ACLS Study Guide

This purpose of this study guide is to assist you in successfully completing the AHA ACLS course. It includes sections on:

- ECG Rhythm Interpretation
- ACLS Drugs
- ACLS Algorithms

ECG Rhythm Interpretation

Electrical Conduction System

⇒ **SA Node**. Primary pacemaker. Rate 60-100

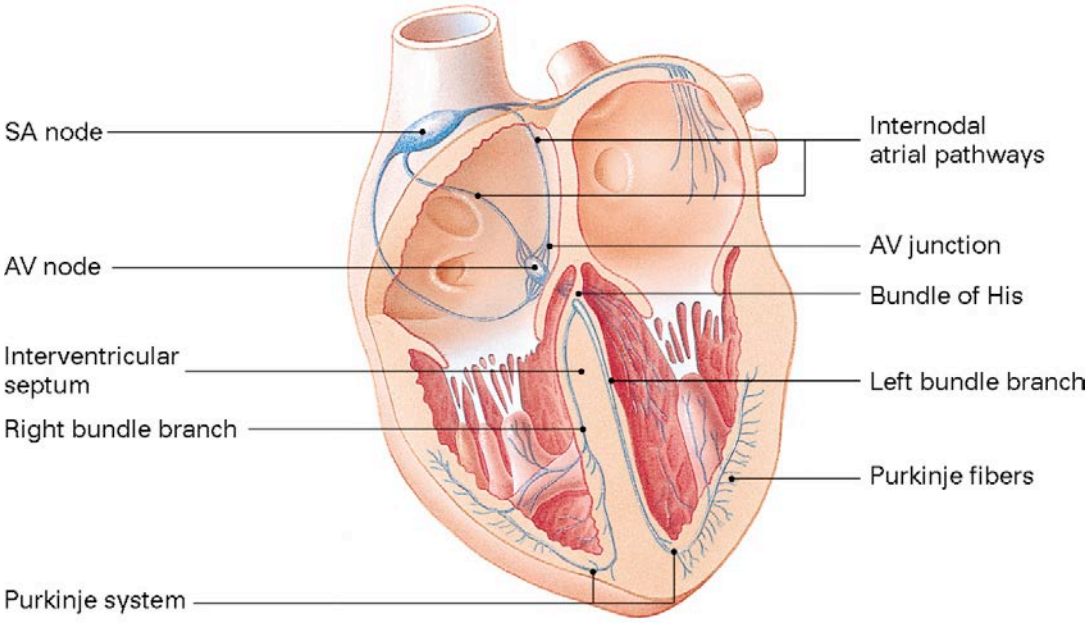
⇒ The impulse travels through the **Intraatrial Pathways** to innervate the atria

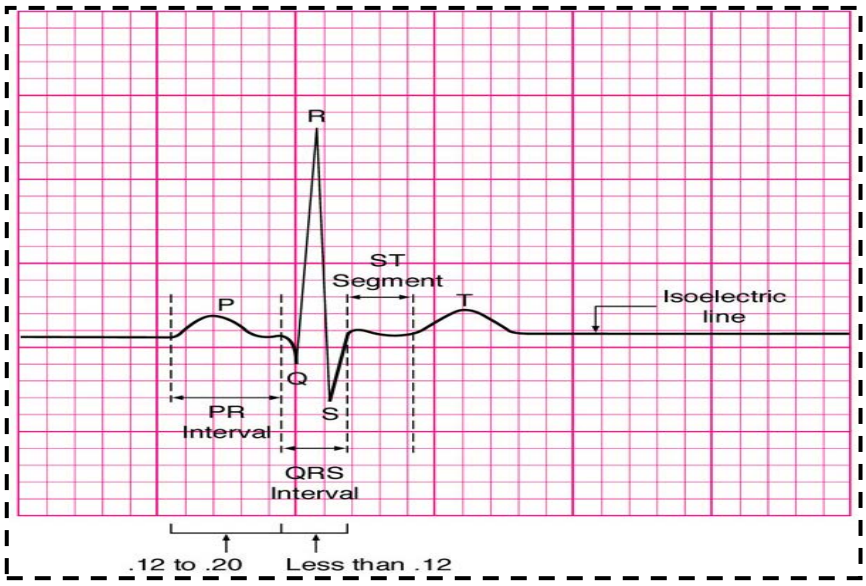
⇒ The impulse reaches the **AV Node** where electrical activity is delayed to allow for more complete filling of ventricles.

⇒ **AV Junction** is comprised of the AV Node and the Bundle of His. Secondary pacemaker. Rate 40-60

⇒ The impulse then travels into the **Right and Left Bundle branches.** Conducts electrical activity from Bundle of His to Purkinje Network.

⇒ The **Purkinje Network** are fibers that spread throughout the ventricles, that carry impulses directly to ventricular muscle cells. Our last pacemaker site. Rate 20-40





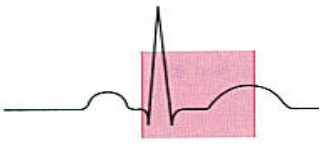
P wave: • Represents Atrial depolarization

PRI: • Represents the time it takes the impulse to travel from the SA Node through the intraatrial pathways in atria to the AV junction and the delay at the AV node.
• Interval from start of P wave to start of QRS, measures 0.12-0.20 sec

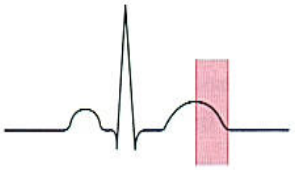
QRS: • Represents conduction of impulse from Bundle of His through the ventricular muscle. Represents ventricular depolarization.
• Should measure less than 0.12 sec

T wave: • Follows ST segment. Slightly rounded, positive deflection
• Represents ventricular repolarization, **"**resting phase "of cardiac cycle

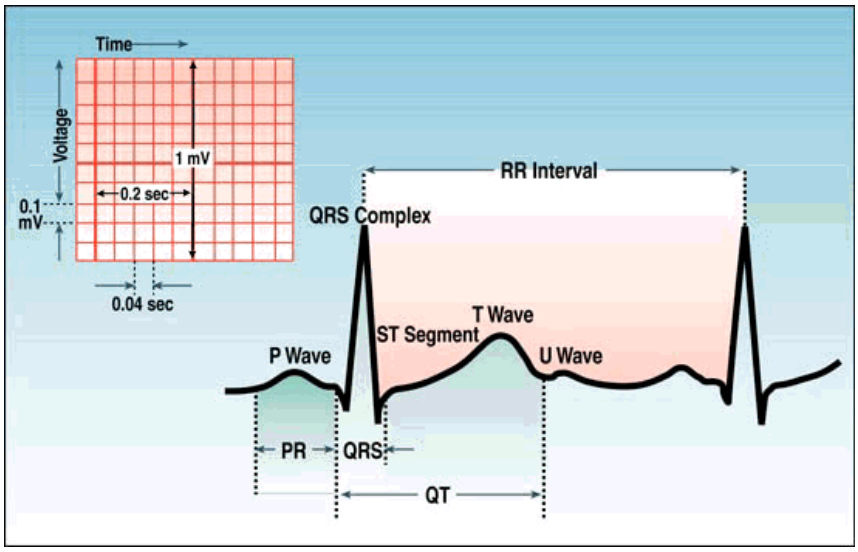
Absolute Refractory Period:
•No outside stimulus can cause cells to depolarization
•From beginning of the QRS complex to the middle of the T wave



Relative Refractory Period:
•A dangerous period. A strong outside stimulus can initiate depolarization of the only partially recharged cells. Possibly causing a lethal arrhythmia
•From the middle of the T wave to its end



Relative Refractory Period



5 Steps for Analyzing a Strip:

Heart Rate: Bradycardia <60, Normal 60-100, Tachycardia >100

- ⇒ Count the # of R waves in a 6 second rhythm strip, then multiply by 10
- ⇒ Find an R wave that lands on a bold line. Count the # of large boxes to the next R wave. If the second R wave is 1 large box away the rate is 300, 2 boxes - 150, 3 boxes - 100, 4 boxes - 75, 5 boxes – 60
- ⇒ Divide 300 by the number of large boxes separating the R waves

Heart Rhythm:

- ⇒ Look at the R – R distances, are they regular or irregular

P Wave:

- ⇒ Are there P waves?
- ⇒ Do the P waves all look alike?
- ⇒ Do the P waves occur at a regular rate?
- ⇒ Is there one P wave before each QRS

PR Interval:

- ⇒ Is the PRI between 0.12-0.20?
- ⇒ Is it consistent across the strip?
- ⇒ If it varies is there a pattern?

QRS Complex:

- ⇒ Do all of the QRS Complexes look alike?
- ⇒ Are they regular?
- ⇒ Is the duration 0.04 – 0.12

Normal Sinus Rhythm

This rhythm represents the normal state with the SA node functioning as the lead pacer with normal conduction through the heart. The intervals should all be consistent and within normal ranges.

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - (60-100 bpm)
- QRS Duration - Normal
- P Wave - Visible before each QRS complex
- P-R Interval - Normal (<5 small squares. Anything above and this would be 1st degree block)
- Indicates that the electrical signal is generated by the sinus node and travelling in a normal fashion in the heart.

Sinus Bradycardia

The sinus beats are slower than 60 BPM. The origin may be in the SA node or in an atrial pacemaker. This rhythm can be caused by vagal stimulation leading to nodal slowing, or by medicines such as beta blockers, and is normally found in some well-conditioned athletes. The QRS complex, and the PR interval may slightly widen as the rhythm slows below 60 BPM. However, they will not widen past the upper threshold of the normal range for that interval. For example, the PR interval may widen, but is should not widen over the upper of 0.20 seconds

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - less than 60 beats per minute
- QRS Duration - Normal
- P Wave - Visible before each QRS complex
- P-R Interval - Normal
- Usually benign and often caused by patients on beta blockers

Sinus Tachycardia

It is an excessive heart rate above 100 beats per minute (BPM) which originates from the SA node. Causes include stress, fright, pain, dehydration, and exercise. Not usually a surprise if it is triggered in response to regulatory changes (e.g. shock).

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate – Usually between 100 – 150 beats per minute
- QRS Duration - Normal
- P Wave - Visible before each QRS complex
- P-R Interval - Normal
- The impulse generating the heart beats are normal, but they are occurring at a faster pace than normal. Seen during exercise

Atrial Flutter

A single irritable focus in the atria fires in a rapid repetitive fashion at a rate of 150 – 350 beats/min. The F waves appear in a saw toothed pattern such as those in this ECG. The QRS rate is usually regular and the complexes appear at some multiple of the P-P interval.

Looking at the ECG you'll see that:

- Rhythm – Usually regular
- Rate – Usually fast 110-150 beats per minute
- QRS Duration - Usually normal
- P Wave - Replaced with multiple F (flutter) waves, usually at a ratio of 2:1 (2F - 1QRS) but sometimes 3:1
- P Wave rate - 300 beats per minute
- P-R Interval - Not measurable

Atrial Fibrillation

Atrial fibrillation is the chaotic firing of numerous atrial pacemaker cells in a totally haphazard fashion. The result is that there are no discernible P waves. And the QRS complexes are innervated haphazardly in an irregularly irregular pattern. The ventricular rate is guided by occasional activation from one of the pacemaking sources. Because the ventricles are not paced by anyone site, the intervals are completely random.

Looking at the ECG you'll see that:

- Rhythm - Irregularly irregular
- Rate - usually 100-160 beats per minute but slower if on medication
- QRS Duration - Usually normal
- P Wave - Not distinguishable as the atria are firing off all over
- P-R Interval - Not measurable
- The atria fire electrical impulses in an irregular fashion causing irregular heart rhythm

Supraventricular Tachycardia (Narrow complex Tachycardia) (SVT)

SVT is a narrow complex tachycardia originating above the ventricles. SVT can occur in all age groups.

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - > 150 beats per minute
- QRS Duration - Usually normal
- P Wave - Often buried in preceding T wave
- P-R Interval - Depends on site of supraventricular pacemaker

1st Degree AV Block

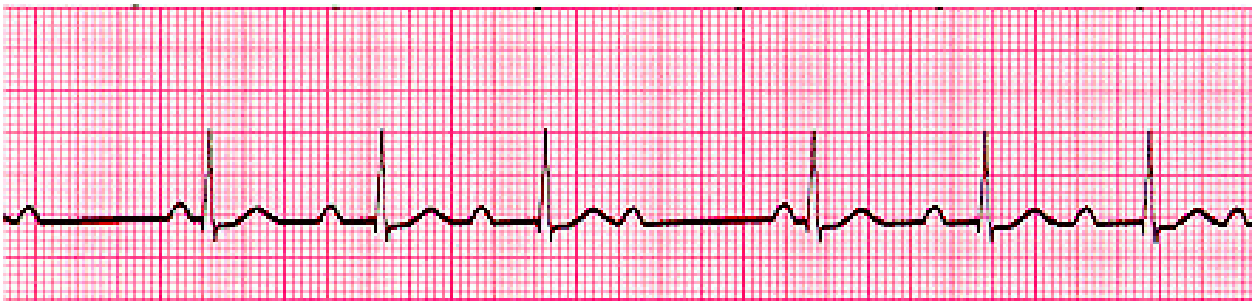
1st Degree AV block is caused by a conduction delay through the AV node but all electrical signals reach the ventricles. This rarely causes any problems by itself and often trained athletes can be seen to have it. The normal P-R interval is between 0.12s to 0.20s in length, or 3-5 small squares on the ECG.

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - Normal
- QRS Duration - Normal
- P Wave - Ratio 1:1
- P Wave rate - Normal
- P-R Interval - Prolonged (>5 small squares)

2nd Degree Block Type 1 (Wenckebach)

Mobitz Type I is also know as Wenckebach (pronounced WEEN-key-bock). It is caused by a diseased AV node with a long refractory period. The result is that the PR interval lengthens between successive beats due to increasing delayed conduction through the AV junction until a beat is dropped. At that point, the cycle starts again.



Looking at the ECG you'll see that:

- Rhythm - Regularly irregular
- Rate - Normal or Slow
- QRS Duration - Normal
- P Wave - Ratio 1:1 for 2,3 or 4 cycles then 1:0.
- P Wave rate - Normal but faster than QRS rate
- P-R Interval - Progressive lengthening of P-R interval until a QRS complex is dropped

2nd Degree Block Type 2

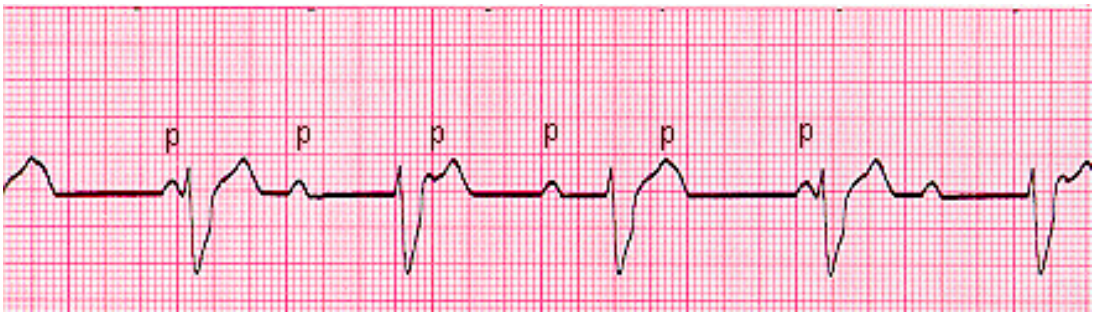
In 2nd degree Type 2, the impulse either passes through the AV junction normally or it is blocked completely. It is an all or nothing type of thing. Beats are intermittently nonconducted and QRS complexes dropped, usually in a repeating cycle of every 3rd (3:1 block) or 4th (4:1 block) P wave

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - Normal or Slow
- QRS Duration - Prolonged
- P Wave - Ratio 2:1, 3:1
- P Wave rate - Normal but faster than QRS rate
- P-R Interval - Normal or prolonged but constant

3rd Degree Block

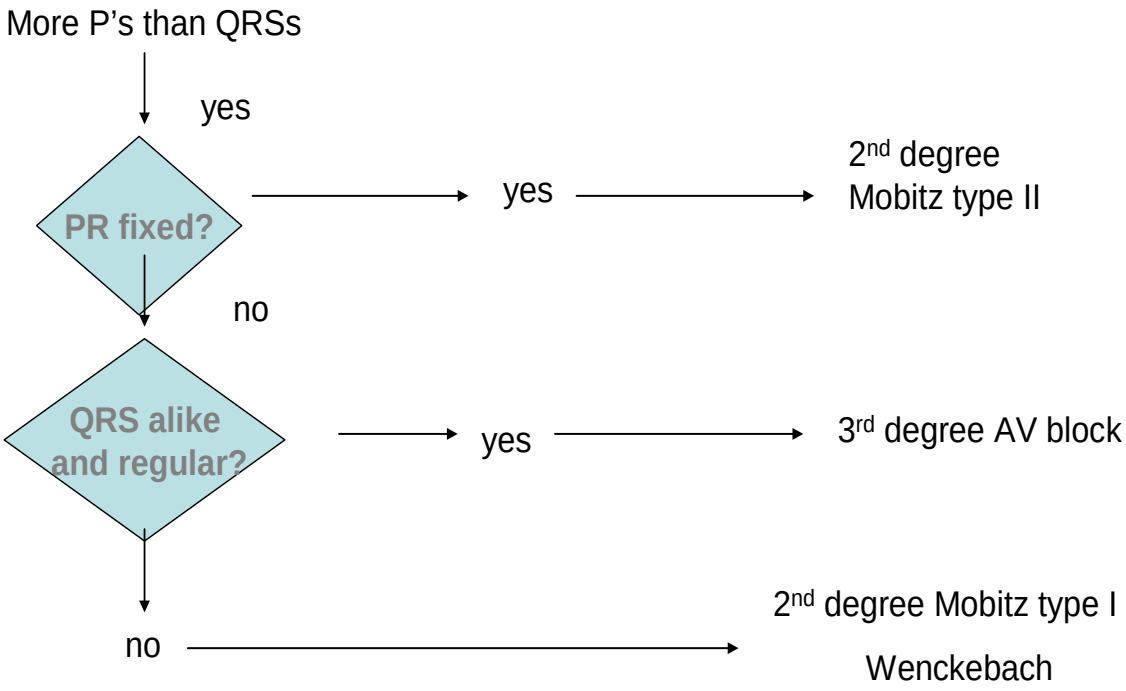
3rd degree block or complete heart block occurs when the impulse travels through the atria normally but is blocked completely at the junction. The atria and ventricles are firing separately – each to its own drummer, so to speak. The atrial rhythm can be bradycardic, normal or tachycardic. The escape beat can be junctional (normal QRS) or ventricular (wide QRS).



Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - Slow
- QRS Duration – Usually wide, but if ventricular impulse is generated low in the junction it could be normal.
- P Wave - Unrelated
- P Wave rate - Normal but faster than QRS rate
- P-R Interval - Variation

Differentiation of Second- and Third-Degree AV Blocks



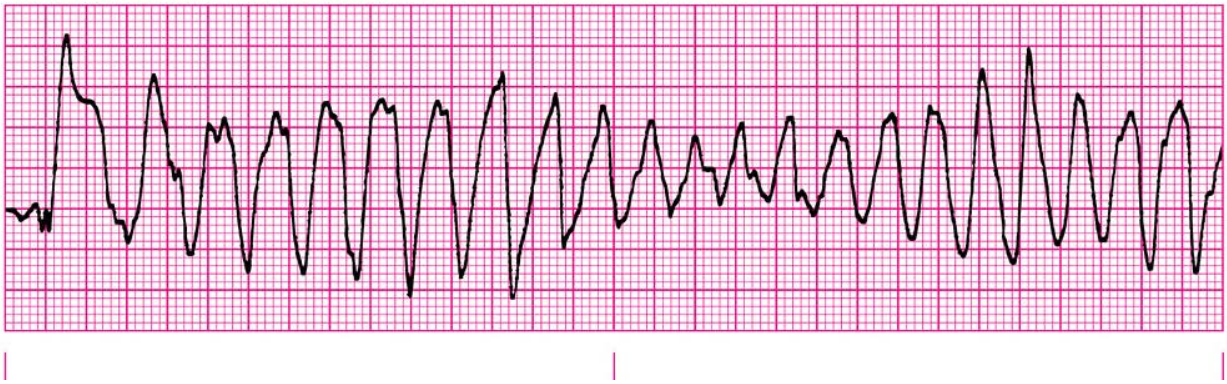
Wide Complex Tachycardia (usually monomorphic ventricular tachycardia) Abnormal

Ventricular tachycardia is simply the presence of three or more ectopic ventricular complexes in a row with a rate above 100. Originates from one irritable focus so the rhythm is regular. Poor cardiac output is usually associated with this rhythm

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate – Fast usually 180-190 Beats per minute
- QRS Duration - Prolonged
- P Wave - Not seen
- Results from abnormal tissues in the ventricles generating a rapid and irregular heart rhythm.

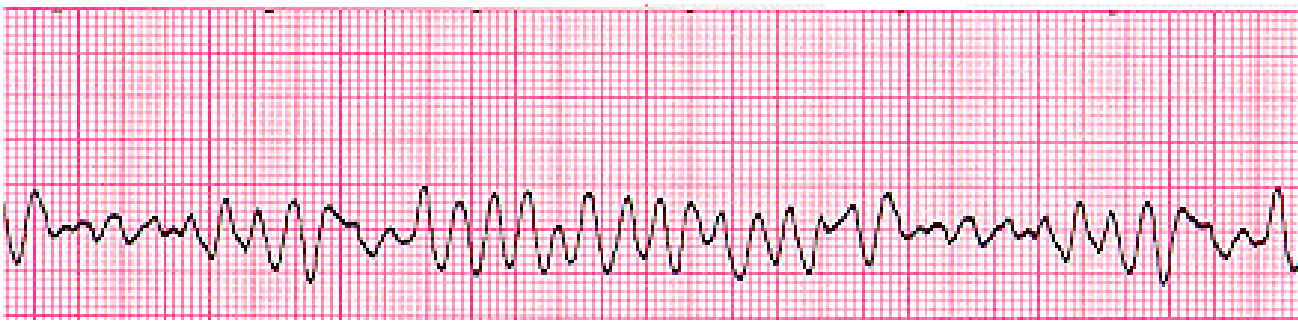
Polymorphic V-Tach (Torsades de Pointes)



- Similar to ventricular tachycardia
- Morphology of QRS complexes shows variations in width and shape
- Resembles a turning about or twisting motion along base line
- May result from hypokalemia, hypomagnesemia, tricyclic antidepressant drug overdose, the use of antidysrhythmic drugs, or combination of these
- Seen in alcoholics, eating disorders and the debilitated patients

Ventricular Fibrillation (VF)

Disorganized electrical signals cause the ventricles to quiver instead of contract in a rhythmic fashion. A patient will be unconscious as there is no cardiac output and blood is not pumped to the brain. Immediate treatment by defibrillation is indicated. This condition may occur during or after a myocardial infarct.



Looking at the ECG you'll see that:

- Rhythm - Irregular
- Rate - 300+, disorganized
- QRS Duration - Not recognizable
- P Wave - Not seen
- This patient needs to be defibrillated!! QUICKLY

Pulseless Electrical Activity (PEA)

PEA occurs when any heart rhythm (other than V-Tach or V- Fib) is observed on the monitor and does not produce a pulse. PEA can be any rhythm (sinus, bradycardia, tachycardia). There is organized electrical activity without a pulse.

- Prognosis for PEA invariably is poor unless an underlying cause can be identified and corrected
- The highest priority of care is to maintain circulation for the patient with basic and advanced life support techniques while searching for a correctable cause

Asystole – Abnormal

Asystole refers to the absence of any electrical cardiac activity. It is defined by < 10 non-perfusing complexes per minute

Looking at the ECG you'll see that:

- Rhythm - Flat or an occasional p wave or QRS complex. The QRS complexes when they occur are wide and bizarre
- Rate - 0 Beats per minute
- QRS Duration - None
- P Wave - None

ACLS Drugs

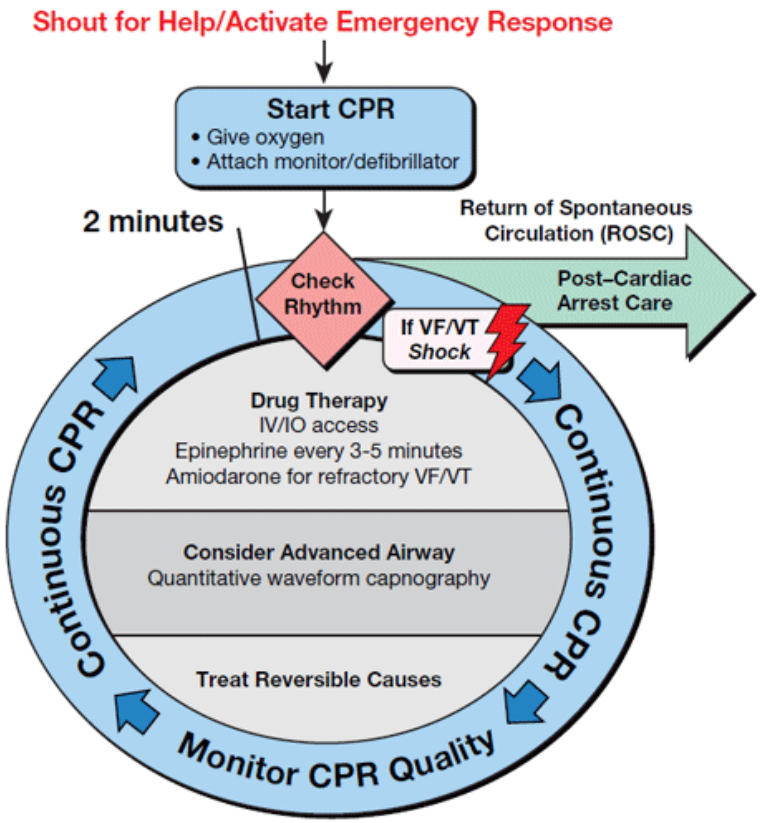
Drug	Action	Indication	Precautions/ Contraindications	Dosage
Adenosine	Slows conduction through the AV node. Can interrupt reentry pathways in the AV node. Negative chronotropic/dromatropic. Very short half live,	Stable narrow complex SVT unresponsive to vagal maneuvers. May consider for unstable narrow-complex reentry tachycardia while preparations are made for cardioversion. Regular and monomorphic wide-complex tach thought to be or previously defined to be reentry SVT.	Transient side effects include flushing, chest pain or tightness, brief periods of asystole or bradycardia, ventricular ectopy. Less effective in patients taking theophylline or caffeine. May cause bronchospasm, caution with asthma patients. Contraindicated in poison/drug-induced tachycardia or second or third degree heart block. Will not terminate atrial fib, atrial flutter or VT.	Initial bolus of 6mg given rapidly over 1 to 3 seconds followed immediately by a 20ml saline flush A second dose of 12 mg can be given in 1 to 2 minutes if needed *Reduce initial dose to 3mg in patients receiving dipyridamole (persantine) or carbamzepine (Tegretol), in heart transplant patients or if given by central venous access.
Amiodarone	Antidysrhythmic Prolongs duration of action potential and effective refractory period. Increases PR and QT intervals. Decreases sinus rate.	Stable VT (preferably after expert consult). Recurrent, unstable VT. VF/pulseless VT unresponsive to shock delivery, CPR and vasopressors.	Rapid infusion may lead to hypotension. Do not administer with other drugs that prolong QT interval. *Caution multiple complex drug interactions	VT with a pulse: 150mg IV in 50 ml piggyback over 10 minutes. VF/Pulseless VT: 300mg IV push, second dose if needed 150mg IV push.

Atropine	Anticholinergic – (para-sympathetic blocker) Increase heart rate and AV conduction. Dries secretions. Dilates bronchioles. Decreased GI motility.	First line drug for acute symptomatic bradycardia	Use atropine cautiously in the presence of acute coronary ischemia or MI. Do not rely on Atropine in Mobitz type II second or third degree AV block. Should not delay implementation of external pacing for patients with poor perfusion.	0.5mg IV every 3 to 5 minutes as needed, not to exceed total dose of 0.04mg/kg (total 3mg)
Diltiazem	Inhibits calcium ion influx across cell membrane during cardiac depolarization; produces relazation of coronary vascular smooth muscles, dilates coronary arteries, slows SA/AV node conduction times, dilates peripheral arteries	To control ventricular rate in atrial fibrillation and atrial flutter. Use after adenosine to treat refractory reentry SVT in patients with narrow QRS complex and adequate blood pressure	Do not use for wide-QRS tachycardias of uncertain origin or for poison/drug-induced tachycardias. Avoid use in patients with WPW. Blood pressure may drop.	15-20mg (0.25mg/kg) IV over 2 minutes. May give another IV dose in 15 minutes at 20 to 25 mg (0.35mg/kg) over 2 minutes
Dopamine	Causes increased cardiac output: acts on beta 1 and alpha receptors, causing vasoconstriction in blood vessels	Second-line drug for symptomatic bradycardia (after atropine). Use for hypotension with signs and symptoms of shock.	Correct hypovolemia with volume replacement first. May cause tachyarrhythmias and excessive vasoconstriction.	Usual infusion rate is 2 to 20mcg/kg/min Titrate to patient response

Epinephrine	Used during resuscitation primarily for is alpha adrenergic effects (vasoconstriction) increasing coronary and cerebral blood flow	Cardiac arrest: VF, pulseless VT, asystole, PEA. Symptomatic bradycardia after atropine as an alternative to dopamine. Severe hypotension when pacing and atropine fail. Anaphylaxis.	Raising BP and increasing HR may cause myocardial ischemia, angina and increased myocardial oxygen demand. High doses do not improve survival	Cardiac Arrest: 1mg (1:10,000) IV administered every 3 to 5 minutes during resuscitation. Follow each dose with 20 ml NS flush Profound bradycardia or hypotension: 2 to 10 mcg per minute infusion titrated to patient response
Magnesium	Reduces SA node impulse formation. Prolongs conduction time in myocardium	Recommended for use in cardiac arrest only if torsades de pointes or suspected hypomagnesemia is present. Life-threatening ventricular arrhythmias due to digitalis toxicity	Occasional fall in blood pressure with rapid administration. Use with caution if renal failure is present	Cardiac arrest (due to hypomagnesemia or Torsades de Pointes) 1 to 2 g diluted in at least 10ml of NS or D5W over 5 minutes Trosades de Pointes with a Pulse of AMI with hypomagnesemia: Loading dose of 1 to 2 Grams mixed in 50 to 100 ml NS or D5W over 5 to 60 minutes, follow with 0.5 to 1 gram per hour IV (titrate to control Torsades)

Sodium Bicarb	Alkalinizing agent – buffers acidosis	Known preexisting hyperkalemia. Known preexisting bicarb responsive acidosis (DKA, overdose of tricyclic antidepressant, ASA, cocaine or diphenhydramine). Prolonged resuscitation with effective ventilation; on return of spontaneous circulation after long arrest interval	Adequate ventilation and CPR, not bicarb, are the major "buffer agents" in cardiac arrest. Not recommended for routine use in cardiac arrest patients.	1 mEq/kg IV bolus Once ROSC, if rapidly available, use arterial blood gas analysis to guide bicarb therapy During cardiac arrest, ABG results are not reliable indicators of acidosis.
Vasopressin	Nonadrenergic peripheral vasoconstrictor, increasing blood flow to heart and brain. Vasopressor effects not blunted by severe acidosis	May be used as alternative pressor to 1st or 2nd dose of epinephrine in VF/pulseless VT, asystole or PEA cardiac arrest.	Potent peripheral vasoconstrictor.	Cardiac arrest: One dose of 40 units may replace first or second dose of epi
Verapamil	Slows depolarization of slow-channel electrical cells Slows conduction through AV node	Alternative drug after Adenosine in SVT To control ventricular rate in atrial fibrillation and atrial flutter.	Do not use for wide QRS tach of unknown origin, WPW, sick sinus syndrome or 2nd or 3rd degree heart block	5 mg IV over 2 min (over 3 min in older adults) May repeat 5 mg every 15 min as needed to total dose of 30mg

Figure 4
Circular ACLS Algorithm



CPR Quality
- Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
  - If P_{ETCO_2} <10 mm Hg, attempt to improve CPR quality
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Abrupt sustained increase in P_{ETCO_2} (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

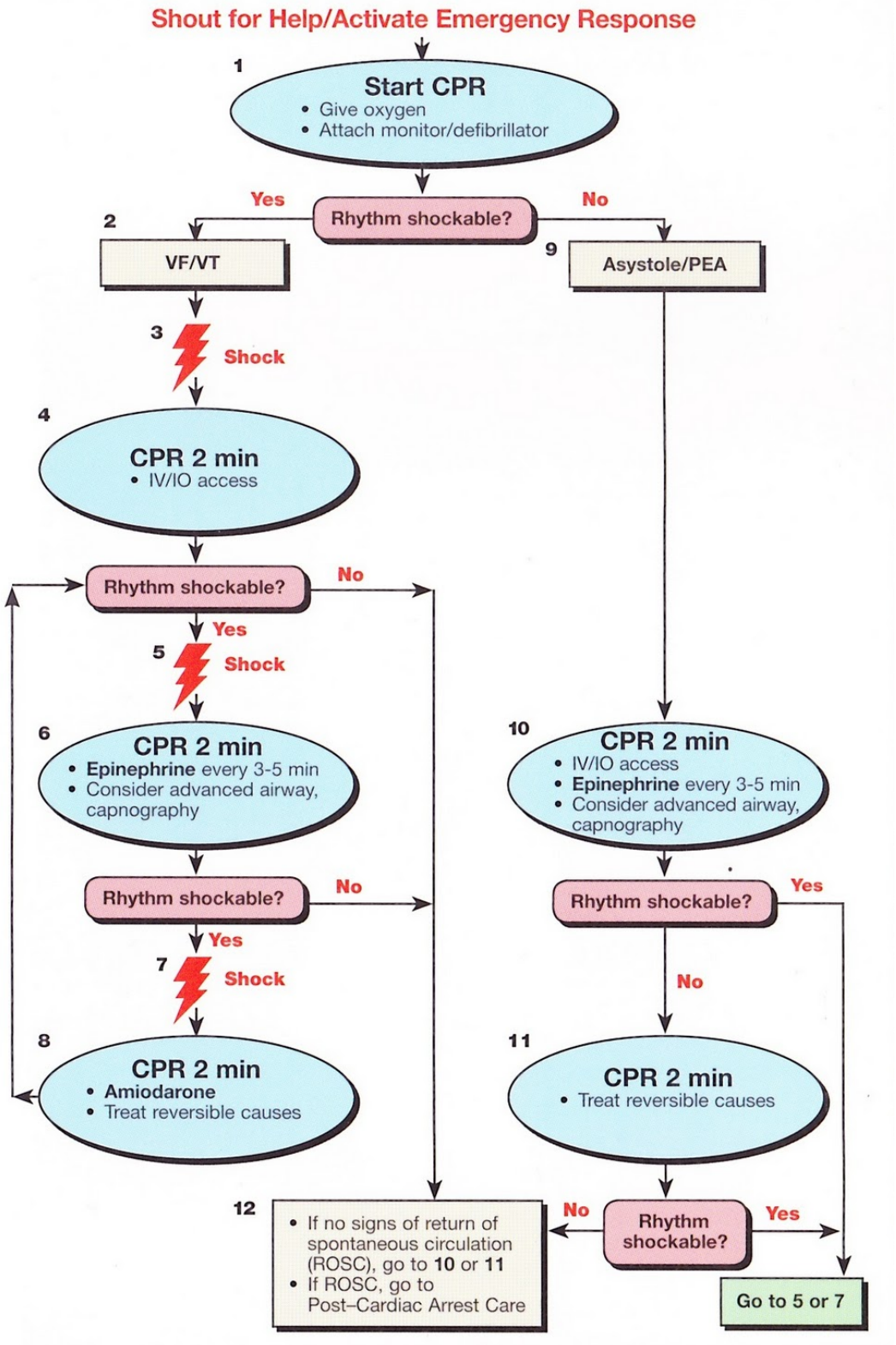
Shock Energy
- **Biphasic:** Manufacturer recommendation (120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy
- **Epinephrine IV/IO Dose:** 1 mg every 3-5 minutes
- **Vasopressin IV/IO Dose:** 40 units can replace first or second dose of epinephrine
- **Amiodarone IV/IO Dose:** First dose: 300 mg bolus. Second dose: 150 mg.

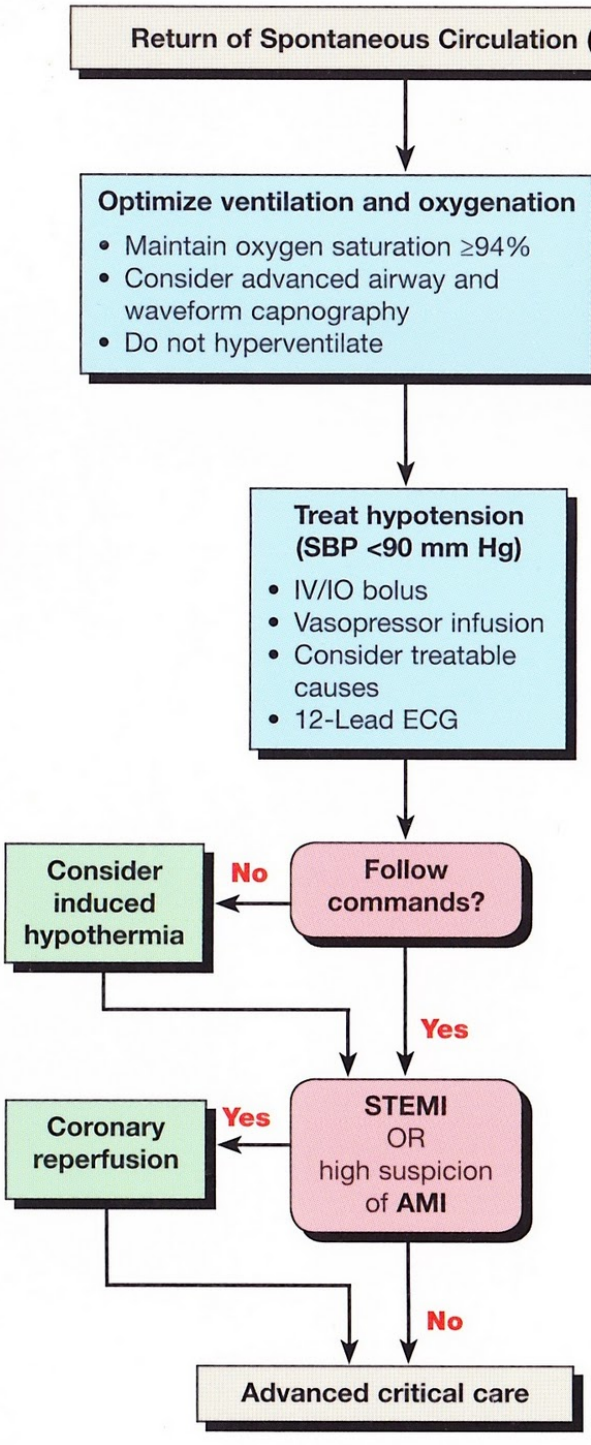
Advanced Airway
- Supraglottic advanced airway or endotracheal intubation
- Waveform capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

Reversible Causes
- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Cardiac Arrest Algorithm



Immediate Post-Cardiac Arrest Care Algorithm



Doses/Details

Ventilation/Oxygenation
Avoid excessive ventilation. Start at 10-12 breaths/min and titrate to target P_{ETCO_2} of 35-40 mm Hg.
When feasible, titrate F_{IO_2} to minimum necessary to achieve Spo_2 ≥94%.

IV Bolus
1-2 L normal saline or lactated Ringer's.
If inducing hypothermia, may use 4°C fluid.

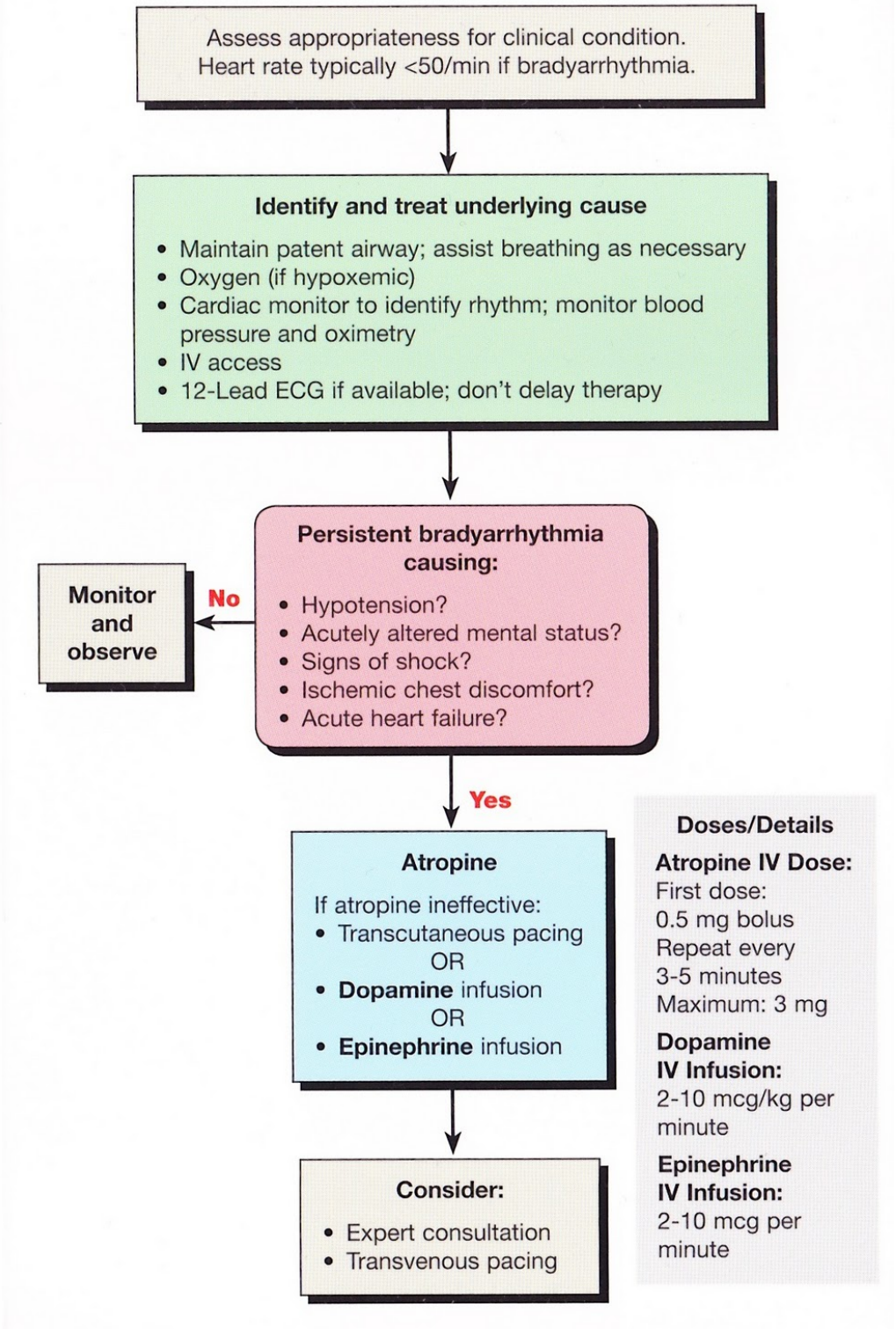
Epinephrine IV Infusion:
0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)

Dopamine IV Infusion:
5-10 mcg/kg per minute

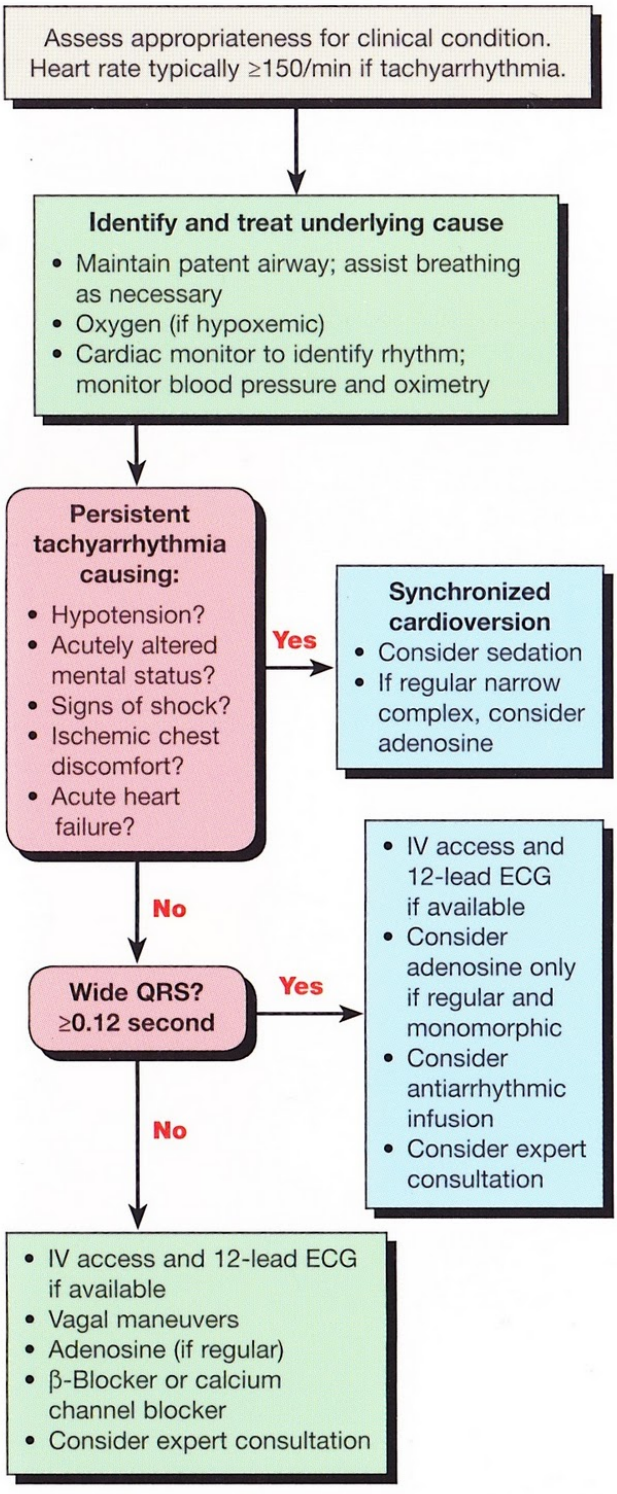
Norepinephrine IV Infusion:
0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)

Reversible Causes
- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Bradycardia With a Pulse Algorithm



Tachycardia With a Pulse Algorithm



Doses/Details

Synchronized Cardioversion

Initial recommended doses:

- Narrow regular: 50-100 J
- Narrow irregular: 120-200 J biphasic or 200 J monophasic
- Wide regular: 100 J
- Wide irregular: defibrillation dose (NOT synchronized)

Adenosine IV Dose:

First dose: 6 mg rapid IV push; follow with NS flush. Second dose: 12 mg if required.

Antiarrhythmic Infusions for Stable Wide-QRS Tachycardia

Procainamide IV Dose:

20-50 mg/min until arrhythmia suppressed, hypotension ensues, QRS duration increases >50%, or maximum dose 17 mg/kg given. Maintenance infusion: 1-4 mg/min. Avoid if prolonged QT or CHF.

Amiodarone IV Dose:

First dose: 150 mg over 10 minutes. Repeat as needed if VT recurs. Follow by maintenance infusion of 1 mg/min for first 6 hours.

Sotalol IV Dose:

100 mg (1.5 mg/kg) over 5 minutes. Avoid if prolonged QT.