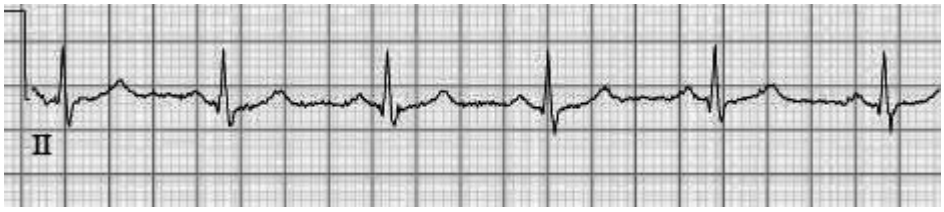
DYSRHYTHMIAS

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 ×1 mm.
3. The horizontal axis measures time; each small division is 0.04 sec/mm.

B. Normal morphology

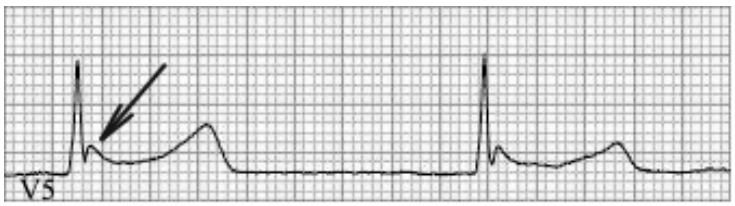


Courtesy of Michael McCrea, MD

1. P wave = atrial depolarization
   a. Upright in leads I, II, and aVF; inverted in lead aVR
   b. Normal PR interval is 0.12–0.20 seconds.
2. QRS complex = ventricular depolarization
   a. Measures <0.10 seconds wide
   b. Q wave
      (1) Generally considered normal if <0.04 seconds wide or <1/3 the size of the entire QRS complex
      (2) Indicative of infarction if ≥0.04 seconds wide or >1/3 of size of the entire QRS complex.
3. QT interval varies with rate and sex but is usually 0.33–0.42 seconds; at normal heart rates, it is normally less than half of the preceding RR interval.
4. T wave = ventricular repolarization
   a. Typically upright, but inverted T waves are typical in leads V_1, aVR, and occasionally III
   b. Slightly rounded and asymmetric in configuration
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 V3
   d. Can be a normal component of the ECG
   e. Prominent U waves may indicate one of the following:
      (1) Hypokalemia
      (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 (often appears when core temperature <89.6°F [32°C])



Courtesy of Michael McCrea, MD

(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).

(4) The exact temperatures at which specific findings or dysrhythmias occur will vary depending on the patient.

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 pulses are not clearly palpable, begin CPR without delay.

(3) Ventricular fibrillation

(a) Often refractory to defibrillation attempts until the patient is rewarmed to a core temperature above 86°F (30°C)

(b) Defibrillation should be attempted with a single shock, but, if unsuccessful, CPR and rapid rewarming measures should be instituted. Further attempts at defibrillation may be withheld until the patient's temperature rises above 86°F (30°C), with repeat attempts at defibrillation with every one degree rise in core temperature.

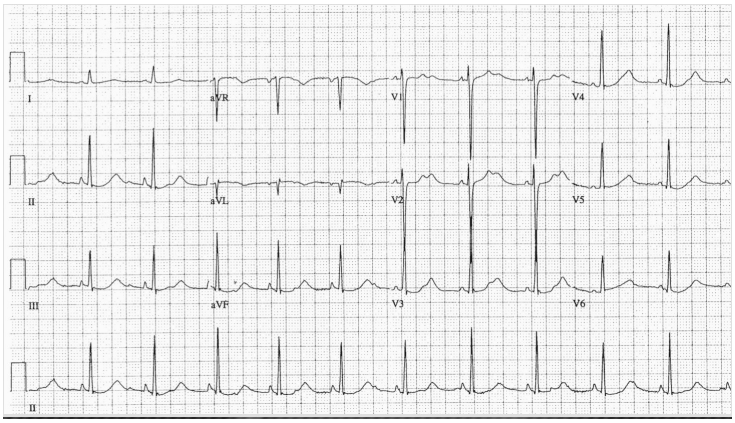
(c) As the myocardium rewarms, ventricular fibrillation may convert spontaneously or resolve in response to defibrillation.

(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 opioid abuse is suspected, naloxone should be considered because it may act on central opioid 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 90°F (32°C).

2. Hypokalemia



Courtesy of Amal Mattu, MD

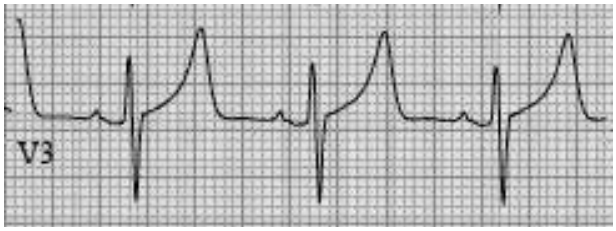
a. Progressively more prominent U wave (best seen in V_3)

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

c. Depression of ST segment

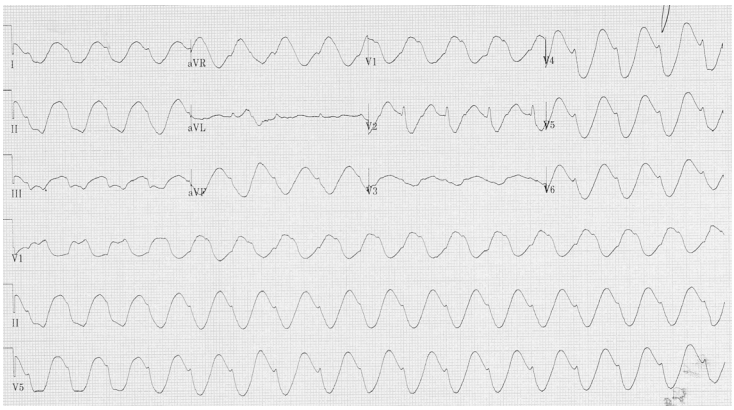
d. Prominent P wave
e. Apparent prolongation of the QT interval (although actually T-U fusion gives the appearance of a prolonged QT interval)
f. ST elevation in lead aVR
g. Ventricular tachycardia/torsades de pointes
h. In the presence of hypokalemia, susceptibility to digitalis toxicity and its associated dysrhythmias is increased.
i. Often associated with chronic digitalis toxicity

3. Hyperkalemia



Courtesy of Michael McCrea, MD

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



Courtesy of Amal Mattu, MD

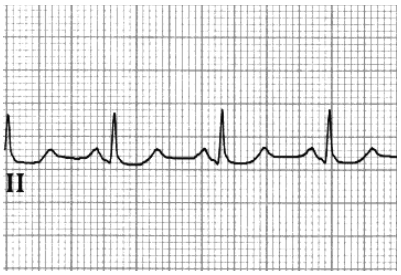
e. Bradyarrhythmias and/or heart blocks
f. QT interval is often prolonged because of associated hypocalcemia
g. ST elevation in lead aVR is common

Table 1: Effects on ECG with Increasing Potassium Concentration

Potassium Concentration (mEq/L)	**ECG Changes (variable depending on patient)**
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 bradycardic rhythms
>8.0	Sine-wave appearance Ventricular fibrillation Asystole

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

   b. Ventricular dysrhythmias (including torsades de pointes)
5. Hypercalcemia
   a. 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. Nonspecific ST segment abnormalities
   c. Predisposition to atrial and ventricular tachydysrhythmias
   d. 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).
   e. 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, inverted, or biphasic 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) Na^+-K^+-ATPase pump inhibition → 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 SA and AV nodes → bradyarrhythmias 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, kidney failure, hypothyroidism)
      (7) Drug interactions (quinidine, calcium channel blockers, erythromycin, amiodarone, captopril, and ibuprofen)
   c. ECG findings
      (1) Premature ventricular contractions (most common dysrhythmia), 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, delirium, and psychosis

e. Acute digitalis toxicity is usually seen in young and otherwise healthy patients from either accidental or intentional overdose; it is commonly associated with hyperkalemia, high digoxin levels, bradydysrhythmias, and AV blocks. Toxicity and morbidity in these patients are most closely correlated with the degree of hyperkalemia, *not* the serum digoxin level.

f. Chronic digitalis toxicity generally occurs in older cardiac patients with reduced kidney 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.

g. 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 8.1 ng/mL. 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 ng/mL and a potassium concentration of 3.0 mEq/L.

h. 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) Consider multiple doses of activated charcoal in 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; overzealous correction can actually exacerbate AV conduction defects)
(b) Hyperkalemia is best treated with Fab fragments (digoxin-specific antibody fragments); use calcium cautiously because there is a theoretical risk of 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. Co-ingestions 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, red squill) 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.
(8) Dialysis is ineffective given the high volume of distribution of digoxin.

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 elevation in a VR			Long		Flat	Progressively more prominent "U" wave; may be associated with chronic digitalis toxicity
Hyperkalemia	Elevation in aVR	Long	Wide		Flat	Peaked	May be associated with acute digitalis toxicity
Hypocalcemia				Long			
Hypercalcemia				Short			
Hypomagnesemia		Long		± Long		Flat	Increased susceptibility to digitalis toxicity
Digitalis effects	Scooped			Short		Flat	
Digitalis toxicity	Scooped						Premature ventricular contractions most common

II. GENERAL APPROACH TO DYSRHYTHMIAS

A. Unstable patients receive electrical therapies.

1. Unstable tachycardias receive synchronized cardioversion. The following are the currently recommended starting energy doses in the 2020 ACLS Guidelines:
   a. Narrow complex regular tachycardia: 50–100 Joules
   b. Narrow complex irregular tachycardia: 120–200 Joules

c. Wide complex regular tachycardia: 100 Joules
d. Wide complex irregular tachycardia: 200 *Joules unsynchronized*

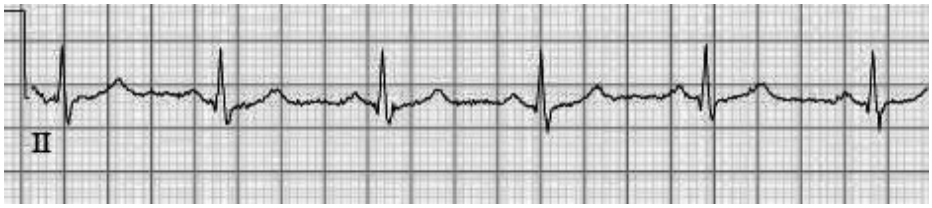
2. Unstable bradycardias receive electrical pacing, either temporary transcutaneous pacing or a transvenous pacemaker.

B. Stable patients receive medication after analysis of the rhythm.

1. Tachydysrhythmias
   a. Narrow complex tachydysrhythmias originate above or at the AV node receive AV-nodal blocking agents.
      (1) Adenosine
      (2) β-blockers
      (3) Calcium channel blockers
      (4) Digoxin
   b. Wide complex tachydysrhythmias generally originate below the AV node, ie, in the ventricles, and receive Na-channel or K-channel blockers.
      (1) Amiodarone
      (2) Lidocaine
      (3) Procainamide
2. Bradydysrhythmias
   a. If ensuring adequate oxygenation and ventilation does not improve the bradycardia, administer atropine.
   b. If atropine is unsuccessful or transient, consider transcutaneous pacing; or begin vasopressors, epinephrine, or dopamine.
3. Drug dosages and administration
   a. Adenosine 6 mg rapid IV push in a proximal vein followed by a 10-mL bolus of normal saline; if there is no response after 1–2 minutes, double the dose to 12 mg.
   b. Calcium channel blockers
      (1) 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.
      (2) 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 older patients.
   c. β-blockers
      (1) 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.
      (2) Metoprolol 5 mg IV over 2 minutes; may be repeated twice every 5 minutes for a total of 3 doses.
      (3) 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.
   d. 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.
   e. Magnesium sulfate 1–2 g slow IV push over 1–2 minutes followed by an infusion of 1–2 g/hour
   f. 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.
   g. Amiodarone 150 mg IV bolus over 10 minutes, followed by maintenance infusion of 1 mg/min for 6 hours, then 0.5 mg/min for 18 hours.
   h. Procainamide 20–50 mg/min until the QRS widens by 50% of its original width, hypotension develops, or a total dose of 17 mg/kg has been administered. This should be followed by a maintenance drip of 1–4 mg/min.
   i. Atropine 1 mg IV every 3–5 minutes as needed until a response is noted or a total of 3 mg.

III. SPECIFIC RHYTHM ASSESSMENTS

A. Sinus rhythm



Courtesy of Michael McCrea, MD

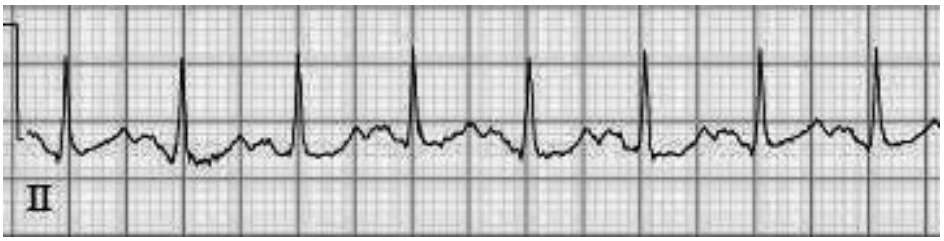
1. Sinus rhythm is 60–100 beats per minute.
2. The rhythm is regular with 1:1 relationship of the P to QRS.
3. P waves are upright in leads I, II, and aVF; and inverted in lead aVR.

B. Premature atrial contractions (PACs)

1. Extra beats that originate outside the sinus node from ectopic atrial pacemakers; appear interspersed throughout an underlying rhythm (usually sinus)
2. These ectopic P waves are different in configuration from normal P waves and may or may not be conducted through the AV node.
3. Usually have a normal PR interval (0.12–0.20 seconds)
4. Generally followed by a noncompensatory pause; the SA node is reset, and the returning sinus beat occurs ahead of schedule.
5. There are multiple causes, but PACs may also occur as a normal variant.
   a. Myocardial ischemia
   b. Hyperthyroidism
   c. Sympathomimetic drugs and medications, ie, stimulants
   d. Metabolic derangements
6. Clinical significance
   a. Can precipitate supraventricular tachycardia, atrial fibrillation, and atrial flutter
   b. Most frequent cause of a pause on the ECG
7. In general, no treatment is indicated. If, however, the PACs are frequent or symptomatic, treatment should be directed toward correcting the underlying cause.

C. Sinus tachycardia

1. ECG features: identical to sinus rhythm except that the rate is >100 (and usually <160) beats per minute

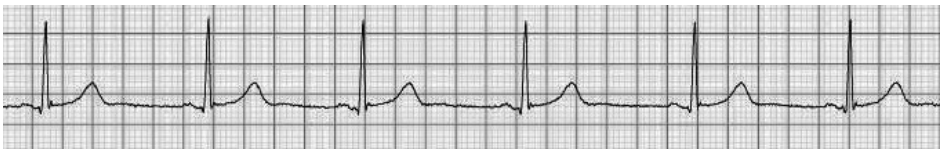


Courtesy of Michael McCrea, MD

2. Etiologies
   a. Stimulant or sympathomimetic drugs (eg, cocaine)
   b. Fever
   c. Hypovolemia
   d. Hyperthyroidism
   e. Pulmonary embolism
   f. Anemia
   g. Hypoxia
   h. Pain
   i. Anxiety (diagnosis of exclusion)
3. Management in most instances should be directed at finding and correcting the underlying cause.
   a. Of note, in the setting of cocaine or other stimulant toxicity, administration of a benzodiazepine is first-line treatment (see pages 598–599).

D. Sinus bradycardia

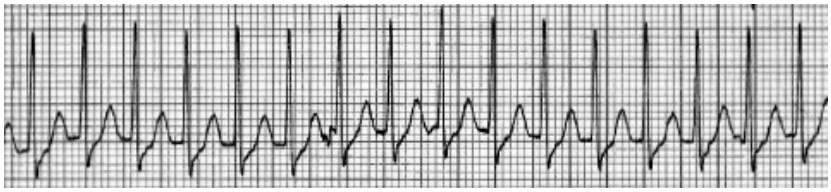
1. ECG features: identical to sinus rhythm except that the rate is <60



Courtesy of Michael McCrea, MD

2. Etiologies
   a. Acute inferior wall MI
   b. Vasovagal events (eg, vomiting)
   c. Drug effect (eg, β-blockers, calcium channel blockers, digoxin, clonidine)
   d. Sick sinus syndrome
   e. Hypothermia
   f. Hypothyroidism
   g. Hyperkalemia
   h. Increased intracranial pressure
   i. 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 heart failure
   b. Atropine should generally be reserved for patients with bradycardias due to excessive vagal tone, which most often will include sinus bradycardia, junctional rhythms, and Mobitz I conduction; it will often be ineffective when used for other types of bradycardia (eg, hyperkalemic bradycardia, Mobitz II conduction, etc).
   b. Intervention sequence
      (1) New in the 2020 ACLS Guidelines, atropine 1 mg IV every 3–5 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 induce tachycardia, which could worsen cardiac ischemia
         (b) Should not be used in patients with Mobitz Type II second-degree AV block and new third-degree AV block with wide complexes → ↑ atrial rate (but not the ventricular rate) → ↑ AV block
         (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; therefore, the generally recommended minimum dose in adults is 0.5 mg.
      (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.

E. Supraventricular tachycardia



Courtesy of Daniel Schwerin, MD

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.
   a. These arise from either reentry circuits or an ectopic pacemaker in the atria.
   b. Most clinicians, however, use the term "SVT" to refer specifically to AV nodal reentry tachycardia (AVNRT).
   c. In the discussion that follows, *"SVT" refers to only AVNRT*. Management is discussed separately for other specific types of supraventricular tachycardias, such as atrial fibrillation.
2. ECG features
   a. P waves are abnormal and may not be visible (often hidden in the QRS complex or the preceding T wave), or they may immediately follow the QRS complex, in which case they are often inverted ("retrograde P waves").
   b. Atrial AND ventricular rate is 120–200 beats per minute.
   c. Rhythm is regular.
   d. QRS complexes are usually narrow but may be wide if aberrant conduction through a bypass tract or bundle branch block is present.
3. Etiologies
   a. Preexcitation syndromes (Wolff-Parkinson-White and Lown-Ganong-Levine)
   b. Mitral valve disease
   c. Digitalis toxicity
   d. Drugs (eg, alcohol, tobacco, caffeine)
   e. Acute MI and pericarditis
   f. Hyperthyroidism
   g. Rheumatic heart disease
4. Management
   a. Determined primarily by the patient's hemodynamic stability and secondarily by the width of the QRS complex
   b. 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 immediate synchronized cardioversion.
   c. 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.
      (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. However, recurrence of SVT is common, seen in 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. Smaller doses should also be used in patients with transplanted hearts.
      (3) Calcium channel blockers (eg, diltiazem, verapamil) are as effective as adenosine but are slower in onset and have more significant adverse effects (decreased myocardial contractility and peripheral vasodilation). Calcium channel blockers 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) have been suggested to prevent 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.
   (a) Esmolol has the advantage of being cardioselective as well as having a very short half-life.
   (b) Propranolol is the drug of choice for SVT secondary to thyrotoxicosis because it partially blocks the conversion of T_4 and T_3.
   (c) Avoid β-blockers in patients with acute reactive airway disease or acute heart failure 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 ideally should be avoided if cardioversion is being considered.

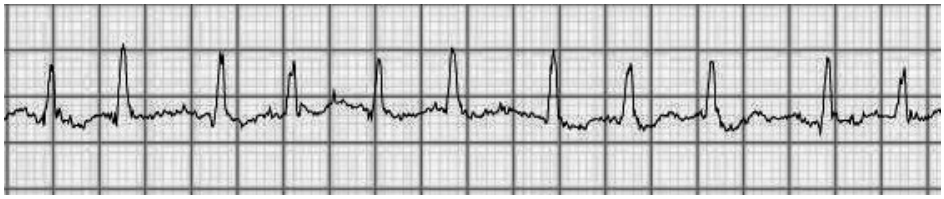
(6) Magnesium sulfate, phenytoin, and lidocaine are the drugs of choice for ectopic SVT caused by digitalis toxicity. Management should also include correction of hyper/hypokalemia (if present) and discontinuation of digitalis. In the presence of hemodynamic instability (or potentially lethal digitalis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

F. Atrial fibrillation

1. An irregularly irregular rhythm due to uncoordinated atrial activation and random occurrence of ventricular depolarization. The atria are not contracting in a coordinated fashion, 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 Michael McCrea, MD

2. ECG findings
   a. P waves are absent but small irregular deflections in the baseline ("f" or "fibrillation waves") may be seen. The atrial rate is > 350 beats per minute.
   b. Because distinct regular P waves are not visible, there is no PR interval.
   c. QRS complexes are normal in configuration unless there is aberrant conduction.
   d. The rhythm is irregularly irregular.
   e. Ventricular response rate is variable but is generally 140–180 beats per minute in untreated patients; a rate >200 beats per minute with QRS complexes of variable width (some narrow, some wide) suggests Wolff-Parkinson-White syndrome with conduction through the accessory pathway; on the other hand, a slow, irregular ventricular rate may be found in patients with hypothermia or in the setting of toxicity from AV node–blocking medications (eg, calcium channel blockers, β-blockers, digitalis).
3. Etiologies
   a. Search for reversible causes and treat any underlying medical condition; then determine the risk of subsequent stroke.
   b. Conditions with high risk of cardiogenic thromboembolism
      (1) Cardiac surgery
      (2) Acute MI
      (3) Hyperthyroidism
      (4) Myocarditis
      (5) Acute pulmonary disease
4. Management
   a. Plan the treatment using the following criteria:
      (1) Cardiovascular stability
      (2) Duration of the dysrhythmia

(3) Underlying cause/condition
(4) Presence/absence of an accessory pathway

b. 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.

(1) Unstable patients → immediate electrical synchronized cardioversion

(2) Stable patients can be treated via rate control or rhythm control (cardioversion with electricity or medications).

(a) Rate control options (choose *one*):

i. Calcium channel blocker (eg, verapamil, diltiazem)
ii. β-blocker (eg, esmolol, atenolol, metoprolol)
iii. Digoxin
iv. Amiodarone
v. The presence of Wolff-Parkinson-White syndrome is a special circumstance requiring changes in treatment protocols (see pages 30–31).

(b) Rhythm control (cardioversion)

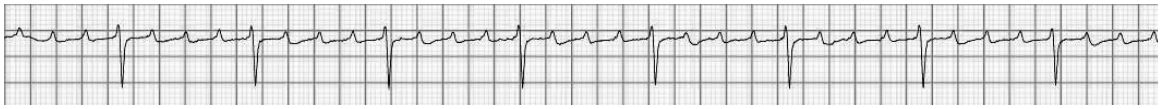
i. Should be reserved for patients with presumed duration of the dysrhythmia (based on duration of symptoms) of <48 hours
- Normal cardiac function: perform electrical cardioversion or pharmacologic cardioversion using one of the following agents:
  - Amiodarone: agent of choice with structural heart disease or ischemia
  - Ibutilide: highest conversion rates but avoid with structural heart disease or ischemia
  - Flecainide
  - Propafenone
  - Procainamide: agent of choice if known or suspected accessory pathway conduction
- Compromised cardiac function: perform synchronized cardioversion or use amiodarone for pharmacologic cardioversion.

ii. >48 hours duration carries a higher risk of systemic embolization
- Avoid immediate cardioversion if possible unless the patient is hemodynamically unstable.
- If early cardioversion (within 24 hours) is anticipated, consider starting heparin and consulting cardiology for transesophageal echocardiography to exclude an atrial thrombus before cardioversion is performed.
- If delayed cardioversion is the best option, anticoagulation for 3 weeks is indicated before cardioversion.

G. Atrial flutter

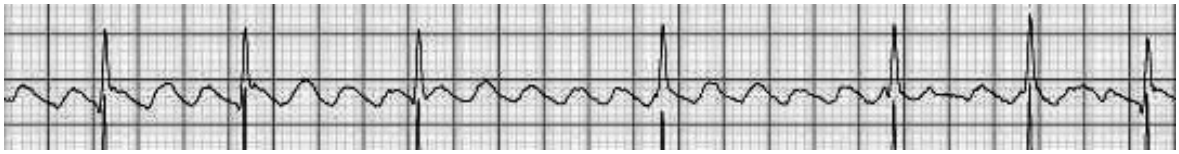
1. A very rapid atrial rhythm but, because of AV nodal delay, ventricular responses are slower. Therefore, atrial flutter usually occurs with some sort of AV block (not all impulses are conducted); the resulting block is either a fixed ratio (2:1, 3:1, 4:1, etc) or variable AV block.

Atrial flutter with 4:1 AV block



Courtesy of Michael McCrea, MD

Atrial flutter with variable AV block



Courtesy of Michael McCrea, MD

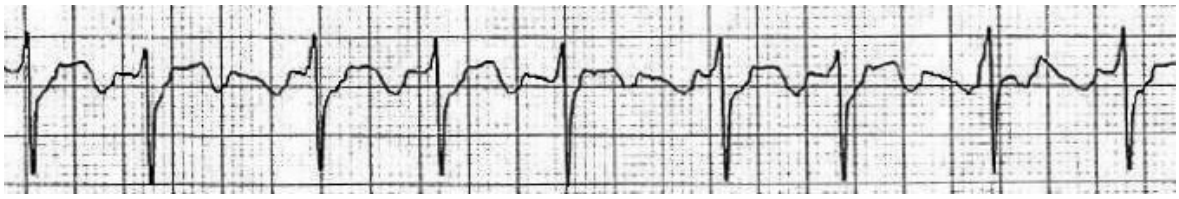
2. ECG features
   a. P waves have a characteristic sawtooth pattern and are called "F" or "flutter waves." They are usually best seen in the inferior leads and leads V_1 and V_2. The atrial rate is 250–350 beats per minute.

b. QRS complexes are normal in configuration.
c. 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.

3. Etiologies
   a. Causes are similar as for atrial fibrillation
   b. Often is a transitional rhythm between sinus rhythm and atrial fibrillation
4. Management
   a. Determined by the patient's cardiovascular stability, duration of the dysrhythmia and if the patient has a known accessory pathway.
   b. Hemodynamically unstable patients should be sedated (if time permits) and treated with immediate synchronized cardioversion.
   c. Hemodynamically stable patients can be managed with rate control or rhythm control, similar to atrial fibrillation.
      (1) Vagal maneuvers or adenosine may be of diagnostic value when there is uncertainty between SVT and atrial flutter; by inducing a transient AV nodal blockade, atrial flutter will demonstrate transient slowing of the ventricular response and thus reveal the characteristic flutter waves of this rhythm and confirm the diagnosis.

H. Multifocal atrial tachycardia

1. An irregular rhythm sometimes mistaken for atrial fibrillation; originates from multiple different atrial sites and is characterized by P waves of varying morphologies

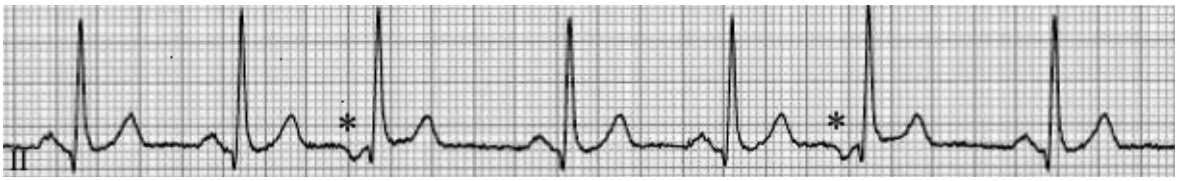


Courtesy of Daniel Schwerin, MD

2. ECG features
   a. There must be at least 3 distinct types of P waves in one lead; atrial rate is 100–180 beats per minute.
   b. The rhythm is irregularly irregular.
   c. The PP, PR, and RR intervals vary.
   d. Nonconducted (blocked) P waves are frequently present, particularly when the atrial rate is rapid. Classically seen with COPD and theophylline toxicity.
3. Etiologies
   a. Decompensated COPD (most common)
   b. CHF
   c. Sepsis
   d. Theophylline toxicity
   e. Asthma
   f. Other pulmonary diseases
4. Management is directed at treatment of the underlying condition. Cardioversion is ineffective. The rhythm itself should not cause hemodynamic instability.
   a. Correct hypoxia with supplemental oxygen (and bronchodilator therapy) in a patient with COPD, and
   b. Evaluate for theophylline toxicity in the right clinical setting
   c. If the above measures are unsuccessful and the patient is symptomatic, other modalities that may be used:
      (1) Calcium channel blockers (eg, diltiazem, verapamil) are usually effective in slowing the ventricular rate and decreasing atrial ectopy and may produce conversion to a sinus rhythm in some patients.
      (2) Magnesium sulfate may decrease atrial ectopy.
      (3) β-blockers are generally not recommended because they may worsen the underlying pulmonary disease process.
   d. Digoxin and cardioversion are usually ineffective and are not recommended.

I. Junctional premature contractions

1. 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.

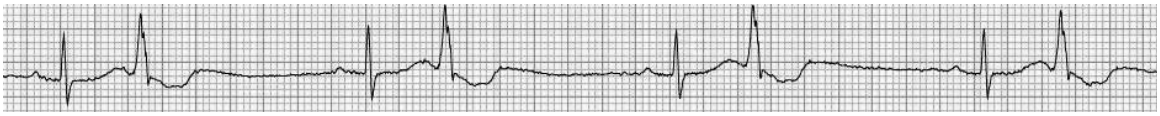


Courtesy of Michael McCrea, MD

2. ECG findings
   a. 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.
   b. When the P wave precedes the QRS, the PR interval is shorter than normal (often <0.12 seconds).
   c. The ectopic QRS complex is premature but has a normal shape unless there has been aberrant conduction.
   d. They are generally followed by a compensatory pause; the SA node is not reset, and the next P wave occurs at its usual time.
3. Etiologies
   a. Digitalis toxicity
   b. Coronary artery disease
   c. CHF
   d. Acute MIs (especially inferior wall MIs)
4. Management
   a. Treat the underlying cause.
   b. If junctional premature contractions precipitate more lethal dysrhythmias, consider using IV procainamide.

J. Premature ventricular contractions (PVCs)

1. Appear as abnormal QRS complexes and T waves that occur in addition to the underlying rhythm



Courtesy of Michael McCrea, MD

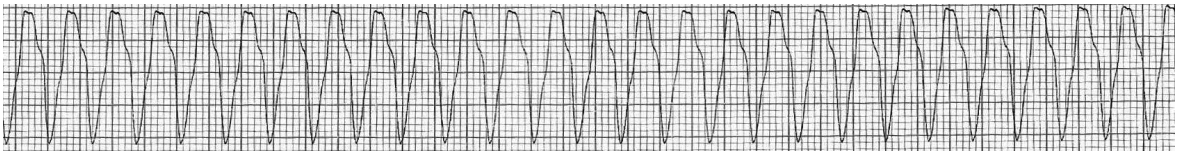
2. ECG findings
   a. Occur earlier than the next expected normal QRS
   b. Wider than a normal QRS (usually ≥0.12 second)
   c. The QRS morphology is generally bizarre.
   d. A preceding P wave is absent; however, retrograde conduction of a premature ventricular contraction can occasionally result in a 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.
3. Etiologies
   a. Hypokalemia
   b. Hypomagnesemia
   c. Hypoxia
   d. MI
   e. Drugs
      (1) Alcohol, tobacco, caffeine
      (2) Cocaine
      (3) Digitalis or quinidine toxicity (PVCs are the most common dysrhythmia with digitalis toxicity)
      (4) Methylxanthines (commonly used by patients with asthma or COPD)
   f. Hyperthyroidism

g. CHF
h. Cardiomyopathy
i. Mechanical: PVCs are not uncommon when a catheter is placed in the right ventricle.
j. Myocardial contusion

4. Management is dictated by the underlying cause and is not indicated in all cases.
   a. Patients who are asymptomatic and have PVCs of unknown cause should not be treated (particularly when the PVCs are an incidental finding); PVCs in this setting may represent a normal variant. Isolated PVCs occur in as many as 50% of young healthy patients and increase in frequency with age.
   b. When an underlying cause is identified, therapy should be directed toward correcting the underlying problem rather than suppressing PVCs. This is usually sufficient for resolution of PVCs.
   c. Mechanical causes, if present, should also be corrected.
      (1) A central line catheter located in the right ventricle may induce PVCs; withdraw the catheter or advance it out of the ventricle.
      (2) If these measures do not ameliorate the PVCs and the patient is symptomatic, lidocaine may be administered.
   d. "Escape" PVCs (those associated with bradycardia) should be treated with atropine (not lidocaine) because administration of lidocaine under these circumstances may suppress the existing functioning rhythm.
   e. Management of PVCs occurring in association with an acute MI or ischemia is more controversial. Optimal treatment of the underlying ischemia/infarction with oxygen, nitroglycerin, aspirin, and percutaneous coronary intervention is clearly the first priority. If these measures fail, most authors currently recommend a conservative course of watchful waiting.
   f. In general, the identification of PVCs should prompt a search for and treatment of the underlying cause, rather than simply pharmacologic suppression of the PVCs. If short bursts of frequent PVCs are occurring, consider empiric administration of β-blockers (to decrease adrenergic stimulation that often causes ventricular dysrhythmias).
   g. Pharmacologic agents
      (1) Lidocaine has historically been used as a first-line agent to suppress PVCs, though it is rarely used empirically now. Studies have not shown any benefit to suppressing ventricular ectopy in the absence of sustained ventricular tachycardia. Instead, search for and treat underlying causes.
      (2) Magnesium sulfate is sometimes effective in decreasing the frequency of PVCs, but it is most useful if the patient has hypomagnesemia.

K. Ventricular tachycardia

1. Three or more consecutive premature ventricular contractions occurring at a regular rhythm and rate >120 beats per minute

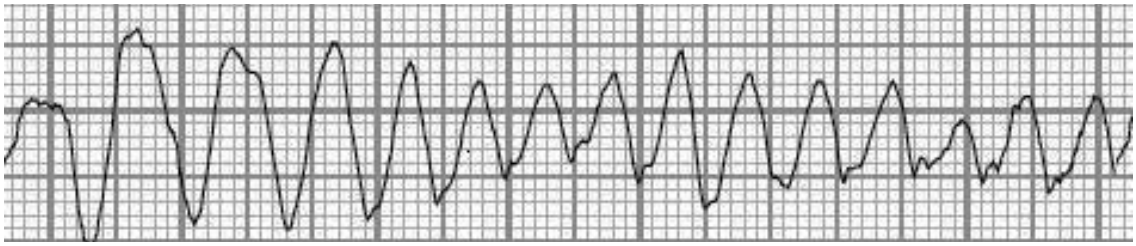


Courtesy of Michael McCrea, MD

2. ECG findings
   a. P waves are usually absent; when present, they are either retrogradely conducted or have no relationship to the QRS (AV dissociation).
   b. QRS complexes are wide (≥0.12 second) and may be bizarre.
   c. 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.
   d. 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.
   e. Deflection of the ST segment and T wave is generally opposite that of the QRS complex.
   f. Rate is >120 (usually 150–200) beats per minute.
   g. Rhythm is generally regular, although beat-to-beat variation may occur at the onset of tachycardia.
   h. QRS axis is generally constant.

i. Ventricular tachycardia is classified as:
   (1) "Monomorphic": *all* QRS complexes look the same
   (2) "Polymorphic": QRS complexes have varying morphologies
   (3) Current therapeutic modalities are based on this classification (see Specific Rhythm Assessments, pages 15–31).

3. Differentiation of supraventricular tachycardia with aberrancy from ventricular tachycardia
   a. 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 diagnostic of ventricular tachycardia.
   c. 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.
   d. Age ≥50 years old favors ventricular tachycardia, whereas age ≤35 years old favors an aberrant supraventricular tachycardia.
4. 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. 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, flecainide), tricyclic antidepressants, methadone, 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 Daniel Schwerin, MD

5. Etiologies
   a. The causes are basically the same as those for PVCs. Torsades, however, is typically caused by factors that produce a prolonged QT interval.
   b. The most common causes are ischemic heart disease and acute MI.
6. Management depends on the status of the patient and QRS morphology.
   a. If there is no pulse for either monomorphic or polymorphic ventricular tachycardia, treat like ventricular fibrillation: begin CPR and defibrillate as soon as a defibrillator is available. Start with 200 Joules.
   b. If the patient has a pulse but is hemodynamically unstable (hypotensive, decreased mental status, ischemic chest pain, acute CHF), immediate synchronized cardioversion is indicated. Sedate first if time allows, but do not delay cardioversion for sedation. Start with 100 Joules and increase energy if unsuccessful.
   c. If the patient is clinically stable (awake, pain free, and normotensive), pharmacologic therapy is appropriate. Patients who do not convert with these agents should be sedated and synchronize cardioverted. If the patient is stable, the following are recommended:
      (1) Monomorphic ventricular tachycardia: procainamide, amiodarone, or sotalol
         (a) Procainamide 20–50 mg/min until rhythm is suppressed, QRS widens by more than 50%, hypotension develops, or maximal bolus dose of 17 mg/kg is reached.
         (b) Amiodarone 150 mg IV bolus over 10 minutes, followed by maintenance infusion of 1 mg/min for 6 hours.
         (c) Sotalol 100 mg (1.5 mg/kg) over 5 minutes; avoid if prolonged QT.
         (d) *Note:* Adenosine will convert some forms of monomorphic ventricular tachycardia to a sinus rhythm; therefore, use of adenosine as the sole diagnostic measure to distinguish ventricular tachycardia from SVT with aberrancy is not recommended.
      (2) Polymorphic ventricular tachycardia
         (a) If the baseline QT interval is normal, all the above agents are considered reasonable.