

- Acute increase in vagal tone
- Acute rheumatic fever
- Congenital heart defects, such as atrial or ventricular septal defects, transposition, or AV canal[10]
- Digoxin toxicity
- Heredity
- Infections
- Maternal collagen vascular diseases such as lupus erythematosus[6,10] or Sjögren syndrome[12,14]
- Myocarditis
- Myopathies
- Post-surgical manipulation
- Trauma

## Treatment

In symptomatic patients, such as those who are hypotensive or have an altered level of consciousness, cardiac compressions may be required to maintain cardiac output if the ventricular response is very slow. Temporary/transesophageal or permanent pacing is the treatment of choice for symptomatic cases.[19] Isoproterenol may be indicated in some situations and will help to increase cardiac output.[29] Atropine may be useful if the cause is associated with increased vagal tone

rather than with AV node dysfunction.[29] Permanent pacemaker treatment is indicated if the block is persistent and due to surgical causes.

Infants with congenital forms, such as those associated with lupus erythematosus, are usually asymptomatic and may not require immediate treatment.[14] However, prophylactic pacing decreases the incidence of complications in infants with congenital heart block.[19] Most will require a pacemaker sometime during childhood or adolescence.[12] Pacing is indicated for a heart rate of less than 55 bpm. If congestive heart failure is present, pacing is indicated with a heart rate less than 70 bpm.[19]

### Complications

Complications include inadequate cardiac output, congestive heart failure, and possible death. The slow rate creates a potential for escape rhythms.

## PACED RHYTHMS

As mentioned in previous sections, pacemakers are often indicated in the treatment of dysrhythmias. Pacemakers deliver electrical impulses to the myocardium in an effort to stimulate depolarization when it does not occur independently. Pacemaker impulses are seen on the ECG rhythm strip as very narrow, spiked waveforms. The location of these waveforms is based on the type of pacemaker in use. There are a variety of pacemakers:

- **Temporary** pacemakers are utilized for dysrhythmias believed to be short term, or as a bridge until permanent pacemaker placement can be done. The transesophageal method is the primary method for temporary pacing and is usually limited to atrial stimulation, because there are difficulties associated with ventricular lead placement.[19]
- **Permanent** pacemakers are indicated when there is irreversible dysfunction or damage to the conduction

system. Permanent pacemakers are available for even the smallest of infants.

**Asynchronous** pacemakers provide an electrical stimulus to the heart at a set rate, regardless of spontaneous cardiac activity. These pacers are seldom used because of their potential to produce lethal dysrhythmias with an improperly timed electrical stimulus.

**Demand** pacemakers sense the electrical activity of the heart and deliver an electrical stimulus only when one is not sensed from the myocardium in a specified time frame.

**Atrial** pacemakers provide impulses to stimulate atrial depolarization only. They can be useful in treating sinus bradycardias in which the AV conduction system is intact; sick sinus syndrome; and some atrial arrhythmias. With an atrial pacemaker, the pacemaker spike is visible just prior to the P wave. (Figure 3-26).

**Ventricular** pacemakers provide impulses that stimulate only ventricular depolarization. This type of pacing may be useful in an infant with a heart block who has normal sinus node function. With a ventricular pacemaker, the ECG may have a normal sinus P wave followed by a pause, a pacemaker spike, and then a QRS complex. Or the P wave may be absent, with only a pacemaker spike followed by a QRS complex (Figure 3-27).

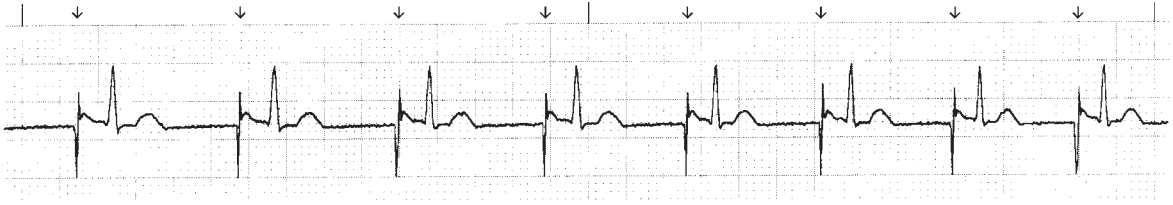

*Figure 3-26 Atrial pacemaker spikes.*

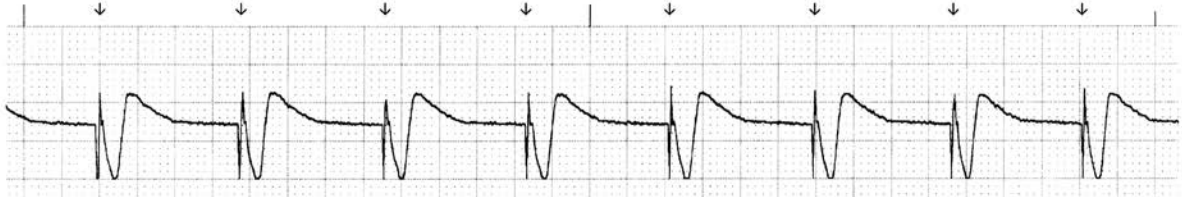

*Figure 3-27 Ventricular pacemaker spikes.*

**Dual-chamber** pacemakers have the ability to provide impulses that can stimulate both the atria and the ventricles. These pacemakers may be used in the treatment of bradycardia, heart block, SA node disease, and atrial arrhythmias. With this type of pacemaker, a pacer spike is seen prior to the P wave and a second spike seen prior to the QRS complex (Figure 3-28).

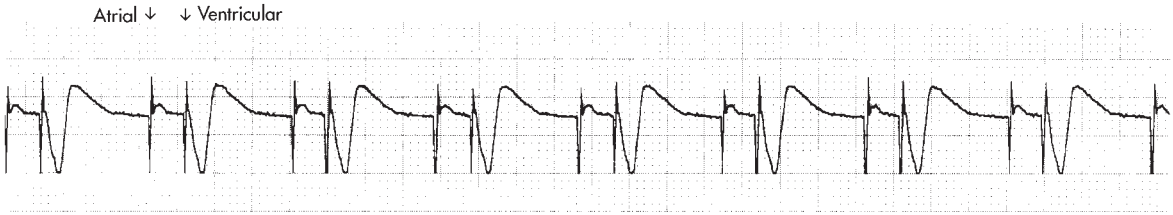


*Figure 3-28 Dual-chamber pacemaker spikes.*

## ECG CHANGES AND ELECTROLYTE ABNORMALITIES

Consistency of wave contour is an important factor in evaluating an ECG strip. Wave contours are particularly important in helping to determine hyperkalemia and hypokalemia.

In hyperkalemia, the most common ECG change is a peaked T wave (Figure 3-29a). Additional findings may include an elevated ST segment (Figure 3-29b), a

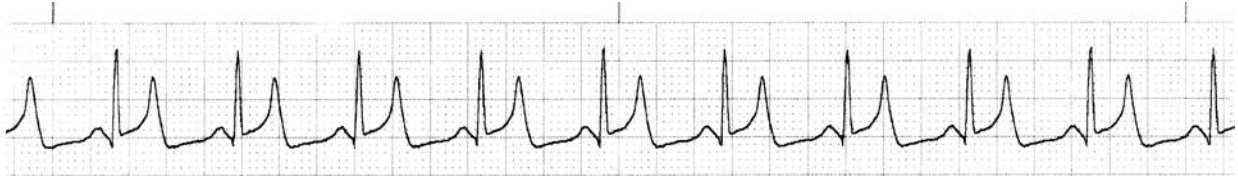

*Figure 3-29a Hyperkalemia, marked by peaked T waves.*

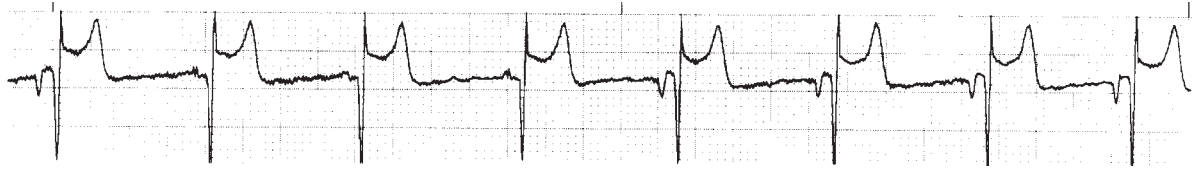

*Figure 3-29b Hyperkalemia, marked by elevated ST segments.*

widened or flattened P wave, a widened QRS complex, and possible PVCs and other ventricular arrhythmias.

In hypokalemia, the T wave is generally flat or inverted (Figure 3-30). There may also be another wave after the T wave, known as a U wave, which is wide and prominent (Figure 3-31). The PR interval may be prolonged, and the QRS may be wide. Untreated hypokalemia can lead to more serious arrhythmias.

Hypercalcemia and hypocalcemia may also cause ECG changes. The QT interval may be shorter than normal with hypercalcemia (Figure 3-32a), or it may be prolonged with hypocalcemia (Figure 3-32b).

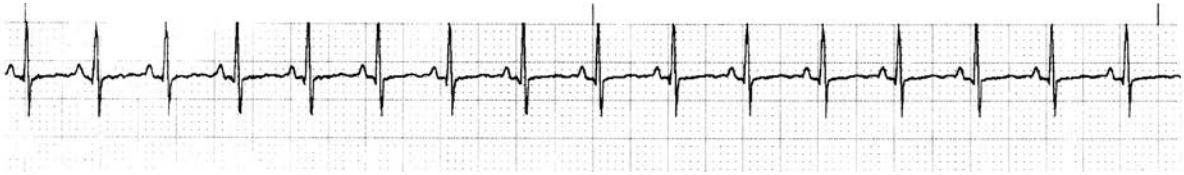

***Figure 3-30 Hypokalemia marked by flat T waves.***

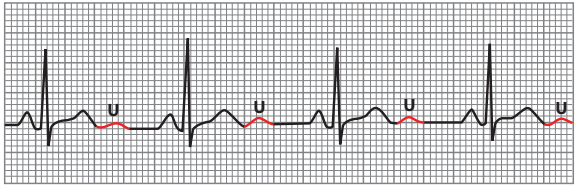


*Figure 3-31 Hypokalemia marked by U waves.*

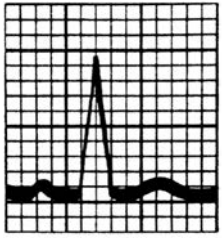

*Figure 3-32a Changes in QT interval with hypercalcemia.*

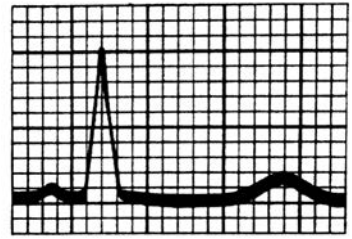

*Figure 3-32b Changes in QT interval with hypocalcemia.*