DYSRHYTHMIAS

I. BASIC PRINCIPLES OF CARDIAC CONDUCTION DISTURBANCES

A. Standard ECG and rhythm strips

1. Recordings are obtained at a paper speed of 25 mm/sec.
2. The vertical axis measures distance; the smallest divisions are 1 mm long ×1 mm.
3. The horizontal axis measures time; each small division is 0.04 sec/mm.

B. Normal morphology

Courtesy of Dr. Michael McCrea

1. P wave = atrial depolarization
   a. Upright in leads I, II, III, aVL, and aVF; inverted in lead aVR
   b. Measures <0.10 seconds wide and <3 mm high
   c. Normal PR interval is 0.12–0.20 seconds.
2. QRS complex = ventricular depolarization
   a. Measures 0.06-0.10 seconds wide
   b. Q wave is <0.04 seconds wide and <3 mm deep; the Q wave is abnormal if it is >3 mm deep or >1/3 of the QRS complex.
   c. R wave ≤7.5 mm high
3. QT interval varies with rate and sex but is usually 0.33–0.42 seconds; at normal heart rates, it is normally <1/2 the preceding RR interval.
4. T wave = ventricular repolarization
   a. Upright in leads I, II, V_3–V_6; inverted in aVR
   b. Slightly rounded and asymmetric in configuration
   c. Measures ≤5 mm high in limb leads and ≤10 mm high in the chest leads
5. U wave = a ventricular afterpotential
   a. Any deflection after the T wave (usually low voltage)
   b. Same polarity as the T wave
   c. Most easily detected in lead V_3
   d. Can be a normal component of the ECG
   e. Prominent U waves may indicate one of the following:
      (1) Hypokalemia (<3 mEq/L)
      (2) Hypercalcemia
      (3) Therapy with digitalis, phenothiazines, quinidine, epinephrine, inotropic agents, or amiodarone
      (4) Thyrotoxicosis
   f. Inverted (negative) U waves may indicate one of the following:
      (1) Acute coronary ischemia
      (2) Ventricular strain, dilation, or overload
      (3) Hypertension
      (4) Intracranial or subarachnoid hemorrhage

C. Causes of abnormal morphologies
1. Hypothermia: core temperature <95°F (35°C)
   a. ECG findings
      (1) "J wave" (also referred to as an "Osborn wave"): a broad, upright deflection at the end of an upright QRS complex
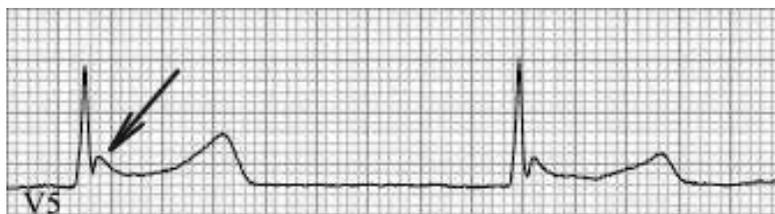
Courtesy of Dr. Michael McCrea

    (2) Conduction delays: PR, QRS, and QT intervals may all be prolonged. QT interval is prolonged primarily because of selective prolongation of the ST segment.

    (3) Dysrhythmias: sinus bradycardia and atrial fibrillation with a slow ventricular response are the most commonly encountered in this setting; the risk of developing dysrhythmias increases as the core temperature falls below 86°F (30°C); at core temperatures below 77°F (25°C), spontaneous ventricular fibrillation and asystole may occur. (These patients must be handled gently, because dysrhythmias are easily introduced).

  b. Management of hypothermia-induced dysrhythmias

    (1) Most usually require only supportive therapy, because the dysrhythmias resolve spontaneously with rewarming.

    (2) Cardiac arrest

      (a) In profoundly hypothermic patients who appear to be in cardiac arrest, palpating pulses may be extremely difficult.

      (b) If unsure, began CPR without delay.

    (3) Ventricular fibrillation

      (a) Often refractory to defibrillation attempts until the patient is rewarmed

      (b) Defibrillation should be attempted with up to three shocks but, if unsuccessful, CPR and rapid rewarming measures should be instituted. Further attempts at defibrillation should be withheld until the patient's temperature rises above 86°F (30°C).

      (c) As the myocardium rewarms, ventricular fibrillation may convert spontaneously or resolve in response to defibrillation.

      (d) Magnesium sulfate produces spontaneous conversion in these patients.

    (4) The role of ACLS medications, including vasopressors, in severe hypothermic patients in cardiac arrest is of uncertain value, and standard algorithms may be used.

    (5) If narcotic abuse is suspected, naloxone should be considered, because it may act on central opiate receptors to decrease the severity of hypothermia seen in overdoses.

    (6) In general, a patient is not considered "all dead" until "warm and dead," with warm being 95°F (35°C).

2. Hypokalemia

  a. Progressively more prominent U wave (best seen in V_3)

  b. Flattening of T wave (earlier) followed by inversion (later)

  c. Depression of ST segment

  d. Prominent P wave

  e. Prolongation of the PR and QT (U) interval

  f. Ventricular tachycardia/torsades

  g. In the presence of hypokalemia, susceptibility to digitalis toxicity and its associated dysrhythmias is increased.

3. Hyperkalemia
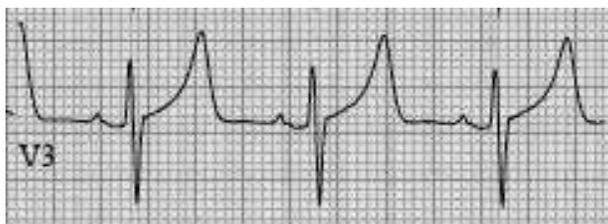
Courtesy of Dr. Michael McCrea

  a. Tall hyperacute T wave (earliest ECG finding)

  b. Prolonged PR interval

   c. Flattened or absent P wave

   d. Wide QRS complex that eventually blends with the T wave to assume a "sine wave" appearance

   e. Heart blocks

   f. QT interval normal or shortened

Table 1: Effects on ECG with Increasing Potassium Concentration

Potassium Concentration (mEq/L)	ECG Changes
5.5–6.5	Large amplitude T waves, peaked, tented symmetric
6.5–8.0	PR interval prolongation P wave flattening/disappearance QRS widening Conduction block with escape beats
>8.0	Sine wave appearance Ventricular fibrillation Asystole

4. Hypocalcemia

   a. Prolonged QT interval because of prolongation of the ST segment

   b. Terminal T wave inversion (less consistent finding)

   c. Ventricular dysrhythmias (including torsades de pointes)

5. Hypercalcemia

   a. Depressed ST segments, widened T waves, and shortened ST segments and QT intervals.

   b. Bradyarrhythmias may occur, with bundle-branch patterns that may progress to second-degree block or complete heart block.

6. Hypomagnesemia

   a. Prolonged PR and QT intervals

   b. Widened QRS complex

   c. ST segment abnormalities

   d. Flattened or inverted T waves (especially in the precordial leads)

   e. Ventricular dysrhythmias (premature ventricular contractions, ventricular tachycardia, torsades de pointes, ventricular fibrillation)

   f. Hypomagnesemia usually occurs in association with other electrolyte abnormalities (particularly hypokalemia), and many of the ECG findings are similar to those seen with hypokalemia and hypocalcemia (pictured above).

   g. In the presence of hypomagnesemia, susceptibility to digitalis toxicity and its associated dysrhythmias is increased.

7. Digitalis effects

   a. Sagging ST segment with its concavity directed upward (resembles a hockey stick)

   b. Short QT interval

   c. Flattened or inverted T wave

   d. Modestly prolonged PR interval

   e. These effects are especially prominent in the lateral leads and occur in most patients who are adequately digitalized; they are not an indication of digitalis toxicity.

8. Digitalis toxicity
   a. Pathophysiology: digitalis produces toxicity by
      (1) Poisoning the Na^+-K^+-ATPase pump → increased intracellular entry of Na^+ and Ca^{++} and egress of K^+ → increased excitability → ectopy and tachydysrhythmias
      (2) Increasing vagal tone and automaticity → decreased conduction in the AV node → bradydysrhythmias and AV blocks
   b. Factors that increase sensitivity to digitalis and predispose to toxicity
      (1) Electrolyte abnormalities (hyperkalemia or hypokalemia, hypomagnesemia, hypercalcemia)
      (2) Hypoxia
      (3) Metabolic alkalosis
      (4) Increasing age
      (5) Presence of underlying cardiac disease (ischemia, CHF, congenital heart disease)
      (6) Presence of chronic underlying systemic illness (COPD, renal failure, hypothyroidism)
      (7) Drug interactions (quinidine, calcium channel blockers, erythromycin, amiodarone, captopril, and ibuprofen)
   c. ECG findings
      (1) Premature ventricular contractions (most common arrhythmia), often bigeminal and multiform: the most common digitalis-induced rhythm disturbance
      (2) Junctional tachycardia (common)
      (3) Sinus bradycardia
      (4) Sinus tachycardia
      (5) Sinoatrial and AV nodal blocks
      (6) Sinus arrest
      (7) Torsades de pointes
      (8) Ventricular tachycardia
      (9) Ventricular fibrillation
     (10) Atrial tachycardias with AV block are very specific but not pathognomonic for digitalis toxicity
     (11) Nonparoxysmal junctional tachycardia
     (12) Atrial fibrillation with a slow ventricular response, ie, AV dissociation
     (13) Bidirectional ventricular tachycardia (highly suggestive of digitalis toxicity but rare)
   d. Clinical symptoms
      (1) Flu-like syndrome with profound malaise, anorexia, nausea, vomiting, and diarrhea
      (2) Visual disturbances (blurred vision, halos around objects, and yellow or green color aberrations)
      (3) Mental status changes, including confusion, drowsiness, and psychosis
   e. Acute digitalis toxicity is usually seen in young and otherwise healthy patients as a result of either accidental or intentional overdose; it is commonly associated with hyperkalemia, high digoxin levels, bradydysrhythmias, and AV blocks. Toxicity in these patients is most closely correlated with the degree of hyperkalemia (not the serum digoxin level). Chronic digitalis toxicity generally occurs in older cardiac patients with reduced renal function who are taking diuretics. These patients are usually normo- or hypokalemic, have digoxin levels that are minimally increased or normal, and most commonly have a ventricular dysrhythmia.
   f. Classic clinical scenario
      (1) Acute intoxication: A 3-year-old is brought in by his parents for evaluation after accidental ingestion of grandpa's "heart pills." Based on information obtained from the parents, the child has ingested 10.7 mg of digoxin sometime within the past 2 hours and has vomited twice. The cardiac monitor shows a junctional rhythm with sinus block and type I second-degree AV block; laboratory studies reveal a potassium level of 6.2 mEq/L, along with a markedly increased digoxin level of 61. The child is on no medications and is otherwise healthy.
      (2) Chronic intoxication: A 65-year-old woman with a past medical history of coronary artery disease, CHF, and renal insufficiency is brought in by ambulance for evaluation. Her medications include furosemide, digitalis, sublingual nitroglycerin, and baby aspirin. According to family members, she has become progressively more confused and weak over the past few days and has not been eating well. The ECG shows a regular wide complex tachycardia with alternating QRS polarity (bidirectional ventricular tachycardia) and laboratory studies reveal a digoxin level of 3.5 and a potassium concentration of 3.0 mEq/L.

g. Management

(1) IV line, oxygen, pulse oximeter, cardiac monitor

(2) Gastric lavage is contraindicated because of risk of vagal stimulation causing bradycardia or asystole.

(3) Administer multiple doses of activated charcoal to all patients with potentially toxic ingestions; activated charcoal prevents systemic absorption and, when multiple doses are given, enhances elimination by interrupting the prominent enterohepatic circulation of digitalis.

(4) Seek and treat factors that may contribute to digitalis toxicity.

    (a) Hypokalemia (correct cautiously in the presence of AV blocks; correction can actually exacerbate AV conduction defects)

    (b) Hyperkalemia is best treated with Fab fragments (digoxin-specific antibody fragments); do not administer calcium; it can potentiate cardiotoxicity.

    (c) Hypomagnesemia

    (d) Hypoxia

    (e) Dehydration

(5) Control tachydysrhythmias

    (a) Phenytoin and lidocaine are the drugs of choice for tachydysrhythmias.

    (b) Magnesium sulfate may also be useful in suppressing ventricular irritability.

    (c) Avoid cardioversion (digoxin decreases the fibrillatory threshold); restrict its use to situations of last resort and use the lowest possible energy level.

    (d) Avoid use of bretylium, Class IA antidysrhythmics (eg, procainamide, isoproterenol) and propranolol; these agents can exacerbate dysrhythmias and AV conduction disturbances.

(6) Manage symptomatic bradycardia or AV block with atropine. If atropine is unsuccessful, cardiac pacing (external or transvenous) may be used while waiting for Fab fragments to take effect. External pacing is preferred, because transvenous pacemaker insertion may induce tachydysrhythmias in these patients.

(7) Fab fragments

    (a) Should be administered to patients with:

        i. Ventricular dysrhythmias (ventricular fibrillation, ventricular tachycardia)

        ii. Symptomatic bradycardias unresponsive to atropine

        iii. Hyperkalemia (K^+ >5.0 mEq/L) secondary to digitalis intoxication

        iv. Coingestions of cardiotoxic drugs (β-blockers, cyclic antidepressants)

        v. Large, potentially lethal digitalis intoxications

        vi. Ingestions of plants known to contain cardiac glycosides (oleander, lily of the valley) with severe dysrhythmias

    (b) Fab fragments bind free digoxin in the vascular and interstitial spaces and form an inert compound that is eliminated by the kidneys. Treatment rapidly corrects conduction defects, ventricular dysrhythmias, and hyperkalemia.

    (c) Dosage

        i. If the serum digoxin level or total amount of digoxin ingested is known, the formulas found in the package insert can be used to calculate the number of vials of Fab fragments to be administered.

        ii. If the amount of digoxin ingested is unknown, the initial dose of Fab fragments should be 5–10 vials (titrated incrementally).

    (d) After administration of Fab fragments, conventional assays for determining digoxin levels (which measure both bound and unbound digoxin) are unreliable for at least a week.

Table 2: ECG Findings in Various Conditions

Condition	ST	PR	QRS	QT	P wave	T wave	Special Features
Hypothermia		Long	Wide	Long			"J" wave, Osborne
Hypokalemia	Depressed	Long		Long		Flat	Progressively more prominent "U" wave
Hyperkalemia		Long	Wide		Flat	Peaked	May be associated with digitalis toxicity
Hypocalcemia				Long		Inverted	
Hypercalcemia				Short			
Hypomagnesemia	Abnormal	Long		Long		Flat	↑Susceptibility to digitalis toxicity
Digitalis effects	Scooped			Short		Flat	
Digitalis toxicity							Premature ventricular contractions most common

II. SPECIFIC RHYTHM ASSESSMENTS
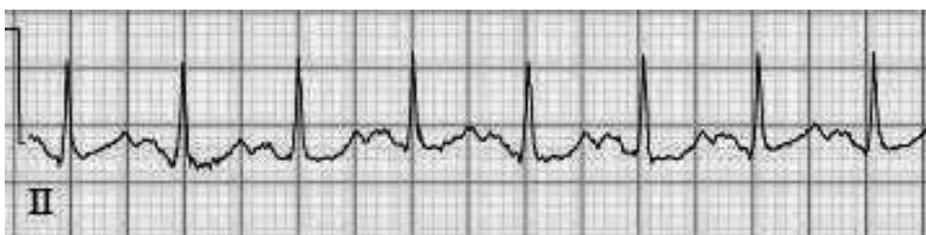
A. Sinus rhythm

Courtesy of Dr. Michael McCrea

1. Sinus rhythm is 60–100 beats per minute.
2. The rhythm is regular with 1:1 relationship of the P to QRS.
3. P waves are upright in leads I, II, and aVF. (Lead II is the typical lead for a rhythm strip.)
4. There are no extra beats.

B. Premature atrial contractions: extra beats that originate outside the sinus node from ectopic atrial pacemakers; appear interspersed throughout an underlying rhythm (usually sinus)

1. These ectopic P waves are different in configuration from normal P waves and may or may not be conducted through the AV node. They usually have a normal PR interval (0.12–0.20 seconds)
2. They are generally followed by a noncompensatory pause; the SA node is reset, and the returning sinus beat occurs ahead of schedule.

C. Sinus tachycardia: exactly like a sinus rhythm except that the rate is >100 (and usually <160) beats per minute

Courtesy of Dr. Michael McCrea

D. Sinus bradycardia: exactly like a sinus rhythm except that the rate is <60 (and usually >45) beats per minute
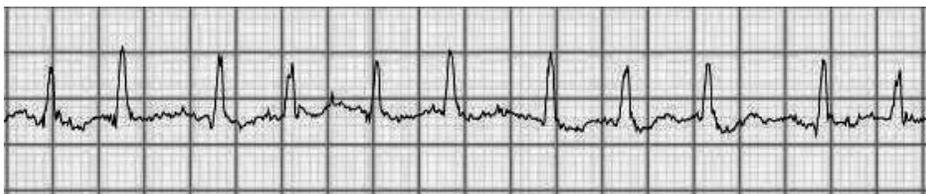

Courtesy of Dr. Michael McCrea

E. Supraventricular tachycardia

Courtesy of Dr. Daniel Schwerin

1. P waves are abnormal and may not be visible (often hidden in the preceding T wave), or they may immediately follow the QRS complex, in which case they are often inverted ("retrograde P waves"); atrial rate is 120–200 beats per minute.
2. Rhythm is regular.
3. QRS complexes are usually narrow but may be wide because of aberrant conduction through a bypass tract or preexisting bundle-branch block; ventricular rate is 120–220 beats per minute.

F. Atrial fibrillation: an irregularly irregular rhythm due to uncoordinated atrial activation and random occurrence of ventricular depolarization. The atria are not pumping, but they do discharge electrical impulses to the ventricles; however, no single impulse depolarizes the atria completely, so only an occasional impulse gets through to the AV node. It is the most common sustained dysrhythmia; it occurs in 2% of the general population and in 5% of people >60 years old.

Courtesy of Dr. Michael McCrea

1. P waves are absent but small irregular deflections in the baseline ("f" or "fibrillation waves") may be seen. They are most easily detected in the inferior leads (II, III, and aVF) and in V_1–V_3. The atrial rate is 400–700 beats per minute.
2. Because P waves are not visible, there is no PR interval.
3. QRS complexes are normal in configuration, unless there is aberrant conduction.
4. The rhythm is irregularly irregular.
5. Ventricular response rate is variable but is generally 160–180 beats per minute in untreated patients; a rate >200 beats per minute with a wide QRS complex suggests Wolff-Parkinson-White syndrome with conduction through the accessory pathway; a regular, slow ventricular rate may be digitalis toxicity.

G. Atrial flutter: a very rapid atrial rhythm but, because of AV nodal delay, ventricular responses are slower. Therefore, atrial flutter always occurs with some sort of AV block (not all impulses are conducted); the resulting block is either a fixed ratio (2:1, 3:1, 4:1, etc) or variable AV block.

Fixed AV block 3:1 atrial flutter


Courtesy of Dr. Michael McCrea

Variable AV block atrial flutter
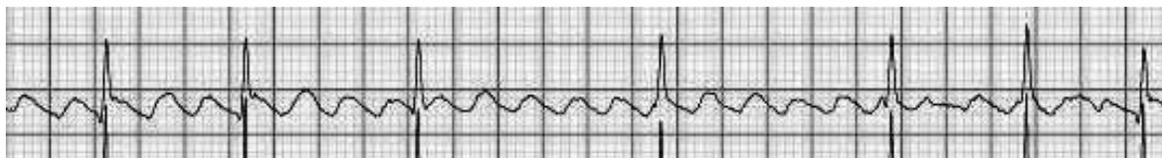
Courtesy of Dr. Michael McCrea

1. P waves have a characteristic sawtooth pattern and are called "F" or "flutter waves." They are usually best seen in the inferior leads and leads V_1 and V_2. The atrial rate is 250–350 beats per minute.
2. The PR interval (when present) is always normal, but not every P wave is followed by a QRS complex.
3. QRS complexes are normal in configuration.
4. The ventricular rate is often 150 + 20 beats per minute but depends on the degree of block present and may be variable. Suspect atrial flutter with a 2:1 block in patients who present with a fixed regular ventricular rate of 150 beats per minute.

H. Multifocal atrial tachycardia: an irregular rhythm sometimes mistaken for atrial fibrillation; originates from multiple different atrial sites and is characterized by P waves of varying shape
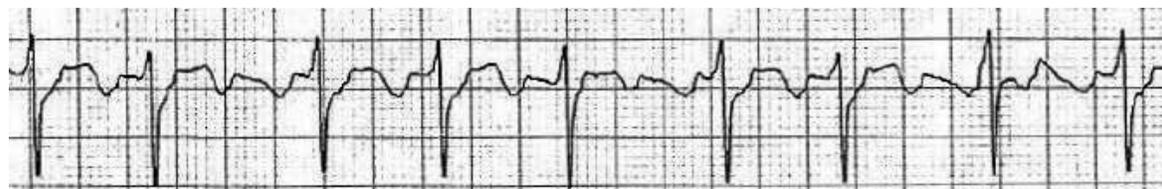
Courtesy of Dr. Daniel Schwerin

1. There must be at least three distinct types of P waves in one lead; atrial rate is 100–180 beats per minute.
2. The rhythm is irregularly irregular.
3. The PP, PR, and RR intervals vary.
4. Nonconducted (blocked) P waves are frequently present, particularly when the atrial rate is rapid. Classically seen with COPD and theophylline toxicity.
5. Management is directed at treatment of the underlying condition. Cardioversion is ineffective. The rhythm itself should not cause hemodynamic instability.

I. Junctional premature contractions: impulses that originate from an ectopic focus within the AV node or the bundle of His above the bifurcation. They may be isolated, multiple, or multifocal.

Courtesy of Dr. Michael McCrea

1. The ectopic P wave has a different shape and deflection (usually inverted in leads II, III, and aVF), and it may occur before, during, or after the QRS complex.
2. When the P wave precedes the QRS, the PR interval is shorter than normal (often <0.12 seconds).
3. The ectopic QRS complex is premature but has a normal shape unless there has been aberrant conduction.
4. They are generally followed by a compensatory pause; the SA node is not reset, and the next P wave occurs at its usual time.

J. Premature ventricular contractions: appear as abnormal QRS complexes and T waves that occur in addition to the underlying rhythm
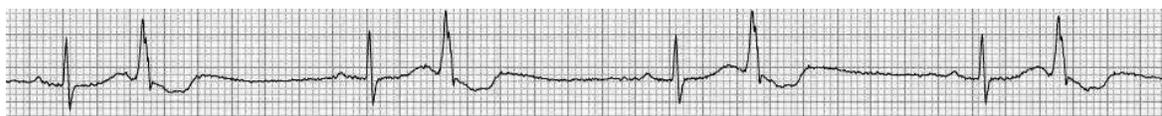
Courtesy of Dr. Michael McCrea

1. Characteristics
    a. Occur earlier than the next expected normal QRS
    b. Wider than a normal QRS (usually ≥0.12 second)
    c. The QRS morphology is generally bizarre.
    d. A preceding P wave is absent; however, retrograde conduction of a premature ventricular contraction can occasionally result in an inverted P wave after the QRS complex.
    e. The deflection of the ST segment and T wave is opposite that of the QRS.
    f. May occur in regular pattern, eg, bigeminy (as above)
    g. Generally followed by a compensatory pause; the sinoatrial node is not reset, and the next P wave occurs at its usual time.

K. **Ventricular tachycardia: three or more consecutive premature ventricular contractions occurring at a regular rate >120 beats per minute**

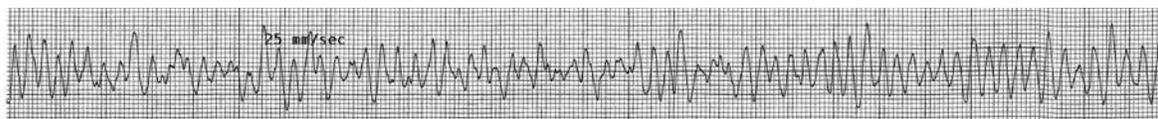*Courtesy of Dr. Michael McCrea*

1. P waves are usually absent; when present, they are either retrogradely conducted or have no relationship to the QRS (AV dissociation).
2. QRS complexes are wide (≥0.12 second) and may be bizarre.
3. Fusion beats may be present; these are intermediate in appearance between a bizarre QRS complex and a normal QRS. When present, the diagnosis of ventricular tachycardia is certain.
4. Capture beats are rarely seen but, when present, confirm the diagnosis of ventricular tachycardia. Capture beats are the result of an atrial impulse penetrating the AV node from above to stimulate ("capture") the ventricles. Because ventricular conduction occurs over the normal pathways, the resulting QRS of the captured beat looks normal (narrow) in appearance.
5. Deflection of the ST segment and T wave is generally opposite that of the QRS complex.
6. Rate is >120 (usually 150–200) beats per minute.
7. Rhythm is generally regular, although beat-to-beat variation may occur at the onset of tachycardia.
8. QRS axis is generally constant.
9. Ventricular tachycardia is classified as "monomorphic" (QRS complexes look the same) or "polymorphic" (QRS complexes have varying morphology). Current therapeutic modalities are based on this classification (see Etiologies and Management of Dysrhythmias, pages 22–32).
10. Differentiation of supraventricular tachycardia with aberrancy from ventricular tachycardia
    a. Most published criteria that can be used to diagnose ventricular tachycardia are fairly reliable; however, there are no reliable criteria to exclude ventricular tachycardia. Because the misdiagnosis of ventricular tachycardia can be deadly, if there is any doubt about the diagnosis whatsoever, always assume that a wide complex tachycardia is ventricular tachycardia and treat as such!
    b. Fusion and capture beats indicate AV dissociation and are practically diagnostic of ventricular tachycardia.
    c. P waves preceding QRS complexes favor aberrancy.
    d. QRS concordance (all the QRS complexes from V_1 to V_6 are either positive or negative) strongly favors ventricular tachycardia. A fully compensatory pause is more likely to occur with ventricular tachycardia.
    e. Response to vagal maneuvers (Valsalva maneuver, carotid sinus massage) may occur with aberrant supraventricular tachycardia, whereas ventricular tachycardia is unaffected. (Carotid sinus massage is contraindicated in older patients with a history of carotid disease/stroke or the presence of a carotid bruit.)
    f. Marked left axis deviation (>30°) suggests ventricular tachycardia; any QRS axis deviation >40° in either direction (or an upright QRS in aVR) favors ventricular tachycardia.
    g. QRS duration >0.14 second favors ventricular tachycardia.
    h. QRS morphology in lead V_1: an RS, R or qR with left "rabbit ear" taller than the right suggests ventricular tachycardia, whereas an RSR' pattern is more likely supraventricular tachycardia with aberrancy; negative QRS morphology in this lead with a wide R wave (>0.03 second), RS interval >0.07 second, and a slurred or notched S wave favors ventricular tachycardia.
    i. QRS morphology in lead V_6: R/S ratio <1, a qS or qR favors ventricular tachycardia.

j. A bundle-branch pattern that varies suggests supraventricular tachycardia with aberrancy.

k. A history of prior heart disease (MI, CHF, coronary artery bypass graft) strongly favors ventricular tachycardia (likelihood 85%), as does a prior history of ventricular tachycardia.

l. Age ≥50 years old favors ventricular tachycardia, whereas age ≤35 years old favors an aberrant supraventricular tachycardia.

11. Torsades de pointes ("twisting of the points") is a type of polymorphic ventricular tachycardia in which the QRS axis swings from a positive to a negative direction in a single lead creating a "sine-wave" appearance. It originates from a single focus and is usually precipitated by diseases or drugs that prolong the QT interval, such as Class IA antidysrhythmics (procainamide, quinidine), Class IC antidysrhythmics (propafenone, fecainide), tricyclic antidepressants, droperidol, and the phenothiazines. The combined use of certain drugs such as terfenadine plus ketoconazole or erythromycin also prolong the QT interval and may therefore precipitate torsades. Other causes include hypomagnesemia and hypokalemia. The rate is typically 200–240 beats per minute.
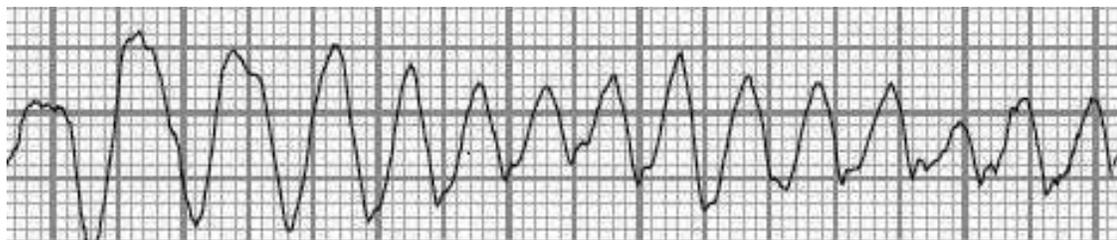
Courtesy of Dr. Daniel Schwerin

L. Ventricular fibrillation

Courtesy of Dr. Michael McCrea

1. Most commonly recognized as a fine or coarse zigzag pattern without discernible P waves, QRS complexes, or T waves representing the presence of unorganized ventricular electrical activity

2. By definition, there is no organized perfusion and therefore absence of a pulse.

3. Sometimes the rhythm may look like ventricular tachycardia. The point is moot if the patient has no pulse and is unresponsive, because the treatment is the same, unsynchronized defibrillation.

M. Pulseless electrical activity (PEA)

1. Refers to a heterogeneous group of rhythms characterized by the presence of some type of electrical activity other than ventricular tachycardia or ventricular fibrillation in the absence of a perceptible pulse

2. Includes electromechanical dissociation, pseudoelectromechanical dissociation, idioventricular rhythms, ventricular escape rhythms, bradyasystolic rhythms, and postdefibrillation idioventricular rhythms

3. These dysrhythmias often occur in association with specific clinical conditions that if promptly identified and treated may result in a return of spontaneous circulation. Remember the "H's and T's," looking for reversible causes.

a. Hypovolemia

b. Hydrogen ion, ie, acidosis

c. Hypothermia

d. Hypo/hyperkalemia

e. Hypoxia

f. Toxins

g. Tamponade, cardiac

h. Tension pneumothorax

i. Thrombosis, coronary

j. Thrombosis, pulmonary

N. Bundle-branch blocks (BBBs)

1. Abnormal conduction abnormalities (not rhythm disturbances) in which the ventricles depolarize in sequence (rather than simultaneously), thus producing a wide QRS complex (0.09–0.11 → incomplete BBB; ≥0.12 second → complete BBB) and an ST segment with a slope opposite that of the terminal half of the QRS complex

2. Right BBB is a unifascicular block in which ventricular activation is by way of the left bundle branch; the impulse travels down the left bundle, thus activating the septum from the left side (as it normally does in the absence of right BBB). This is followed by activation of the free wall of the left ventricle and, finally, the free wall of the right ventricle. Because of the two changes in direction, there is a tendency toward triphasic complexes in right BBB.
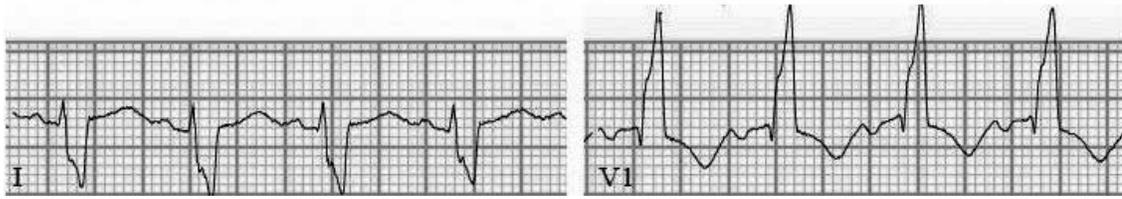
Courtesy of Dr. Michael McCrea

    a. Wide QRS complex (≥0.12 second)
    b. Triphasic QRS complex (rSR' variant) in lead V_1
    c. Wide S waves in leads I, V_5, and V_6
    d. Normal septal Q waves in leads I and V_6 because the initial activation of the ventricle occurs in the normal manner
    e. T wave has a deflection opposite that of the terminal half of the QRS complex.
    f. Associated axis is variable; a normal axis, left axis deviation, or even right axis deviation may be present.

3. Left BBB is a bifascicular block in which the left ventricle is activated by way of the right bundle branch; the impulse travels down the right bundle, activating the septum and the free wall of the right ventricle, and then continues on in the same direction to activate the free wall of the left ventricle. Because the dominant forces are traveling in the same direction, there is a tendency toward monophasic QRS complexes.
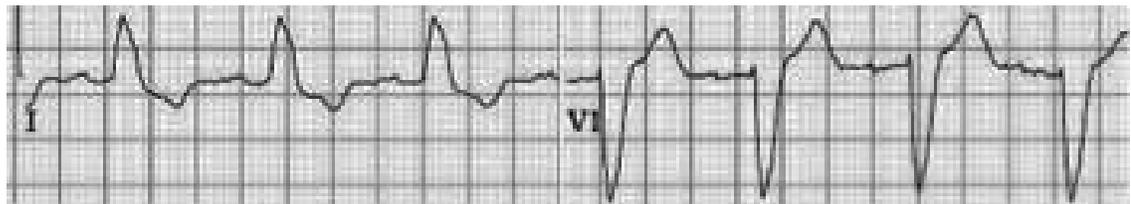
Courtesy of Dr. Michael McCrea

    a. Wide QRS complex (≥0.12 sec)
    b. Negative wave (QS or rS) in lead V_1
    c. Large, wide R waves in leads I, aVL, V_5, and V_6
    d. Absence of normal septal Q waves in leads I and V_6
    e. T wave has a deflection opposite that of the terminal half of the QRS complex.
    f. Associated left-axis deviation is most common and implies the presence of additional myocardial disease.

O. Sinoatrial block

1. Occurs when there is abnormal conduction between the sinus node and atrial muscle; recognized by the unexpected absence of a P wave and its associated QRS complex. Like AV block, sinoatrial block is also divided into first-, second-, and third-degree varieties.

2. First-degree sinoatrial block
    a. Impulse is delayed in its conduction from the sinoatrial node to the atria.
    b. Cannot be diagnosed from a surface 12-lead ECG

3. Second-degree sinoatrial block
    a. Some of the sinus node discharges are blocked.
    b. Recognized on an ECG as the absence of an expected P wave and its associated QRS complex

4. Third-degree sinoatrial block (sinus arrest)
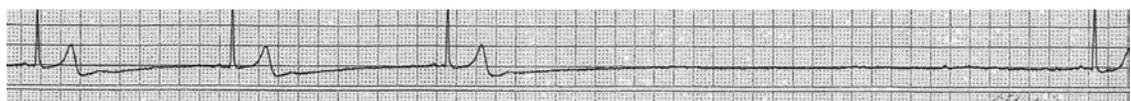
Courtesy of Dr. Michael McCrea

      a. All of the sinus node discharges are blocked.

      b. On ECG, it may appear as a long sinus pause/arrest or a junctional/ventricular escape rhythm.

P. Sick sinus syndrome

    1. An abnormality of cardiac impulse formation as well as intra-atrial and AV nodal conduction

    2. Manifests as a wide variety of, or combinations of, bradyarrhythmias and tachyarrhythmias

    3. Most common in the elderly

    4. Presenting symptoms may include dizziness, palpitations, dyspnea, fatigue, lethargy, or syncope. Documentation of a bradyarrhythmia or tachyarrhythmia in association with these symptoms is the cornerstone of diagnosis.

Q. Atrioventricular blocks

    1. Occur when the conduction between the atria and ventricles is abnormal; the conduction delay can occur in the atria, the AV node, or the proximal His-Purkinje system.

    2. First-degree AV block: normal AV conduction is slightly prolonged.

       Note PR 390 msec.

Courtesy of Dr. Michael McCrea

      a. P waves and QRS complexes are normal.

      b. There is a 1:1 relationship between the P and QRS.

      c. PR interval is prolonged (>0.20 second)

      d. The block is most often at the level of the AV node.

    3. Second-degree AV block: some atrial impulses are not conducted.

      a. Mobitz Type I (Wenckebach)

        (1) P waves and QRS complexes are normal, but there are P waves without QRS complexes (nonconducted P waves).

        (2) PR interval progressively lengthens and the RR interval progressively shortens until a beat is dropped. This cycle repeats itself, producing a pattern referred to as "group beating."

        (3) The longest cycles (those of the dropped beats) are less than twice the length of the shortest cycles (those of the impulses following the dropped beats).

        (4) The block is almost always within the AV node.

      b. Mobitz Type II
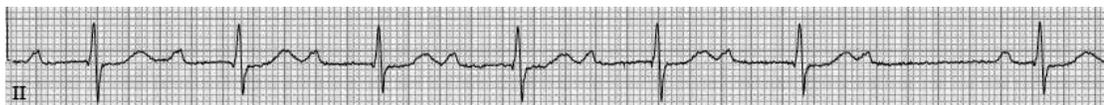
Courtesy of Dr. Michael McCrea

        (1) P waves are normal.

        (2) QRS complexes are often (but not always) wide because of the common occurrence of a coexisting bundle-branch block.

        (3) PR intervals (when they occur) are always the same duration.

        (4) There are dropped beats.

        (5) The block is below the level of the AV node, generally in the His-Purkinje system.

4. Third-degree AV block: no atrial impulses are conducted; the atria and ventricles beat independently of one another.
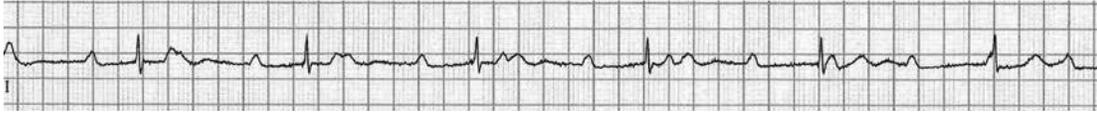
Courtesy of Dr. Michael McCrea

    a. P waves appear normal.

    b. May occur at the level of the AV node, the bundle of His, or the bundle branches

    c. QRS complexes may be narrow or wide, depending on the location of the block: if the block is located above the His bundle, the QRS complexes will be narrow and reflect a junctional escape rhythm, whereas if the block is located at or below the bundle of His, the QRS complexes will be wide and reflect a ventricular escape rhythm.

    d. There is no relationship between P waves and QRS complexes.

        (1) There is an independent and regular atrial rate (constant PP interval) and a slower independent and constant ventricular rate (constant RR interval).

        (2) The P waves are not related (not conducted) to the QRS complexes but rather "march through them" as if they were not there.

        (3) The PR intervals are variable.

R. Preexcitation syndromes

1. Result from abnormal connections (accessory pathways) between the atria and ventricles. Impulses traveling down these pathways bypass all or part of the normal conduction system. This results in the ventricles being activated by atrial impulses sooner than would normally be anticipated (preexcitation) and is reflected by changes in the surface ECG. The ECG changes seen are determined by the exact pathway the impulse travels. Wolff-Parkinson-White syndrome and Lown-Ganong-Levine syndrome are the two major variants of preexcitation. Their characteristic ECG findings are listed below. Patients with preexcitation syndromes are prone to tachydysrhythmias (especially paroxysmal supraventricular tachycardia and atrial fibrillation) with very rapid ventricular rates (up to 300 beats per minute).

2. Wolff-Parkinson-White: the accessory pathway (bundle of Kent) connects the atria directly to the ventricles, completely bypassing the AV node and the infranodal conduction system.
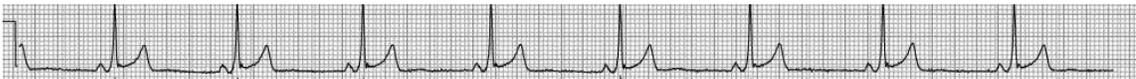
Courtesy of Dr. Michael McCrea

    a. Short PR interval (<0.12 second)

    b. Delta wave (a slurred upstroke to the QRS)

    c. Wide QRS

    d. Secondary ST–T wave changes; deflection of the T wave may be opposite that of the QRS vector if the classic triad is present. (Otherwise, the QRS–T may appear normal.)

    e. There is a propensity to develop atrial tachyarrhythmias, most commonly supraventricular tachycardia but occasionally atrial fibrillation.

3. Lown-Ganong-Levine syndrome: the accessory pathway (James fibers) connects the atria directly to the proximal His bundle, completely bypassing the AV node.

Courtesy of Dr. Daniel Schwerin

    a. Short PR interval (<0.12 second)

    b. No delta wave

    c. Normal QRS

    d. There is a propensity to develop atrial tachydysrhythmias.

III. ETIOLOGIES AND MANAGEMENT OF DYSRHYTHMIAS

A. Premature atrial contractions

1. There are multiple causes (drugs or underlying disease), but they may also occur as a normal variant.
2. Clinical significance
   a. Can precipitate supraventricular tachycardia, atrial fibrillation, and atrial flutter
   b. Most frequent cause of a pause on the ECG
3. In general, no treatment is indicated. If, however, the premature atrial contractions are frequent or symptomatic, treatment should be directed toward correcting the underlying cause.

B. Sinus tachycardia

1. There are multiple causes; common ones include:
   a. Anxiety (diagnosis of exclusion)
   b. Stimulant or sympathomimetic drugs (eg, cocaine)
   c. Fever
   d. Hypovolemia
   e. Hyperthyroidism
   f. Pulmonary embolism
   g. Anemia
   h. Hypoxia
   i. Pain
2. Management in most instances should be directed at finding and correcting the underlying cause.
3. In the setting of cocaine or stimulant toxicity, administration of a benzodiazepine is first-line treatment.

C. Sinus bradycardia

1. Common causes
   a. Acute inferior wall MI
   b. Vasovagal events (eg, vomiting)
   c. Drug effect (eg, β-blockers, calcium channel blockers)
   d. Sick sinus syndrome
   e. Hypothermia
   f. Hypothyroidism
   g. A normal variant, especially in those individuals who exercise aerobically on a regular basis
2. **Management**
   a. **Indicated for patients who demonstrate signs of hypoperfusion due to the bradycardia: those with shock, hypotension, ischemic chest pain, decreased mentation, or acute CHF**
   b. **Intervention sequence**
      (1) **Atropine 0.5 mg every 5 minutes as needed until a response is noted or a total of 0.03–0.04 mg/kg has been administered (3 mg for most adults).**
         (a) **Should be used cautiously in patients with an acute MI, because it may worsen ischemia or precipitate ventricular tachycardia or ventricular fibrillation**
         (b) **Should also be used with caution in patients with Mobitz Type II second-degree AV block and new third-degree AV block with wide complexes → ↑ atrial rate → ↑ AV block → ↓ ventricular rate and ↓ blood pressure**
         (c) **Atropine is ineffective in patients with heart transplants because of lack of vagal innervation to the transplanted heart. Proceed directly to transcutaneous pacing and/or catecholamine infusion.**
         (d) **Can be parasympathomimetic in doses <0.5 mg, further decreasing heart rate**
      (2) **Transcutaneous pacing**
         (a) **The treatment of choice for patients who are unresponsive to atropine and for those with severe symptoms**
         (b) **Analgesics or sedatives may be required by some patients to be able to tolerate the pacing stimulus.**

        (3) Dopamine 5–20 mcg/kg/min or epinephrine 2–10 mcg/min

          (a) Should be used when bradycardia is unresponsive to atropine and a transcutaneous pacer is not readily available

          (b) Most useful when associated hypotension is present

        (4) Transvenous pacing may be required if symptomatic bradycardia persists despite vasopressors and/or transcutaneous pacing.
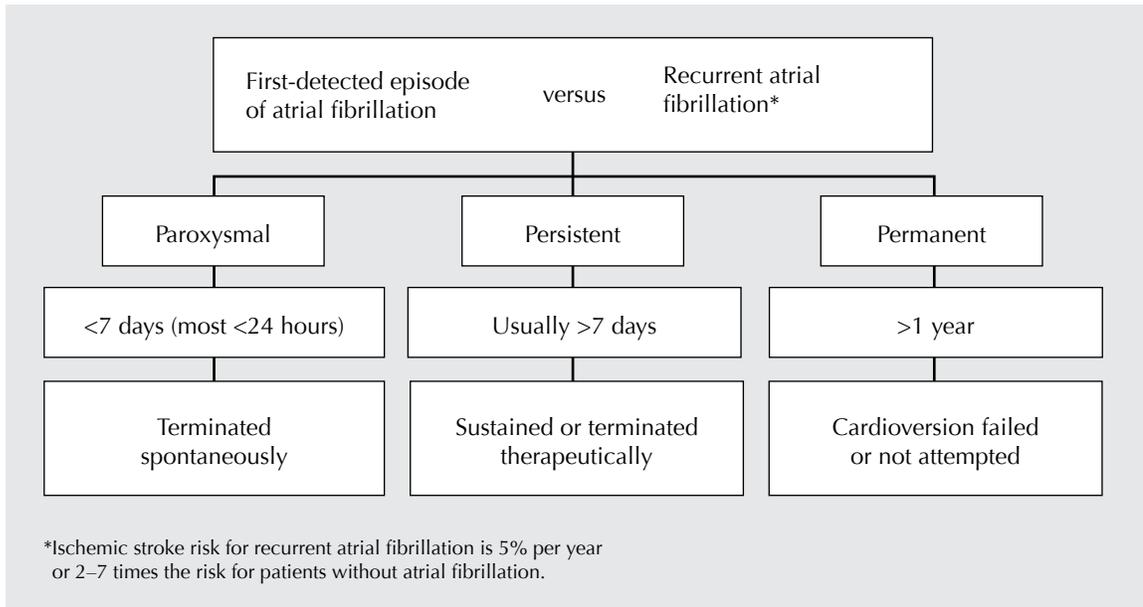
D. Supraventricular tachycardia (SVT)

1. A generic term that refers to all tachydysrhythmias arising above the bifurcation of the bundle of His, including sinus tachycardia, atrial fibrillation, atrial flutter, multifocal atrial tachycardia, paroxysmal supraventricular tachycardia, and nonparoxysmal junctional tachycardia. It arises from reentry or an ectopic pacemaker in the atria. Most clinicians, however, use the term SVT to refer specifically to AV nodal reentry tachycardia (AVNRT) and other undetermined supraventricular rhythms. In the discussion that follows, SVT refers to AVNRT. Management is discussed separately for other specific forms of SVT (such as atrial fibrillation).

2. Causes

   a. Preexcitation syndromes (Wolff-Parkinson-White and Lown-Ganong-Levine)

   b. Mitral disease (prolapse, stenosis)

   c. Digitalis toxicity

   d. Drugs (eg, alcohol, tobacco, caffeine)

   e. Acute MI and pericarditis

   f. Hyperthyroidism

   g. Rheumatic heart disease

3. **Management is determined primarily by the patient's hemodynamic stability and secondarily by the width of the QRS complex.**

   a. **Hemodynamically compromised patients (those with hypotension, ischemic chest pain, a decrease in mental status, or acute CHF) with a narrow complex SVT should be sedated (if possible) and treated with synchronized cardioversion. Start with 50 joules.**

   b. **Vagal maneuvers and pharmacologic therapy may be used in the hemodynamically stable patient with narrow complex SVT.**

      (1) Vagal maneuvers (such as carotid sinus massage [should not be done if digitalis toxicity has not been excluded] or Valsalva maneuver) increase vagal tone and may be effective in either terminating the dysrhythmia or slowing the ventricular rate enough to uncover the actual underlying rhythm. These maneuvers should be attempted before starting pharmacologic therapy and may also be used to supplement it. The vagal maneuver of choice, ie, the most effective, is the Valsalva maneuver.

      (2) Adenosine, because of its safety profile, is the drug of choice for the hemodynamically stable patient with narrow complex SVT. It is an ultra-short-acting AV nodal blocker that is very effective in converting SVT. Its major advantages over verapamil are its short half-life (<10 seconds) and its lack of hypotensive and myocardial depressant effects. Although it does produce adverse effects (flushing, dyspnea, chest pain), they are transient. Recurrence of SVT, however, is common (up to 50%–60% of patients). Adenosine does have several significant drug interactions. Its effects are antagonized by the methylxanthines (theophylline, caffeine) and potentiated by dipyridamole and carbamazepine. Therefore, large doses of adenosine may be required in the presence of methylxanthines, whereas smaller doses (or an alternative agent) should be used in the presence of dipyridamole and carbamazepine.

      (3) Calcium channel blockers (eg, diltiazem, verapamil) are as effective as adenosine but slower in onset and produce more significant adverse effects (decreased myocardial contractility and peripheral vasodilation). Calcium channel blockers should not be used concomitantly with IV β-blockers and are contraindicated in patients with wide complex tachycardias, atrial fibrillation with Wolff-Parkinson-White, sick sinus syndrome, and advanced AV block.

        (a) Pretreatment with a fluid bolus and calcium chloride (0.5–1 g IV over several minutes) are useful in preventing the hypotension induced by the vasodilatory effects of verapamil.

        (b) Diltiazem seems to be as effective as verapamil in the treatment of narrow complex SVT and has the advantage of producing less myocardial depression.

(4) **β-blockers such as esmolol, metoprolol, or propranolol are also effective in the treatment of narrow complex SVT. Esmolol has the advantage of being cardioselective as well as having a very short half-life. Propranolol is the drug of choice for SVT secondary to thyrotoxicosis, because it partially blocks the conversion of T_4 and T_3. Avoid these drugs in patients with COPD, asthma, or CHF; in those who have received IV calcium channel blockers; and in those with atrial fibrillation with Wolff-Parkinson-White, sick sinus syndrome, and advanced AV block.**

(5) **Digoxin is vagotonic. Compared with the other agents listed above, its effects are mild and have a much slower onset (may take 2–4 hours or more to work). Digoxin should be avoided if cardioversion is being considered.**

(6) **Magnesium sulfate, phenytoin, and lidocaine are the drugs of choice for ectopic SVT caused by digitalis toxicity. Management should also include correction of hyper/hypokalemia (if present) and discontinuation of digitalis. In the presence of hemodynamic instability (or potentially lethal digitalis intoxication), administration of Fab fragments should be considered.**

(7) **Other antidysrhythmic agents (eg, procainamide, amiodarone, sotalol) may also be effective.**

(8) **Patients who do not respond to drug therapy may be treated with synchronized cardioversion (as described above) or overdrive cardiac pacing.**

c. **Drug dosages and administration**

(1) **Adenosine 6 mg rapid IV push in a proximal vein followed by a 20-mL bolus of normal saline; if there is no response after 1–2 minutes, double the dose to 12 mg.**

(2) **Verapamil 2.5–5 mg IV over 2–3 minutes; a second dose of 5–10 mg may be given in 15–30 minutes if necessary.**

(3) **Diltiazem 0.25 mg/kg IV over 2 minutes, followed in 15 minutes by a second bolus of 0.35 mg/kg if the first bolus was tolerated but ineffective. Smaller dosages should be considered in elderly patients.**

(4) **Esmolol 300–500 mcg/kg bolus over 1 minute followed by an infusion of 50 mcg/kg/min; the loading dose may need to be repeated and the infusion rate increased by 50 mcg/kg/min every 5 minutes as needed to a maximum of 200 mcg/kg/min.**

(5) **Metoprolol 5 mg IV over 2 minutes; may be repeated twice every 5 minutes for total of three doses.**

(6) **Propranolol 1 mg IV over 1 minute; this dose may be repeated every 5 minutes up to a total dosage of 0.1–0.5 mg/kg.**

(7) **Digoxin 0.5 mg IV push initially, with repeated doses of 0.25 mg every 30–60 minutes as needed; total dosage should not exceed 0.02 mg/kg.**

(8) **Magnesium sulfate 1–2 g slow IV push over 1–2 minutes followed by an infusion of 1–2 g/hr**

(9) **Phenytoin 18 mg/kg IV bolus infusion; dissolve dosage in normal saline and administer at a rate of 50 mg/min or less.**

(10) **Lidocaine 1–1.5 mg/kg bolus infusion, repeat dosages 0.5–0.75 mg/kg every 5–10 minutes, to maximum bolus dose of 3 mg/kg. This can be followed by maintenance infusion of 1–4 mg/min.**

d. **Patients with wide complex tachycardia should be presumed to have ventricular tachycardia.**

(1) **If unstable → synchronized cardioversion**

(2) **If stable → procainamide or amiodarone**

(a) **Both convert SVT or ventricular tachycardia.**

(b) **Both should be avoided in patients with tricyclic antidepressant overdose or in the setting of toxicity from other sodium-channel blockers.**

(c) **Adenosine may initially slow either rhythm, but dysrhythmia may recur (short therapeutic effect of the drug).**

(d) **If drug therapy fails → synchronized cardioversion**

E. Atrial fibrillation (most common SVT)
  1. Identify the type of atrial fibrillation the patient has by determining the probable duration of the dysrhythmia.

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┌─────────────────────────────┬─────────┬──────────────────────────┐
│ First-detected episode      │ versus  │ Recurrent atrial         │
│ of atrial fibrillation      │         │ fibrillation*            │
└─────────────────────────────┴─────────┴──────────────────────────┘
        │                          │                       │
  ┌───────────┐            ┌───────────┐            ┌───────────┐
  │ Paroxysmal│            │ Persistent│            │ Permanent │
  └───────────┘            └───────────┘            └───────────┘
        │                          │                       │
┌──────────────────┐    ┌──────────────────┐    ┌──────────────────┐
│<7 days (most     │    │ Usually >7 days  │    │ >1 year          │
│ <24 hours)       │    │                  │    │                  │
└──────────────────┘    └──────────────────┘    └──────────────────┘
        │                          │                       │
┌──────────────────┐    ┌──────────────────┐    ┌──────────────────┐
│ Terminated       │    │ Sustained or     │    │ Cardioversion    │
│ spontaneously    │    │ terminated       │    │ failed or not    │
│                  │    │ therapeutically  │    │ attempted        │
└──────────────────┘    └──────────────────┘    └──────────────────┘
```

*Ischemic stroke risk for recurrent atrial fibrillation is 5% per year
 or 2–7 times the risk for patients without atrial fibrillation.

  2. **Search for reversible causes and treat any underlying medical condition; then determine the risk of subsequent stroke. Conditions with high risk of cardiogenic thromboembolism:**
     a. **Cardiac surgery**
     b. **Acute MI**
     c. **Hyperthyroidism**
     d. **Myocarditis**
     e. **Acute pulmonary disease**
  3. **Plan the treatment using the following criteria:**
     a. **Cardiovascular stability**
     b. **Duration of the dysrhythmia**
     c. **Underlying cause/condition**
     d. **Presence/absence of an accessory pathway**
  4. **There are fundamentally two ways to manage atrial fibrillation: restore and maintain sinus rhythm, or allow atrial fibrillation to continue and ensure that the ventricular rate is controlled.**
     a. **Unstable patients → immediate electrical synchronized cardioversion**
     b. **Stable patients with significant symptoms → pharmacologic therapy**
        (1) **Control the ventricular rate first (choose *one*)**
            (a) **Calcium channel blocker (eg, verapamil, diltiazem)**
            (b) **β-blocker (eg, esmolol, atenolol, metoprolol)**
            (c) **Digoxin**
            (d) **Amiodarone**
            (e) **The presence of Wolff-Parkinson-White syndrome is a special circumstance requiring changes in treatment protocols (see pages 31–32).**
        (2) **Cardiovert the dysrhythmia based on the duration of atrial fibrillation.**
            (a) **<48 hours duration**
                i. **Normal cardiac function: perform electrical cardioversion or use one of the following agents:**
                   • **Amiodarone**
                   • **Ibutilide**
                   • **Flecainide**
                   • **Propafenone**
                   • **Procainamide**
                ii. **Compromised cardiac function: perform synchronized cardioversion or use amiodarone**