



Banner
Staffing Services

ECG Study Guide (PALS)

Placement Assessment Testing is an assessment of your basic knowledge and should be completed as part of your new hire process. Please adhere to the following guidelines:

- The study guide is a good overall review of the components of the EKG waveform and more common Dysrhythmia. Although we are sure that you know the information—you need to study this information to be prepared to pass the test with an 85%.
- Use this study guide and your resource books to review what interventions you would choose for each rhythm strip. You will be expected to know your meds appropriate with interventions (Per ACLS guidelines). You will be expected to measure the PQRS on each strip. Practice measuring your strips in the study guide. **The rhythm strips on the exam are NOT 6 second strips.** Use calipers or the attached handout to determine rates.
- Memorize your blocks...there are several rhythm strips on blocks. You will need to describe unstable treatment for all.
- **Back to the Basics-** Don't forget your basics (check pulse, IV, O2, CPR, Assess patient). Although you are documenting treatment following the ACLS guidelines; it is imperative that you start with the basics.
- When assessing an unstable patient it is important to use your critical thinking skills.
- The 2010 ECC Pediatric algorithms for cardiac arrest, bradycardia with a pulse, and tachycardia with a pulse are included in this study guide. Refer to these algorithms for treatment and interventions. Changes have occurred with the 2010 guidelines, so please take time to review the following link:
http://static.heart.org/eccguidelines/pdf/90-1043_ECC_2010_Guidelines_Highlights_noRecycle.pdf
- Don't forget when to consider transcutaneous pacing (TCP) where appropriate.
- If it's slow---you will want to speed it up. If it is too fast---you will want to slow it down. Memorized which drugs you will do for each action.
- Treatment of lethal rhythms
 - Remember to assess your patient---Unstable treatment
 - Check Pulse
 - Start CPR
 - Transcutaneous External Pacemaker (TCP)

Questions, Give us a call.

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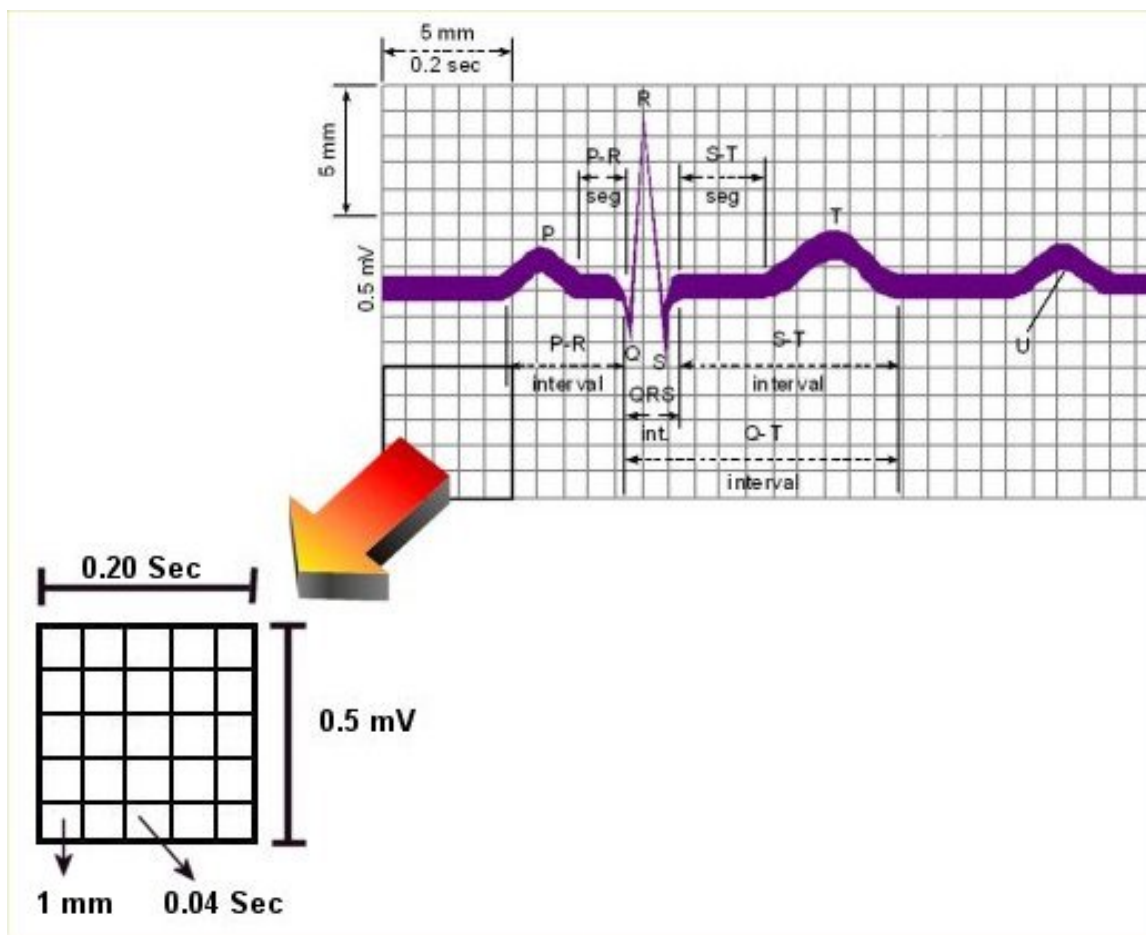
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EKG Graph Paper

The paper used to record EKG tracings is grid like in nature and has specific markings utilized to mark length in seconds. The paper is divided into large and small boxes. Thicker darker lines separate large boxes; thinner lighter lines separate small boxes.

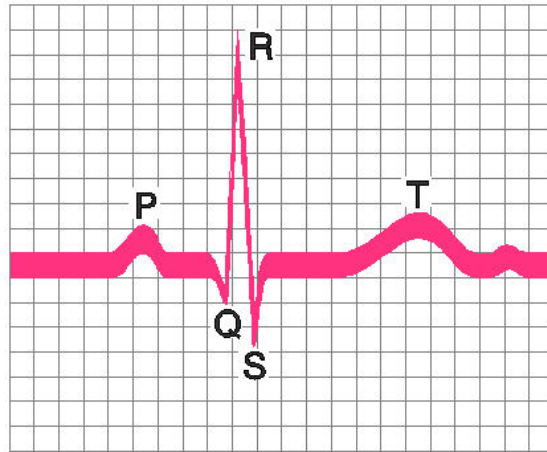
- The dark vertical lines are 0.20 seconds apart.
- The lighter vertical lines are 0.04 seconds apart
- Each small box is 1mm in size
- Each small box represents an electrical current that is equal to 0.1 (millivolt) mV
- One mV is equal to two large boxes





Normally EKG graph paper has thick dark lines at the top or bottom of the paper. In the strip presented here, the lines are at the bottom of the paper. The space between the lines is equal to 3 seconds. A standard EKG strip is run over 6 seconds. When printing an EKG rhythm strip the paper prints at a standard speed of 25 mm per second.

Components of the Normal EKG Waveform



The EKG is a real-time recording of the hearts electrical activity, produced by depolarization and repolarization of the hearts cells.

The EKG waveform consists of :

- **P Wave:** Represents atrial depolarization
- **QRS:** Represents ventricular depolarization
- **T Wave:** Represents ventricular repolarization

Intervals are described as the length of time between one waveform and the next.

Isoelectric Line: Flat line of the EKG tracing represents no electrical activity, and is referred to as the baseline. Deflections above the isoelectric line are positive, and deflections below the isoelectric line are negative.

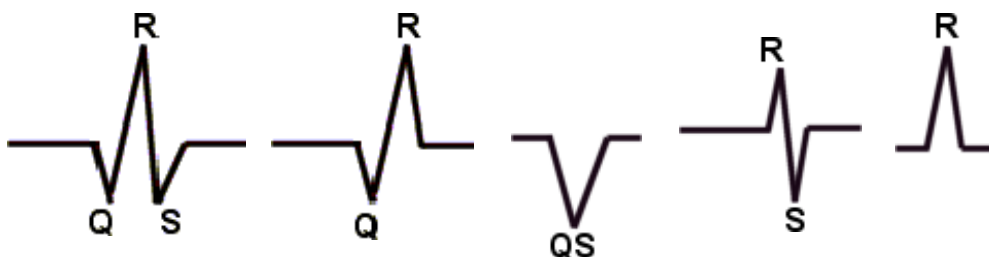
P-Wave: The SA node produces an electrical stimulus the P wave, normally upright, usually no more than 3mm in height.

PRI (PR Interval): Is representative of the spread of the atrial depolarization wave, and the time it takes for the impulse to conduct through the AV node and to the ventricles. The PRI is marked from the start of the P-Wave to the beginning of the QRS. The PRI is normally no more than 0.20 seconds in length.

QRS Complex: The QRS represents depolarization of the ventricles and ventricular conduction time of the electrical impulse. Typically the QRS is narrow with a conduction time of no more than 0.12 seconds. The QRS is measured from the beginning of the first waveform to the point at which the waveform returns to the isoelectric line. The QRS is made up of:

- **Q-Wave:** First negative deflection from baseline (below the isoelectric line),
- **R-Wave:** Positive deflection from the baseline (above the isoelectric line).
- **S-Wave:** Negative deflection following the R-Wave (below the isoelectric line)
- **R' (Prime)** is a secondary positive wave that may represent abnormal ventricular conduction.

Below are examples of the shapes that the QRS complex may take:



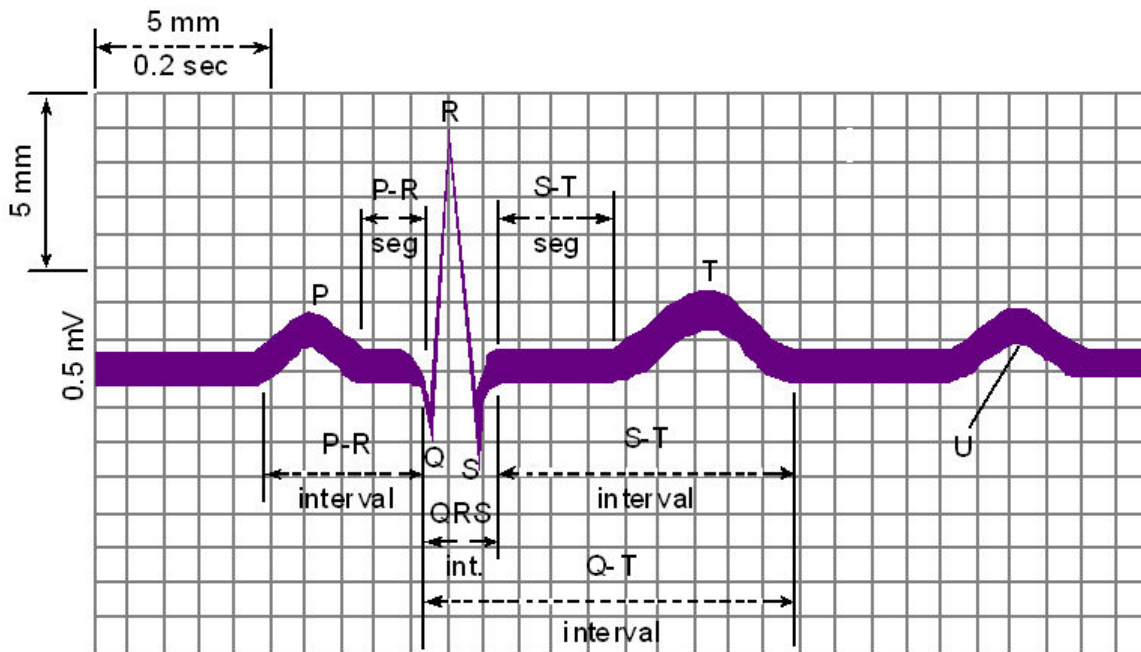
ST-Segment: Represents early ventricular repolarization, and extends from the end of the QRS to the beginning of the T-Wave, it is normally even with the isoelectric line. A deviation either above or below the isoelectric line represents myocardial injury or ischemia.

T-Wave: Represents ventricular repolarization as the ventricles return to a state of relaxation. The T-Wave is typically rounded and systematic. The T-Wave is typically upright however, this may vary if myocardial injury or ischemia is present.

QT-Interval (QTI): The QTI represents the refractory period of the ventricles, as they depolarize and repolarize. As rule of thumb, a normal QTI is less than 0.40 seconds. The QTI is directly related to the heart rate.

Summary of Heart Measurements:

- **P-Wave:** Upright in lead II
- **PRI:** < 0.20 Sec
- **QRS:** < 0.12 Sec
- **ST-Segment:** Even with isoelectric line
- **T-Wave:** Upright in lead II
- **QTI:** < 0.40 Sec



Basic EKG Rhythm Analysis Guide

When looking at a cardiac rhythm ask your self the following questions.

1. What is the rate? Is it fast or slow?

Rhythm	Rate
NSR	60-100
Sinus Brady	< 60
Sinus Tachycardia	100-150
SVT	>150
Junctional	40-60
Accelerated Junctional	60-100
Junctional Tachycardia	>100
Idioventricular Rhythm	20-40
Accelerated Idioventricular	40-100
Ventricular Tachycardia	>100

• For regular rhythms count the number of large boxes between R waves and divide into 300 or look up on chart. Use the same method to calculate the atrial rate by counting large boxes between P waves.

• For irregular heart rates count the number of R waves in a 6 second strip and multiply by 10. Do the same for P waves to calculate atrial rate. Some rhythms may have atrial rates that differ from the ventricular rate.

Calculating Heart Rates:

# Of Large Boxes	Rate
1	300
2	150
3	100
4	75
5	60
6	50
7	43
8	37
9	33
10	30

3. Measure the following: PRI, QRS, QT.

A. Is there a P wave for each and every QRS Complex?

Are the P waves upright and regular?

Do the P waves march out regularly?

B. Is the QRS narrow or wide? Grossly abnormal in appearance?

If the QRS is narrow .12 or less, the impulse will have originated above the ventricles.

If the QRS is very wide, .16 or greater with a T waves the opposite direction of the QRS complex. It would be suspected that the impulse is ventricular in origin.

4. Is the rhythm regular or irregular?

What rhythms are regular? Which rhythms are irregular?

5. Can you name the rhythm yet?

Putting It All Together

If there are consistently more P waves than QRS you might consider the following:

Second Degree AVB Type I and Type II

Complete Heart Block will have independent P wave rate and QRS rate. P waves are non conducted.

If the QRS complex is very wide $> .16$ and no P waves consider:

Idioventricular Rhythm

Accelerated Idioventricular Rhythm

Ventricular Tachycardia

If there are P waves for each and every QRS and the QRS complex is narrow $< .12$ consider:

Sinus Bradycardia

NSR

Sinus Tachycardia

If the QRS complex is narrow $< .12$ and the rate is > 150 consider:

SVT (P waves may or may not be visible due high rate)

If the P waves are absent or upside down with a QRS width $< .12$ and the rhythm is regular consider:

Junctional Rhythm

Accelerated Junctional Rhythm

Junctional Tachycardia

If the P waves are absent and the QRS is $< .12$ and the rhythm is irregular consider:

Atrial Flutter

Atrial Fibrillation

If the PRI is $> .20$ with a P wave for every QRS and the QRS is $< .12$ consider:

1st AVB

If there is no QRS present consider:

Are the leads attached to the patient?

Are the leads plugged into the monitor?

Confirm absence of QRS in another lead.

Does your patient have a pulse?

After checking all the above consider the following.

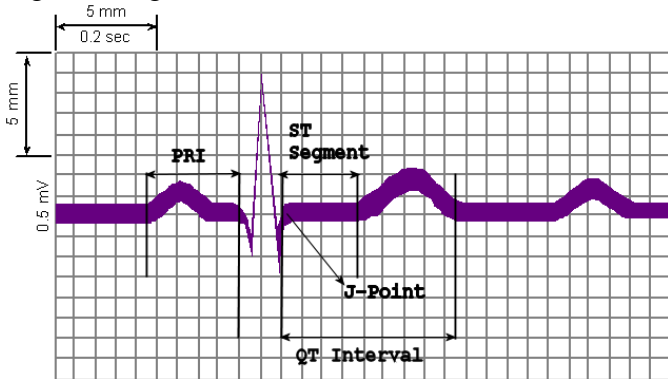
Asystole

Ventricular Fibrillation

ST Segment

Basic facts of the ST segment.

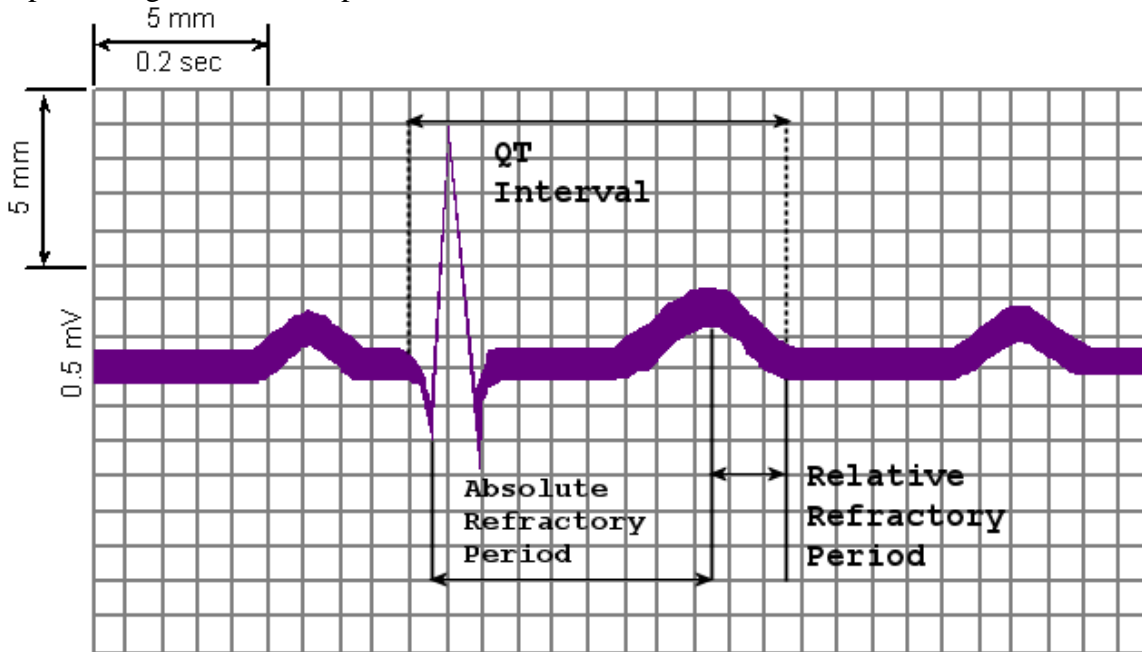
The ST segment starts at the end of the QRS and ends at the start of the T Wave. The ST segment represents the early beginning of ventricular repolarization. The portion of the EKG tracing where the QRS ends and the ST segment begins is called the *J Point*.



Characteristics: The ST-segment normally remains "*Isoelectric*" which is the normal baseline of the EKG. Elevation greater than 1mm in two or more reciprocal leads may indicate injury. Depression greater than 1mm in two or more reciprocal leads may indicate myocardial ischemia.

QT Interval

Definition: The QT interval represents total ventricular activity. It begins with the first wave in the *QRS complex* representing ventricular depolarization, and ends when the *T Wave* returns to baseline at the isoelectric line, representing ventricular repolarization.



Normal Measurements of the QTI: 0.36-0.40

It is best to measure the QTI in a lead in which the T wave is most pronounced. The normal overall length of the QTI should be equal to or less than $\frac{1}{2}$ of the R-R interval.

Normal Sinus Rhythm



Rate: 60-100 BMP

PRI: .12-.20 sec

QRS: < .12

QT: < .40

Rhythm: Regular

Source of pacer: SA Node

Sinus Bradycardia



Rate: < 60 BPM

PRI: .12-.20 sec

QRS: < .12

QT: < .40 May be prolonged with excessively low heart rates.

Rhythm: Regular

Source of pacer: SA Node

Characteristics: This rhythm may be normal for well-conditioned athletes and during sleep. Sinus Bradycardia, may be caused by several factors such as increased vagal tone from vomiting, bearing down to have a bowel movement or from medications such as digitalis, calcium channel blockers, beta blockers and many other antiarrhythmic medications Common with inferior wall MI, obstructive jaundice and increased intracranial pressure (ICP).

Treatment: Treatment is only necessary if the patient is symptomatic. Atropine 0.5 –1.0 mg, to a maximum of 3mg. Consider external transcutaneous pacing. Be prepared to assist the physician with the placement of an external temporary pacer. Treatment of associated hypotension may also need to be addressed.

Sinus Tachycardia



Rate: 100-150 BPM

PRI: .12-.20 sec

QRS: < .12

QT: < .40

Rhythm: Regular

Source of pacer: SA Node

Characteristics: Sinus tachycardia is a normal response to stress and exercise. If it is persistent, at rest it may indicate a more severe underlying problem such as fever, dehydration, blood loss, anemia, anxiety, heart failure, hypermetabolic states or ingestion of a significant stimulant such as cocaine or methamphetamine. Drugs that can cause Sinus Tachycardia are atropine, isuprel, epinephrine, dopamine, dobutrex, norepinephrine, nipride and caffeine. Sinus Tachycardia increases the hearts need for oxygen, decreases ventricular diastolic time and decreases coronary artery perfusion. Reflexive Sinus Tachycardia is often seen in hypotensive patients, in an attempt to maintain adequate blood pressure.

Treatment: The underlying cause must be identified and treated. Drugs that may be given to slow the heart are: digitalis, beta blockers, calcium channel blockers, sedatives and various other antiarrhythmic medications.

Supraventricular Tachycardia



(PSVT/PSAT)



(SVT)

Rate: > 150 BPM

PRI: Usually < .12 sec

QRS: Narrow, < .12 sec

QT: < .40 sec

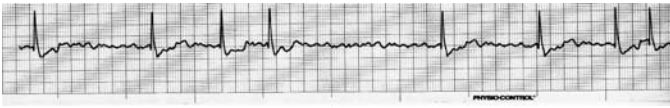
Rhythm: Regular

Source of pacer: Atrial re-entry current

Characteristics: Tachycardias with a narrow QRS < .12 seconds and faster than 150 BPM do not originate from the SA Node, since the upper limit rate for the SA NODE is 150. The rhythms can vary in name from Supraventricular Tachycardia (SVT), Atrial Tachycardia, or a rhythm that may speed up and slow down called Paroxysmal Atrial Tachycardia or Paroxysmal Supraventricular Tachycardia. If P waves are visible, the rhythm may be called Atrial Tachycardia, if no P waves are visible due to a very fast rate, label the rhythm SVT.

The onset of PSVT/PAT is typically abrupt, with the onset being initiated by a premature atrial beat. The arrhythmia may self terminate in a few minutes to a few hours.

Atrial Fibrillation



Rate: Variable

PRI: None

QRS: < .12 sec

QT: < .40

Rhythm: Irregular

Source of pacer: Atrial

Characteristics: Atrial fibrillation is caused by chaotic ectopic or reentry current activity, which causes the atria to quiver rather than contract. The atria quiver at a high rate producing the fuzzy and garbled wave forms seen where a flat isoelectric line should be. Atrial fibrillation can generate a ventricular response rate that is controlled or very fast and can place the patient at risk for hemodynamic instability.

Cardiac output is reduced with the loss of “Atrial Kick” since the atria are not contracting. The ventricular rate may also be very fast resulting in further decreased cardiac output. In addition, since the atria are not contracting, the patient is at risk for the formation, of emboli leading to pulmonary embolism or stroke.

Causes: MI, Rheumatic heart disease, COPD, CHF, ischemia chest trauma, CAD and open-heart surgery.

Treatment: Treatment may vary. If this is an acute arrhythmia, the patient may be cardioverted, or treated with beta-blockers, calcium channel blockers, digoxin, amiodarone or procainamide. If this is a chronic rhythm that would not convert with cardioversion or medications, it is important that the patient be evaluated and possibly placed on anticoagulation medication before discharge home.

Junctional Rhythm



Rate: 40-60 BPM

PRI: Absent or variable

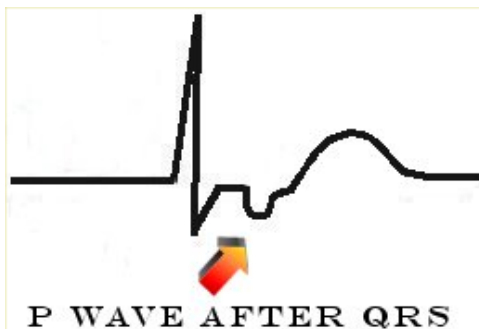
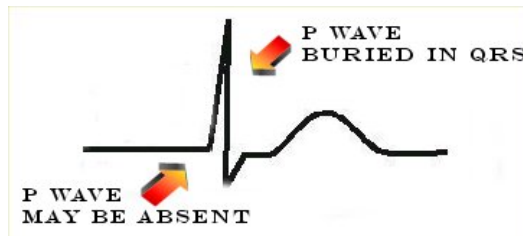
QRS: Usually $< .12$ may be wider depending on pacer site.

QT: $< .40$ may be prolonged with slower heart rates

Rhythm: Regular

Source of pacer: AV Node/Junction/Bundle of HIS

Characteristics: Rhythms that originate from the AV Junction: Cells around the AV Node have automaticity and are capable of becoming the primary pacemaker if the SA Node should fail. Junctional escape beats occur when the SA Node fails to initiate an impulse or its rate falls below the rate of the AV Node. Junctional rhythms can accelerate above the rate of SA Node, and become the primary pacemaker and assume control of the heart's rhythm and rate. Atrial kick may be lost resulting in decreased cardiac output.



The P wave will always be inverted, and may appear before or after the QRS or be completely absent.

Causes: Electrolyte imbalance, sick sinus syndrome, digitalis toxicity, inferior-wall MI, rheumatic heart disease, hypoxemia.

Treatment: Find and treat reversible causes, temporary or permanent pacer, atropine 0.5 – 1.0mg may cause the SA Node to overdrive the AV Node and increase the heart rate.

Treatment is only needed if the patient is hypotensive or presents with hemodynamic instability.

1st Degree AV Block



Rate: Variable

PRI: > .20 sec

QRS: < .12

QT: < .40 May be longer with slower heart rates.

Rhythm: Regular

Source of pacer: SA Node

Characteristics: 1st degree AV block, is characterized by what appears to be a normal sinus rhythm. Closer examination of the PRI will show that it is longer than .20 seconds. The PRI should not vary from one beat to the next. 1st degree AV block represents a delay in the conduction of electrical impulses through the AV Node, which under normal circumstances would only delay the impulse .10 seconds or less. 1st degree AV block may progress into higher degrees of block.

Causes: Acute inferior MI, right ventricular infarction, increased vagal tone, ischemic heart disease, digitalis toxicity, beta-blockers, amiodarone, calcium channel blockers, electrolyte imbalances, rheumatic heart disease or myocarditis.

Treatment: Find and treat causative agent, observe for progression to higher blocks. Be prepared to pace, if the patient is bradycardiac and symptomatic.

2nd Degree AV Block (Mobitz Type I, Wenkebach)



Rate: Variable

PRI: Progressive elongation of PRI until a P wave is not conducted.

QRS: < .12

QT: < .40 May be longer with slower heart rates.

Rhythm: Irregular due to dropped P waves.

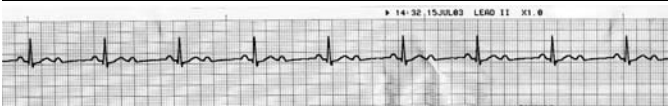
Source of pacemaker: SA Node

Characteristics: This is a progressive slowing in the conduction from the atria to the ventricles until a beat is dropped. 2nd degree AV block (Wenkebach) appears on the rhythm strip as a progressively longer PRI on conducted beats until the impulse is not conducted through the AV node. This is denoted by a P wave (non-conducted) that is not followed by a QRS complex. The rhythm is repetitive in nature. 2nd degree AV block type I can be transient and reversible. However, it can progress to a higher degree of AV block.

Causes: Acute inferior MI, right ventricular infarction, increased vagal tone, ischemic heart disease, digitalis toxicity, beta-blockers, amiodarone, calcium channel blockers, electrolyte imbalances, rheumatic heart disease or myocarditis.

Treatment: Most of the time this rhythm produces no signs and symptoms and requires no treatment. If needed 2nd degree Type I AVB will respond to atropine if the patient becomes bradycardic and hypotensive. Temporary pacing should also be considered. Find and treat reversible causes, and observe for progression into higher forms of block.

2nd Degree AV Block (Mobitz Type II)



Rate: Variable

PRI: .12-20 when conducted

QRS: > .12 may be longer depending on where the location of the block and if a bundle branch block is present.

QT: < .40 May be longer with slower heart rates.

Rhythm: Irregular due to blocked beats.

Source of pacemaker: SA Node

Characteristics: 2nd Degree Type II AVB is a sudden failure of the conduction of a SA Node impulse without a progressive elongation of the PRI segment of conducted P waves. This type of block is caused by the complete block of the impulse in bundle branch and an intermittent block in the other bundle branch. This rhythm is characterized by more P waves than QRS complexes and a normal PRI when an impulse is conducted. The QRS complex is typically abnormal (wider than .12 seconds) due to the bundle branch block. The block may be in a ratio of 2:1 (two P waves for every QRS), 4:3, 3:2.

Causes: Can be caused by damage to the bundle branch system following an acute anterior AMI. This is not caused by medications or increased vagal tone

Treatment: This rhythm should be considered more serious than 2nd degree type I AVB. Type II can progress into complete heart block, or even ventricular asystole. A temporary pacemaker may have to be used if the patient becomes bradycardic and hypotensive. Atropine is not recommended.

3rd Degree AB Block / Complete Heart Block



Rate: Depends on site of pacer.

PRI: No relationship between P waves and QRS complex.

QRS: Normal if junctional rhythm, wider if ventricular rhythm.

QT: May be < .40 seconds if it is a junctional rhythm or may be wider (> .40 sec) with slower ventricular rhythms.

Rhythm: Usually regular

Source of pacer: Variable. Will be below the atria, since the connection between the SA Node and AV Node has been severed. Usually Junctional or Ventricular in origin.

Characteristics: The connection between the atria and the ventricles has been severed. The resulting rhythm will either be junctional or ventricular in origin. It is important to march the P waves and QRS complexes out to establish that there is truly no relationship between the two. Since there is no connection between the atria and ventricles, it is possible to have an atrial rhythm such as atrial fibrillation or flutter, and a junctional or idioventricular rhythm driving the ventricles.

This is illustrated in the EKG strip below.



Causes: 3rd degree AVB may be transient and reversible or permanent. Common causes are: acute inferior or right ventricle MI, ischemic heart disease in general, increased vagal tone, digitalis toxicity, amiodarone, beta-blockers, calcium channel blockers, electrolyte imbalances.

Treatment: Signs and symptoms are similar to that of symptomatic sinus bradycardia. This rhythm can progress to ventricular asystole if no back up pacemaker takes over. Pacing is the treatment of choice at first a temporary pacer may be deployed until the patient can have a permanent pacer implanted.

Ventricular Tachycardia



Rate: > 100 BPM

PRI: P waves may be present if SA node is functional, however there is no relation to the QRS. It is unlikely that P waves will be visible, since this rhythm typically moves at a very fast rate, and the P waves will be buried in the QRS.

QRS: Wide/Bizarre > .12

QT: Rate dependant may be < .40 seconds with a fast rhythm.

Rhythm: Regular

Source of pacer: Ventricular/Purkinje Fibers

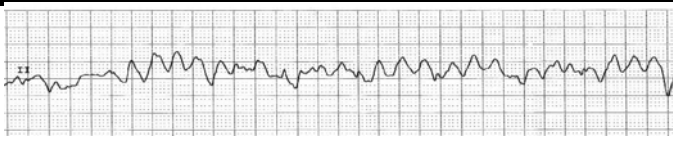
Characteristics: VT is a rhythm of ventricular origin with a rate faster than 100 beats per minute. VT can appear as polymorphic (different QRS complexes) or monomorphic (QRS complexes are all the same). VT results from an ectopic focus or a ventricular reentry pathway.

Depending on the rate of the tachycardia, the patient can present as stable or unstable. If VT occurs at a relatively slow rate in a healthy heart, the rhythm can be well tolerated. However, if the rhythm is fast, ventricular diastolic filling time is limited resulting in poor cardiac output and hemodynamic instability. Patients with poor left ventricular function and ejection fraction do not tolerate any form of tachycardia. The longer that a patient is left in sustained VT, the harder it may be to convert to a regular sinus rhythm.

Causes: Ventricular tachycardia may be caused by: R on T PVC phenomenon, hypoxia, ischemia, AMI, acidosis, cardiomyopathy, mitral valve prolapse, digitalis toxicity, antiarrhythmics, electrolyte imbalances, liquid protein diets, increased intracranial pressure and central nervous system disorders.

Treatment: Stable patients are given medications to attempt to chemically convert them. Unstable patients are to be treated promptly with defibrillation and medications. Please consult your ACLS manual for detailed treatment algorithms.

Ventricular Fibrillation



Rate: Unable to measure due to rapid and uncoordinated electrical activity.

PRI: 0

QRS: 0

QT: 0

Rhythm: Rapid and chaotic.

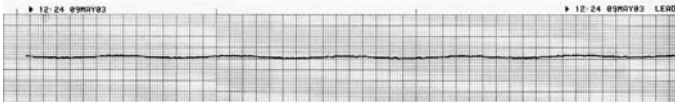
Source of pacer: Multiple sources of ectopy in the Ventricular/Purkinje Fibers

Characteristics: VF is chaotic electrical activity in the ventricles that results in quivering of the ventricles and total loss of cardiac output. VF is fatal unless treated promptly with defibrillation. There are no QRS complexes or P waves present. VF can be coarse or very fine and look almost like asystole.

Causes: CAD, AMI, trauma, hypoxia, acidosis, antiarrhythmics, electrolyte imbalances, cardiac catheterization, cardioversion, accidental electrocution, cardiac pacing and extreme hypothermia.

Treatment: CPR, immediate treatment with antiarrhythmic medications and defibrillation with appropriate joule settings. Refer to your ACLS manual for detailed treatment algorithms.

Asystole



Rate: 0

PRI: 0

QRS: 0

QT: 0

Rhythm: None

Source of pacer: None

Characteristics: Asystole is the sudden loss of ventricular electrical activity, resulting in no ventricular contractions and no cardiac output. Asystole is fatal unless reversed immediately. Asystole should always be confirmed in another monitor lead before treatment. A true asystole will not respond to defibrillation.

Causes: End stage cardiac disease, ischemia, MI, severe electrolyte imbalances, acidosis, and hypoxia.

Treatment: CPR, epinephrine, atropine and external pacing. Refer to ACLS manual for detailed treatment algorithms.

Torsade de Pointes



Rate: 100-250 BPM

PRI: P waves may be present if SA node is functional, however there is no relation to the QRS. It is unlikely that P waves will be visible, since this rhythm typically moves at a very fast rate, and the P waves will be buried in the QRS.

QRS: Wide/Bizarre $> .12$

QT: Usually, $< .40$ seconds due to fast heart rate.

Rhythm: Usually regular

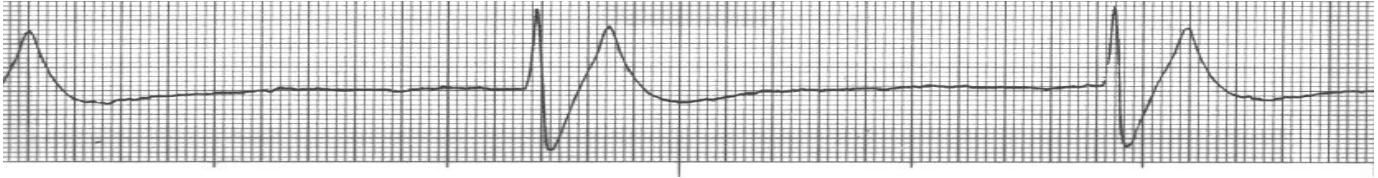
Source of pacer: Ventricular/Purkinje Fibers

Characteristics: Torsade de Pointes is essentially a polymorphic VT. It is characterized by a widening and narrowing of the QRS amplitude. The arrhythmia may be paroxysmal, which starts and stops suddenly and may suddenly deteriorate into VF.

Causes: The cause of Torsades may be reversible. The most common causes are drugs that lengthen the QT interval such as antiarrhythmics (quinidine, procainamide and sotalol). Other causes include myocardial ischemia, and hypokalemia, hypomagnesemia and hypocalcemia.

Treatment: Find and treat reversible causes. Overdrive pacing with the use of an external pacer or Isuprel may overdrive the ventricular rate and break the triggering mechanism of the arrhythmia. Magnesium sulfate may also be effective. Refer to your ACLS manual for detailed treatment algorithms.

Idioventricular (IVR)



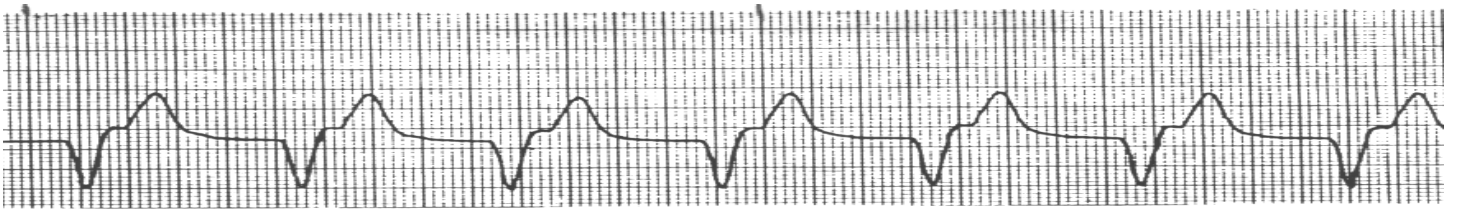
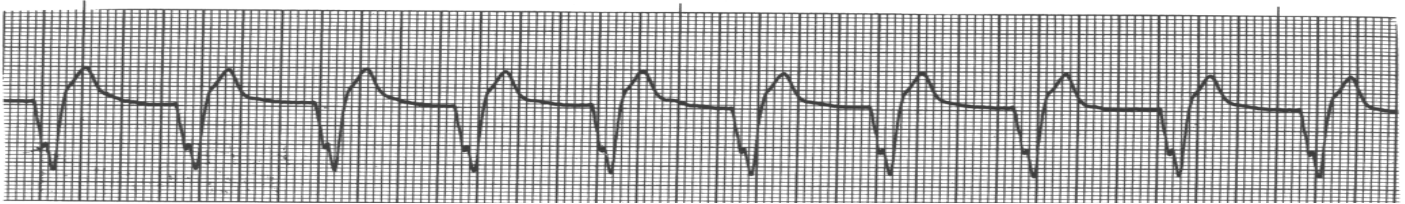
Rate—20-40bpm

PRI—absent since the electrical activity is coming from the ventricles

QRS—Greater than 0.12seconds (Wide and bizarre)

Treatment: Assess patient; check pulse; External Transcutaneous Pacemaker (TCP); treat cause

Accelerated Idioventricular (AIVR)



Rate—40-100bpm

Rhythm—Irregular

QRS—Wide—If the QRS is wider than 0.16 seconds and with a T-wave the opposite direction of the QRS complex; the impulse is most generally ventricular

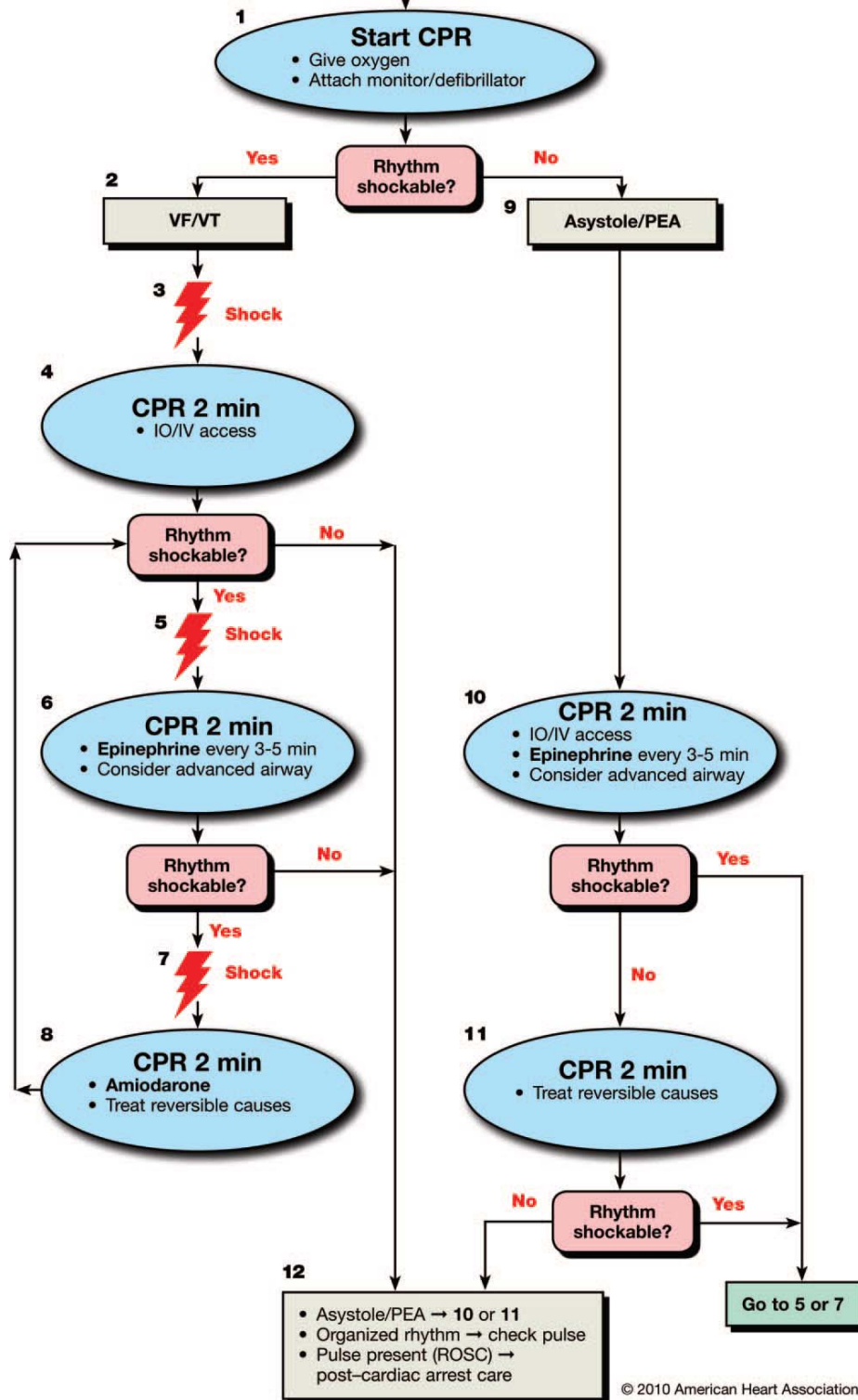
PRI—P waves absent or retrograde conduction

Treatment: Assess patient; usually no treatment necessary; treat cause (drug overdose; heart disease).

PALS Algorithms

Pediatric Cardiac Arrest

Shout for Help/Activate Emergency Response



Doses/Details

CPR Quality

- Push hard ($\geq 1/3$ of anterior-posterior diameter of chest) and fast (at least 100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 15:2 compression-ventilation ratio. If advanced airway, 8-10 breaths per minute with continuous chest compressions

Shock Energy for Defibrillation

First shock 2 J/kg, second shock 4 J/kg, subsequent shocks ≥ 4 J/kg, maximum 10 J/kg or adult dose.

Drug Therapy

- **Epinephrine IO/IV Dose:** 0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration). Repeat every 3-5 minutes. If no IO/IV access, may give endotracheal dose: 0.1 mg/kg (0.1 mL/kg of 1:1000 concentration).
- **Amiodarone IO/IV Dose:** 5 mg/kg bolus during cardiac arrest. May repeat up to 2 times for refractory VF/pulseless VT.

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place give 1 breath every 6-8 seconds (8-10 breaths per minute)

Return of Spontaneous Circulation (ROSC)

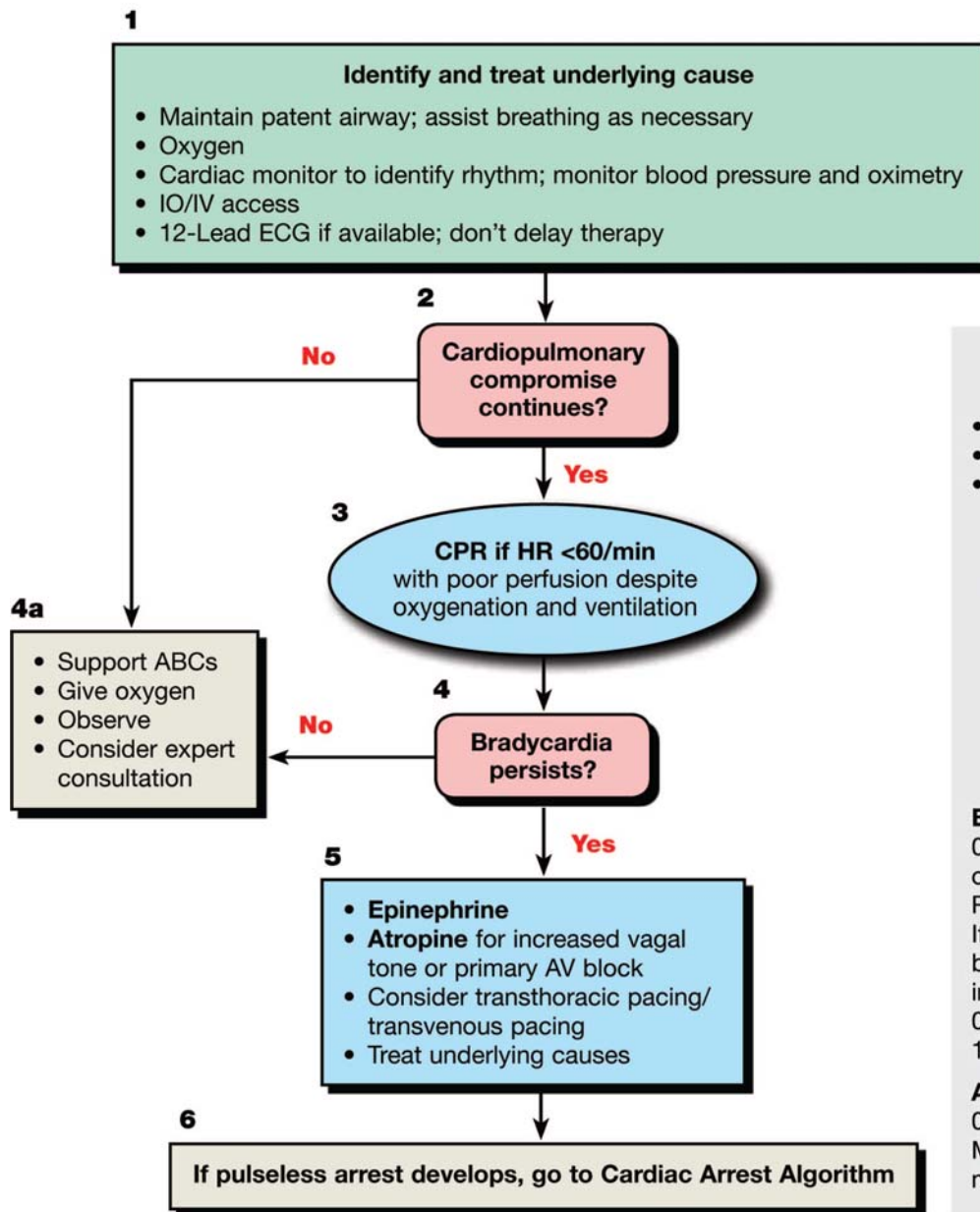
- Pulse and blood pressure
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypoglycemia
- Hypo-/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

Pediatric Bradycardia

With a Pulse and Poor Perfusion



Cardiopulmonary Compromise

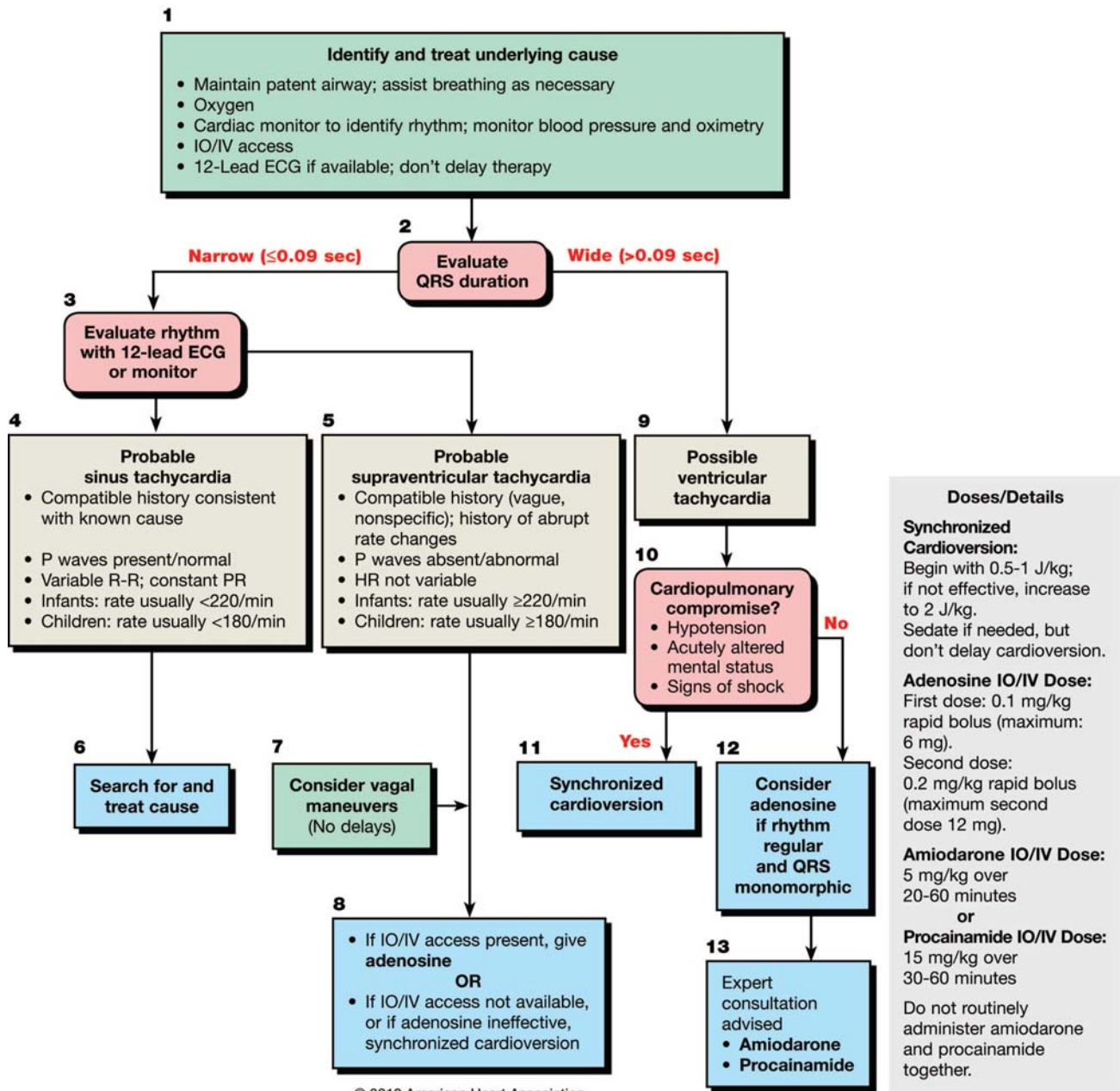
- Hypotension
- Acutely altered mental status
- Signs of shock

Doses/Details

Epinephrine IO/IV Dose:
0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration). Repeat every 3-5 minutes. If IO/IV access not available but endotracheal (ET) tube in place, may give ET dose: 0.1 mg/kg (0.1 mL/kg of 1:1000).

Atropine IO/IV Dose:
0.02 mg/kg. May repeat once. Minimum dose 0.1 mg and maximum single dose 0.5 mg.

Pediatric Tachycardia With a Pulse and Poor Perfusion



Doses/Details

Synchronized Cardioversion:
Begin with 0.5-1 J/kg; if not effective, increase to 2 J/kg. Sedate if needed, but don't delay cardioversion.

Adenosine IO/IV Dose:
First dose: 0.1 mg/kg rapid bolus (maximum: 6 mg).
Second dose: 0.2 mg/kg rapid bolus (maximum second dose 12 mg).

Amiodarone IO/IV Dose:
5 mg/kg over 20-60 minutes
or
Procainamide IO/IV Dose:
15 mg/kg over 30-60 minutes

Do not routinely administer amiodarone and procainamide together.