ACLS *Study Guide*

2015

Bulletin: **New resuscitation science and American Heart Association treatment guidelines were released October 28, 2015!**

The new AHA Handbook of Emergency Cardiac Care (ECC) contains these 2015 Guidelines and is required study for this course. The 2015 ACLS Provider Manual is not yet available. This study guide will provide you with additional study information.

Website: www.heart.org/eccstudent Password: ACLS15 (Pretest, Videos and ACLS Supplemental Information)
www.phsinstitute.com (study info. For class for rhythm review and ACLS Supplemental Information)

What is required to successfully complete ACLS?

For ACLS RENEWALS ONLY: You must successfully score 84% on a ECG rhythm test. This includes naming the rhythm and two causes and two treatments. This information can be found in the ACLS Manual and Supplemental Information.

- ♥ **Completed ACLS Pre-test is required for admission** to the course.
- ♥ **Score 84% on the multiple-choice post-test**.
  It is a timed test and you may be allowed to **use your ECC Handbook**.
- ♥ **You must be able to demonstrate**:
  - the ACLS rapid cardiopulmonary assessment
  - using an AED
  - safe defibrillation with a manual defibrillator
  - maintaining an open airway
  - confirmation of effective ventilation
  - addressing vascular access
  - stating rhythm appropriate drugs, route and dose
  - consideration of treatable causes

What happens if I do not do well in the course?

The Course Director or Instructor will first "remediate" **(tutor)** you and you may be allowed to continue in the course. If it is decided you need more time to study, you will be placed into the next course.

Where do I start?

- **CPR/AED: You will be tested with no coaching. If you cannot perform these skills well without coaching, you can/may be directed to take the course at another time. Know p. 7-11 of this study guide well.**

- **Arrhythmias: Before you come be sure you can identify:** Sinus Rhythm **(SR)**, Sinus Bradycardia **(SB)**, **Sinus Tachycardia (ST), Supraventricular Tachycardia (SVT), Ventricular Tachycardia (VT), Ventricular Fibrillation (VF), Torsades de Pointes, Pulseless Electrical Activity (PEA) and Asystole, Atrall Fibrillation, Atrail Flutter, Junctional rhythm, 1st degree Atrial Ventricular Block(1st Degree AVB), 2nd Degree AVB type I (Mobitz I or Wenckebach)/ 2nd Degree AVB, 2nd degree Type II AVB (Mobitz II) , 3rd Degree Heart Block and more**

You will need to know:

Treat Possible Causes

.

5 Hs	5 Ts
Hypo xia	**T** amponade
Hypo volemia	**T** ension pneumothorax
Hyper-thermia	**T** oxins – poisons, drugs
Hypo /hyper kalemia	**T** hrombosis – coronary **(AMI)**
Hydro gen ion **(acidosis)**	**T** hrombosis – pulmonary **(PE)**

Spacing separations may help as a memory aid.

Rapid Cardiopulmonary Assessment *and* *A*lgorithms

This is a systematic head-to-toe assessment used to identify in respiratory distress and failure, shock and pulseless arrest. **Algorithms** are "menus" that guide you through recommended treatment interventions.

Know the following assessment because it begins all ACLS case scenarios. The information you gather during the assessment will determine which algorithm you choose for the patient's treatment. **After each intervention** you will reassess the patient again using the head-to-toe assessment.

<***Start with general appearance:***

Is the level of consciousness: **A**= awake **V**= responds to verbal **P**= responds to pain **U**= unresponsive

<***Then assess CABs:*** **(stop and give immediate support when needed, then continue with assessment)**

Circulation: **Is central pulse** present or absent?
Is the rate normal or too slow or too fast?
Is the rhythm regular or irregular?
Is the QRS narrow or wide?

Airway: **Check Airway if patient can maintain / if not Open and hold** with head tilt-chin lift

Breathing: **Is it** present or absent?
Is the rate normal or too slow or too fast?
Is the pattern regular or irregular or gasping?
Is the depth normal or shallow or deep?
Is it Noisy
Is there stridor or wheezing?

<*Next look at perfusion*:

Is the central pulse versus peripheral pulse strength equal or unequal?

<*And check*:

BP acceptable or hypotensive?

<*Now classify the physiologic status*:

Stable: needs little support; **reassess frequently**
Unstable: needs **immediate support** and intervention

.

<*Apply the appropriate treatment algorithm*:

- Bradycardia with a Pulse
- Tachycardia with Adequate Perfusion
- Tachycardia with Poor Perfusion
- Pulseless Arrest: VF/VT and Asystole/PEA

Advanced Airway

A **cuffed E**ndotracheal **T**ube **(ET)**.

Immediately confirm tube placement **by clinical assessment and a device**:

▶ **Clinical assessment:**
- Look for bilateral chest rise.
- Listen for breath sounds over stomach and the 4 lung fields **(left and right anterior and midaxillary)**.
- Look for water vapor in the tube **(if seen this is helpful but not definitive)**.

▶ **Devices:**
- **End-Tidal CO_2 Detector** (**ETD**):
  - ƒ Attaches between the ET and Ambu bag; give 6 breaths with the Ambu bag:
    - Litmus paper center should change color with **each inhalation** and **each exhalation**.
    - **Original color** on inhalation = **Okay** **O_2 is being inhaled:** expected.
    - **Color change** on exhalation = **CO_2!!** **Tube is in** trachea.
    - **Original color on exhalation** = **Oh-OH!!** **Litmus paper is wet**: replace ETD.
      Tube is not in trachea: remove ET.
      Cardiac output is low during CPR.
- **Esophageal Detector** (**EDD**):
  Resembles a turkey baster:
  - Compress the bulb and attach to end of ET.
  - Bulb **inflates quickly!** Tube is in the trachea.

- Bulb **inflates poorly?** Tube is **in the esophagus**.

ƒ No recommendation for its use in cardiac arrest.

► **When sudden deterioration of an intubated patient occurs, immediately check:**

Displaced	= tube is not in trachea	or has moved into a bronchus **(right mainstem most common)**	
Obstruction	= consider secretions	or kinking of the tube	
Pneumothorax	= consider chest trauma	or barotraumas	or non-compliant lung disease
Equipment	= check oxygen source	and Ambu bag	and ventilator

Supraventricular Tachyarrhythmia The recommended initial biphasic energy dose for cardioversion of atrial fibrillation is 120 to 200 J. The initial monophasic dose for cardioversion of atrial fibrillation is 200 J.

2015 (New) There is inadequate evidence to support the routine use of lidocaine after cardiac arrest. However, the initiation or continuation of lidocaine may be considered immediately after ROSC from cardiac arrest due to VF/pVT.

While earlier studies showed an association between giving lidocaine after myocardial infarction and increased mortality, a recent study of lidocaine in cardiac arrest survivors showed a decrease in the incidence of recurrent VF/pVT but did not show either long-term benefit or harm.

For ease of placement and education, the anterior-lateral pad position is a reasonable default electrode placement. Anyof 3 alternative pad positions (anterior-posterior, anterior-left infrascapular, and anterior–right infrascapular) may beconsidered on the basis of individual patient characteristics.

Placement of AED electrode pads on the victim' s bare chest inany of the 4 pad positions is reasonable for defibrillation.

2015 Continuous quantitative waveform capnography is now recommended for intubated patients throughout the periarrest period. When quantitative waveform capnography is used for adults, applications now include recommendations for confirming tracheal tube placement and for monitoring CPR quality and detecting ROSC based on end-tidal carbon dioxide

Capnography to monitor effectiveness of resuscitation efforts. PETCO2 should read 35 to 40mm Hh in individual of ROSC, High Quality CPR is confirmed by a Capnography read of >10mm Hg on the vertical axis over time. This patient is intubated and receiving CPR. Note that the ventilation rate is approximately 8 to 10 breaths per minute. Chest compressions are given continuously at a rate of slightly faster than 100/min but are not visible with this tracing.

ACLS Drugs

In Arrest:

Epinephrine: catecholamine ECC Handbook
Increases heart rate, peripheral vascular resistance and cardiac output; **during CPR** increases myocardial and cerebral blood flow.

IV/IO: 1 mg of 1:10 000 solution (10ml of 1:10 000) repeat q. 3–5 min
IV Infusion 2 to 10 mcg /minute
IV Infusion 0.1 to 0.5 mcg/ kg/minute (ROSC)

Antiarrhythmics:

Amiodarone: atrial and ventricular antiarrhythmic ECC Handbook
Slows AV nodal and ventricular conduction, increases the QT interval and may cause vasodilation.

VF/PVT:	IV/IO: 300 mg bolus
Perfusing VT:	IV/IO: 150 mg over 10 min
IV Infusion:	IV/IO: 1 mg/min first 6 hours
Max:	450 mg
Caution:	hypotension, Torsade; half-life is up to 40 days

Lidocaine: ventricular antiarrhythmic to consider when amiodarone is unavailable ECC Handbook
Decreases ventricular automaticity, conduction and repolarization.

VF/PVT:	IV/IO: 1 – 1.5 mg/kg bolus first dose, then 0.5 to 0.75 mg/kg, maximum 3 doses or 3mg/kg
Perfusing VT:	IV/IO: 1 – 1.5 mg/kg bolus
Infusion:	20-50 mcg/kg/min
Caution:	neuro toxicity → seizures

Magnesium: ventricular antiarrhythmic for Torsade and hypomagnesemia ECC Handbook
Shortens ventricular depolarization and repolarization **(decreases the QT interval)**.

IV/IO:	1 - 2 g
Max:	2 gm
Caution:	hypotension, bradycardia

Increase heart rate:

Atropine: vagolytic to consider after oxygen, ventilation and Fuild Bolus ECC Handbook
Blocks vagal input therefore increases SA node activity and improves AV conduction.

IV/IO:	0.5 mg; may double amount for second dose 1mg for AV Block (First Degree, Second Degree Type I)
Max:	3 mg
Caution:	**do not give less than 0.1 mg** or may worsen the bradycardia

Atropine is not recommended for routine use in the management of PEA/asystole and has been removed from the ACLS Cardiac Arrest Algorithm. The treatment of PEA/ asystole is now consistent in the ACLS

Decrease heart rate:

Adenosine: drug of choice for symptomatic SVT & Wide Complex Monomorphic VT See ECC Handbook

Blocks AV node conduction for a few seconds to interrupt AV node re-entry.

IV/IO: first dose: max: 6 mg
second dose: max: 12 mg
Third dose: max: 12 mg

Adenosine is recommended *in the initial diagnosis and treatment of stable, undifferentiated regular*, **monomorphic wide-complex tachycardia**

Increase blood pressure:

Dobutamine: synthetic catecholamine ECC Handbook

Increases force of contraction and heart rate; causes mild peripheral dilation; may be used to treat shock.

IV/IO infusion: 2- 20 mcg/kg/min infusion
Caution: tachycardia

Dopamine: catecholamine ECC Handbook

May be used to treat shock; effects are dose dependent.

Low dose: increases force of contraction and cardiac output.
Moderate: increases peripheral vascular resistance, BP and cardiac output.
High dose: higher increase in peripheral vascular resistance, BP, cardiac work and oxygen demand.
IV/IO infusion: 2–20 mcg/kg/min
Caution: tachycardia
IV/IO infusion: 5–10 mcg/kg/min (ROSC)

Miscellaneous:

Glucose: ECC Handbook p

Increases blood glucose in hypoglycemia; prevents hypoglycemia when insulin is used to treat hyperkalemia.

Naloxone: opiate antagonist ECC Handbook

Reverses respiratory depression effects of narcotics.

IV/IO: 0.4 to 2 mg/ **dose** IV/IM/subcutaneously. May repeat every 2 to 3 minutes

Caution: half-life is usually less than the half-life of narcotic, so repeat dosing is often required;
ET dose can be given but is **not preferred**; can also give IM or SQ.

Sodium bicarbonate: pH buffer for prolonged arrest, hyperkalemia, tricyclic overdose: ECC Handbook

IV/IO: Increases blood pH helping to correct metabolic acidosis.
Moderate metabolic acidosis: 50 to 150 mEq sodium bicarbonate diluted in 1 L of D5W to be intravenously infused at a rate of 1 to 1.5 L/hour during the first hour.

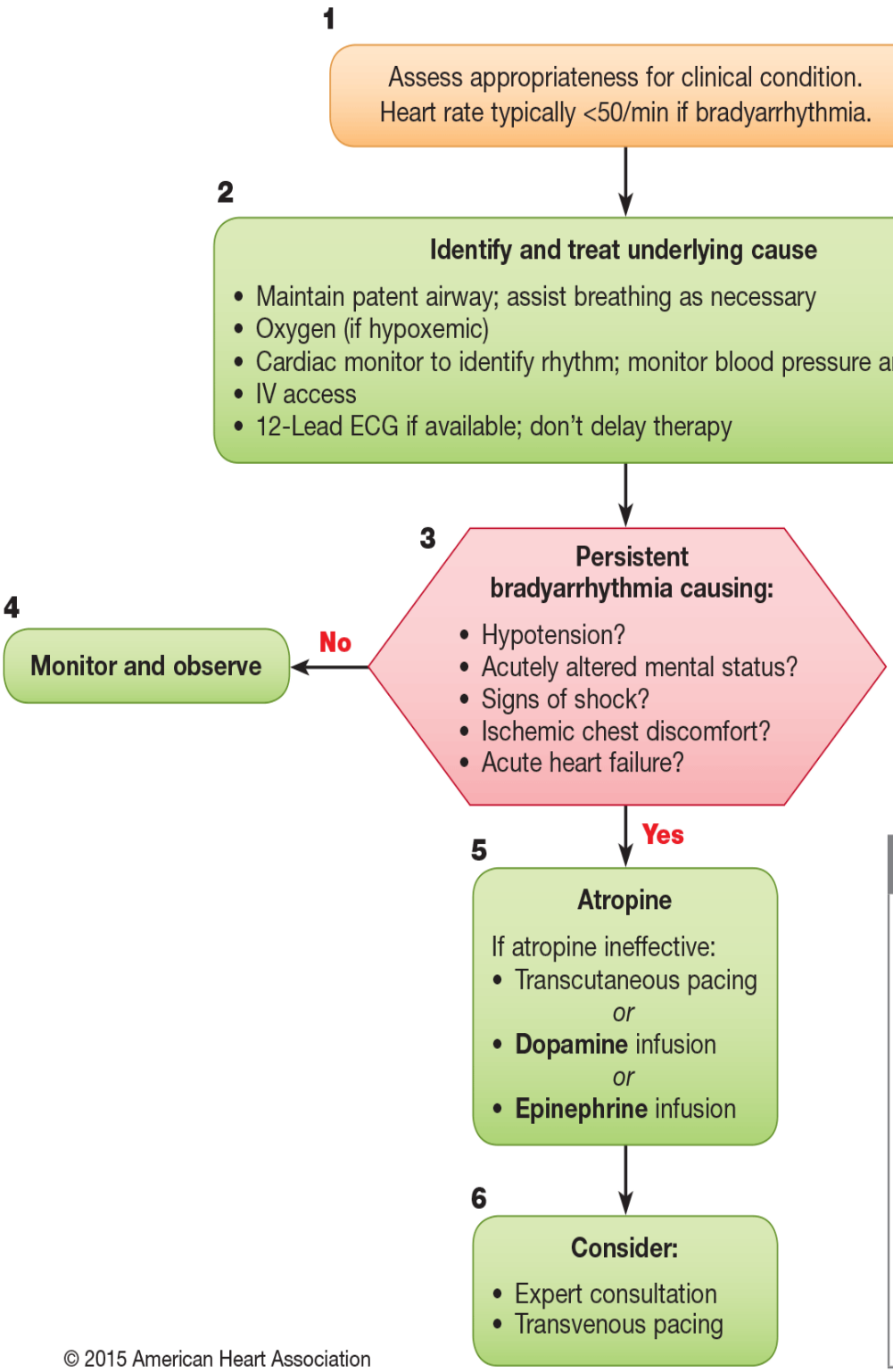
Severe metabolic acidosis: 90 to 180 mEq sodium bicarbonate diluted in 1 L of D5W to be intravenously infused at a rate of 1 to 1.5 L/hour during the first hour.

If acid-base status is not available, dosages should be calculated as follows: 2 to 5 mEq/kg IV infusion over 4 to 8 hours; subsequent doses should be based on patient's acid-base status.

Caution: causes other drugs to precipitate so flush IV tubing before and after

ET drug administration: distribution is unpredictable as is the resulting blood level of the drug; **if there is no IV/IO** access, give the drug down the ET and flush with 5-!0 mL NS then give 5 ventilations to disperse the drug.

Adult Bradycardia With a Pulse Algorithm



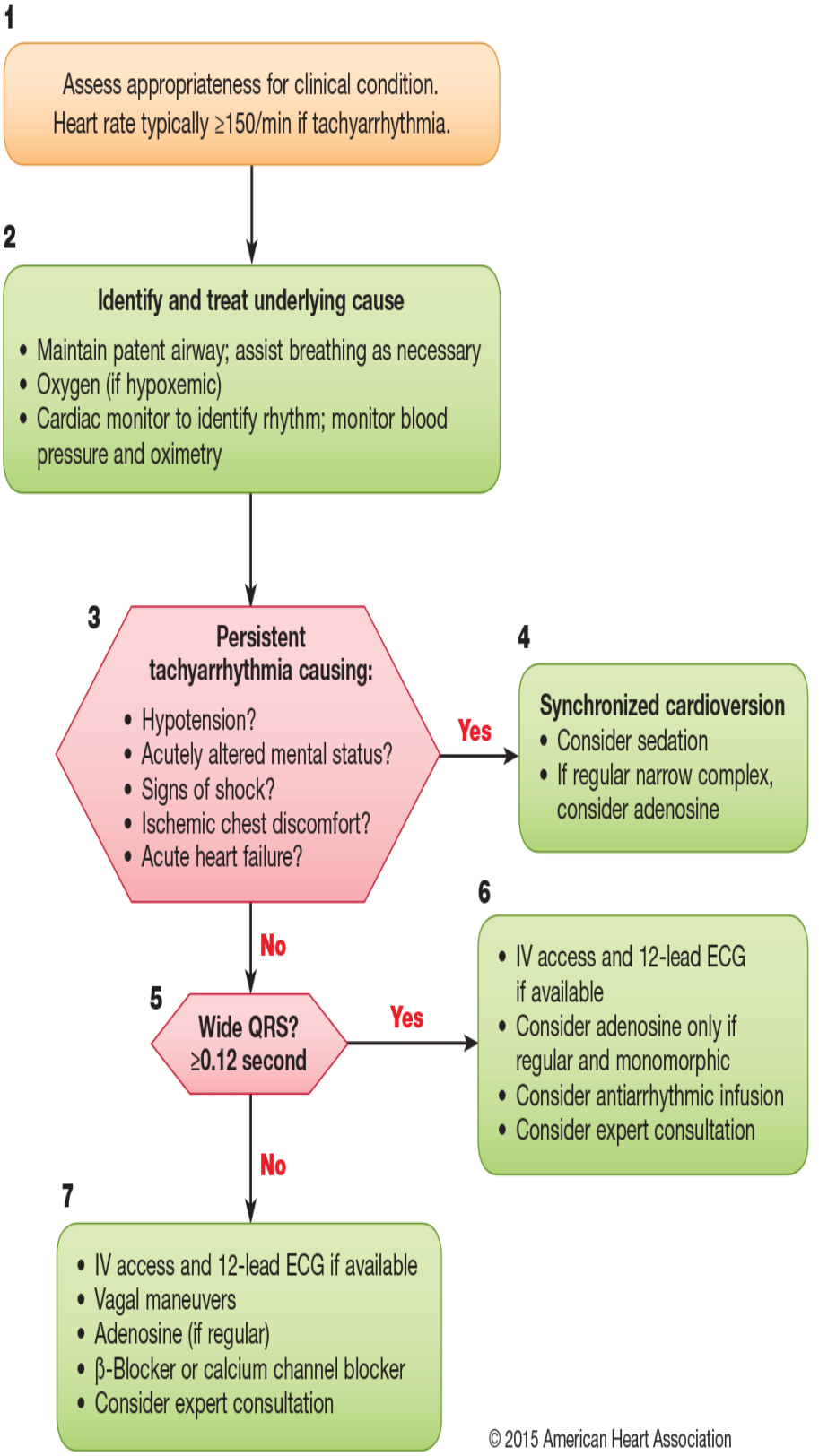
Doses/Details

Atropine IV dose:
First dose: 0.5 mg bolus. Repeat every 3-5 minutes. Maximum: 3 mg.

Dopamine IV infusion:
Usual infusion rate is 2-20 mcg/kg per minute. Titrate to patient response; taper slowly.

Epinephrine IV infusion:
2-10 mcg per minute infusion. Titrate to patient response.

Adult 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.

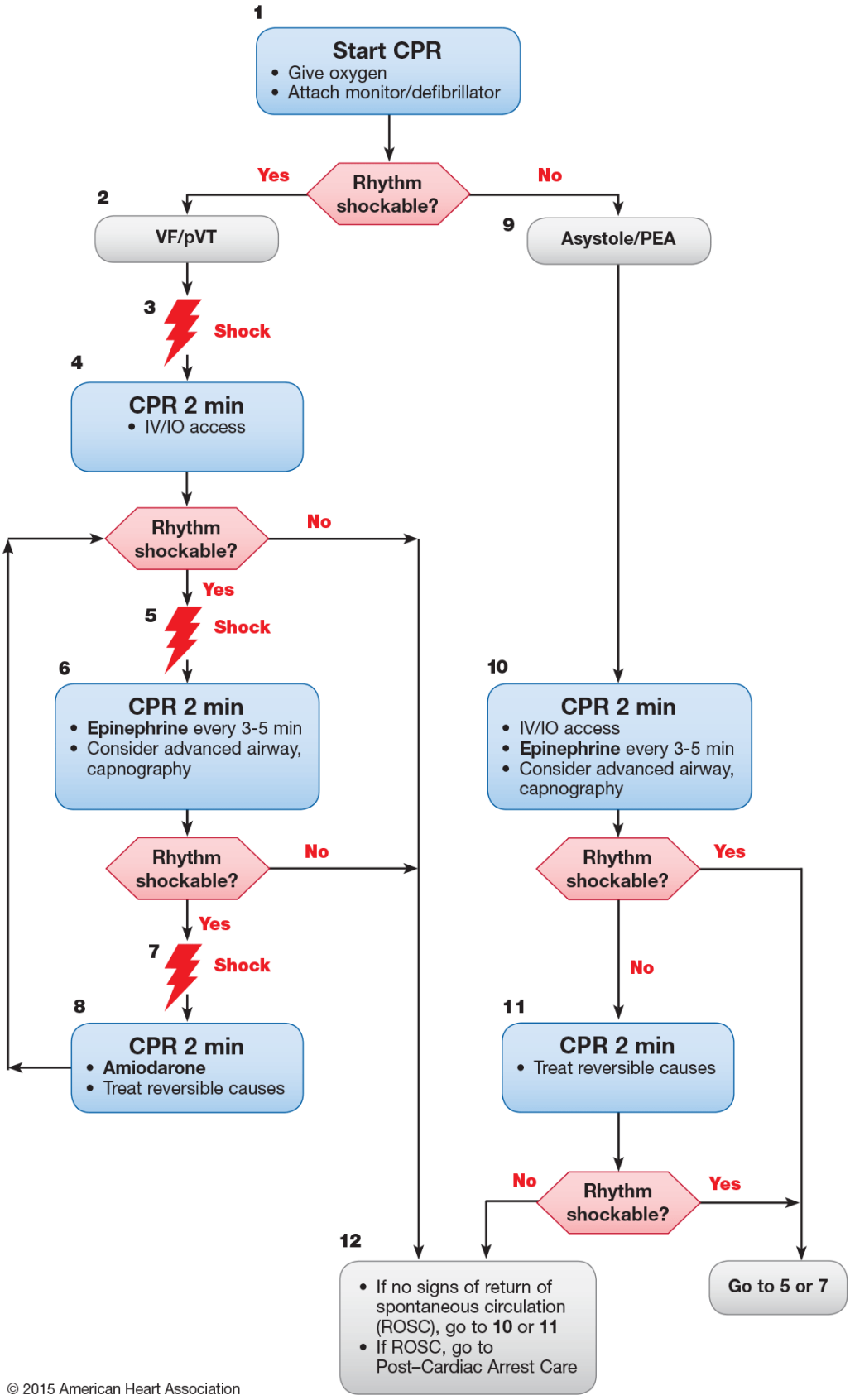
Antiarrhythmic Infusions for Stable Wide-QRS Tachycardia

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.

Adult Cardiac Arrest Algorithm—2015 Update



CPR Quality

- Push hard (at least 2 inches [5 cm]) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Rotate compressor every 2 minutes, or sooner if fatigued.
- 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.

Shock Energy for Defibrillation

- **Biphasic:** Manufacturer recommendation (eg, initial dose of 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
- **Amiodarone IV/IO dose:** First dose: 300 mg bolus. Second dose: 150 mg.

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 seconds (10 breaths/min) with continuous chest compressions

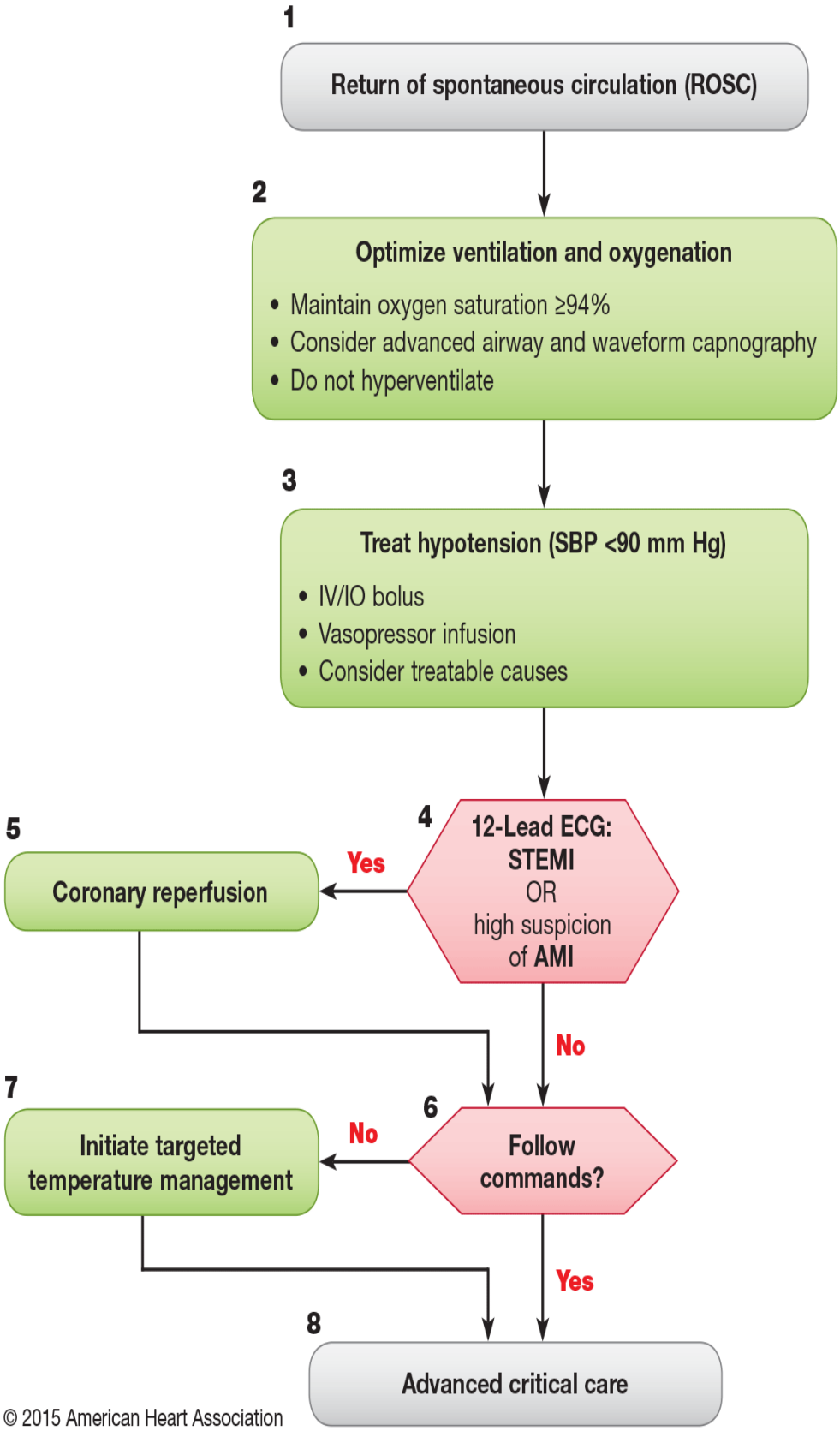
Return of Spontaneous Circulation (ROSC)

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

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

Adult Immediate Post–Cardiac Arrest Care Algorithm—2015 Update



Doses/Details

Ventilation/oxygenation:
Avoid excessive ventilation. Start at 10 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:
Approximately 1-2 L normal saline or lactated Ringer's

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

Acute Coronary Syndromes Algorithm—2015 Update

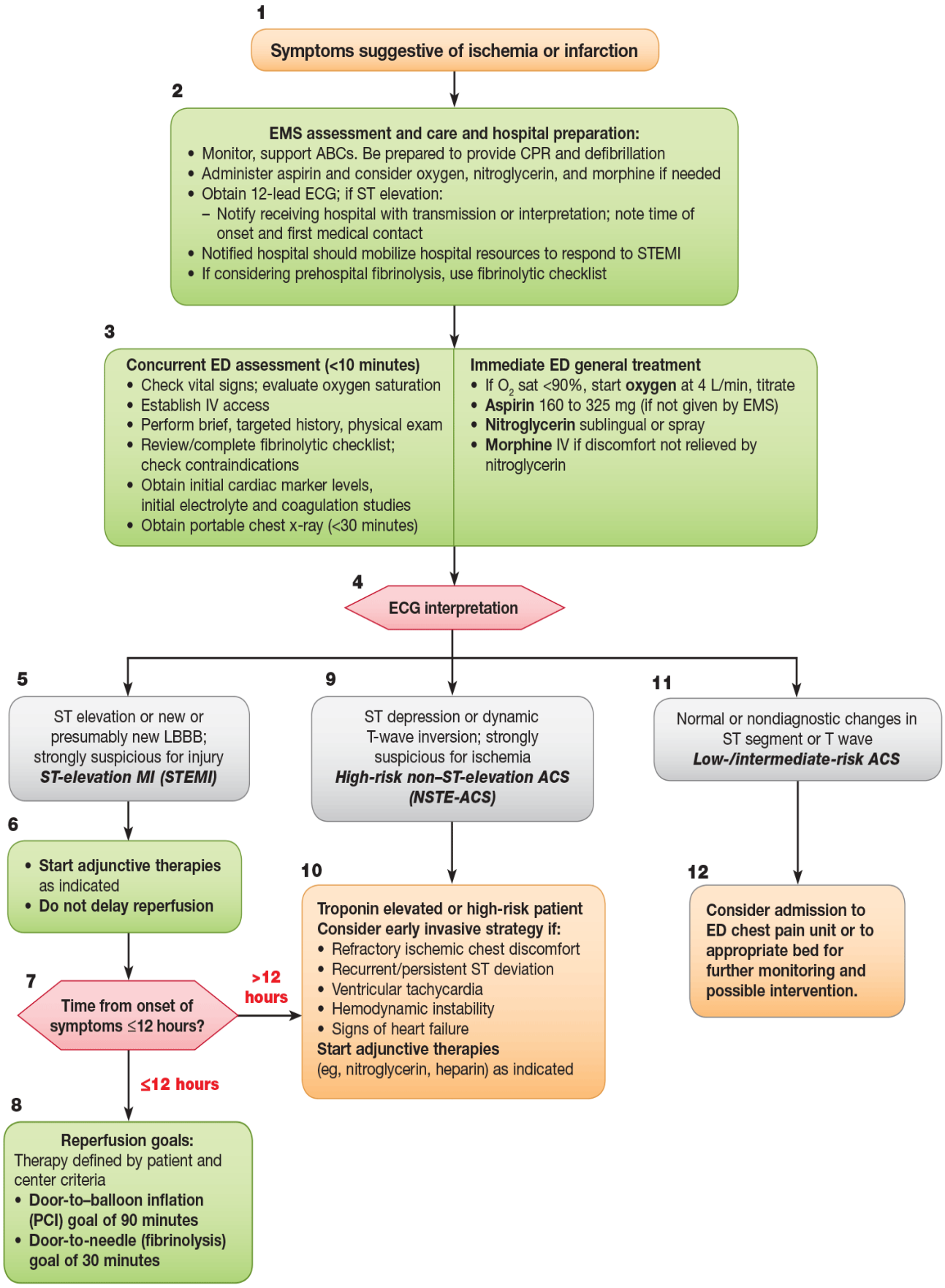
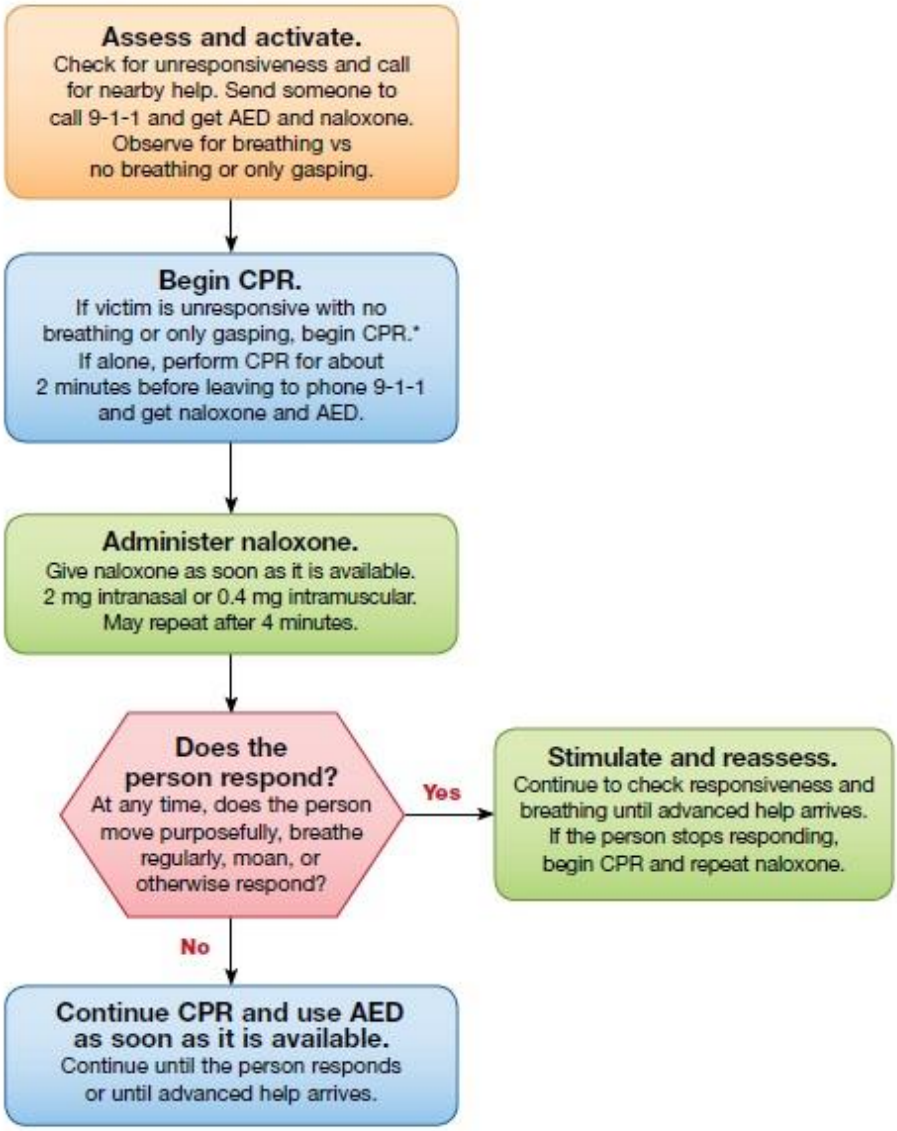


Figure 6

Opioid-Associated Life-Threatening Emergency (Adult) Algorithm—New 2015



*CPR technique based on rescuer's level of training.

arrest, the provision of naloxone may help an unresponsive patient with severe respiratory depression who only appears to be in cardiac arrest (ie, it is difficult to determine if a pulse is present).

Intravenous Lipid Emulsion

2015 (Updated): It may be reasonable to administer ILE, concomitant with standard resuscitative care, to patients who have premonitory neurotoxicity or cardiac arrest due to local anesthetic toxicity. It may be reasonable to administer ILE to patients with other forms of drug toxicity who are failing standard resuscitative measures.

2010 (Old): It may be reasonable to consider ILE for local anesthetic toxicity.

Why: Since 2010, published animal studies and human case reports have examined the use of ILE for patients with drug toxicity that is not the result of local anesthetic infusion. Although the results of these studies and reports

Positions for 6-Person High-Performance Teams*

American Heart Association®
life is why™

Resuscitation Triangle Roles

Compressor

- Assesses the patient
- Does 5 cycles of chest compressions
- Alternates with AED/Monitor/Defibrillator every 5 cycles or 2 minutes (or earlier if signs of fatigue set in)

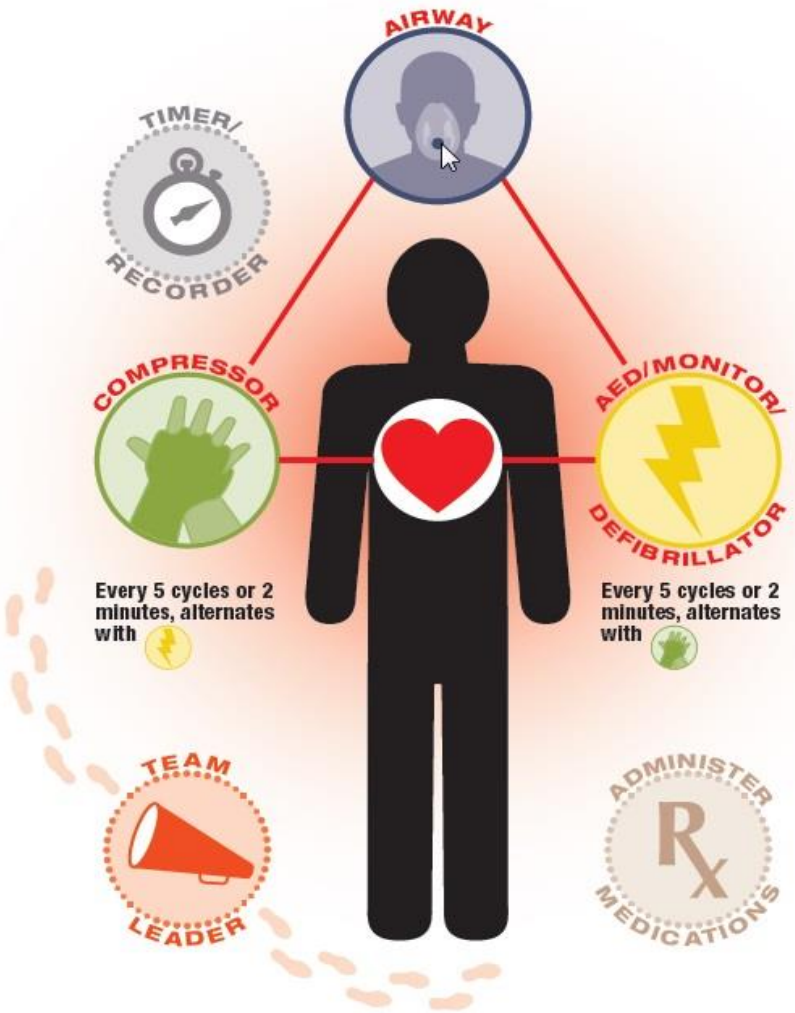
AED/Monitor/Defibrillator

- Brings and operates the AED/monitor/defibrillator
- Alternates with Compressor every 5 cycles or 2 minutes (or earlier if signs of fatigue set in), ideally during rhythm analysis
- If a monitor is present, places it in a position where it can be seen by the Team Leader (and most of the team)

Airway

- Opens and maintains the airway
- Provides ventilation

The team owns the code. No team member leaves the triangle except to protect his or her safety.



Leadership Roles

Team Leader

- **Every resuscitation team must have a defined leader**
- Assigns roles to team members
- Makes treatment decisions
- Provides feedback to the rest of the team as needed
- Assumes responsibility for roles not assigned

Administer Medications

- An ALS provider role
- Administers medications

Timer/Recorder

- Records the time of interventions and medications (and announces when these are next due)
- Records the frequency and duration of interruptions in compressions
- Communicates these to the Team Leader (and the rest of the team)

*This is a suggested team formation. Roles may be adapted to local protocol.

ECG REVEIW

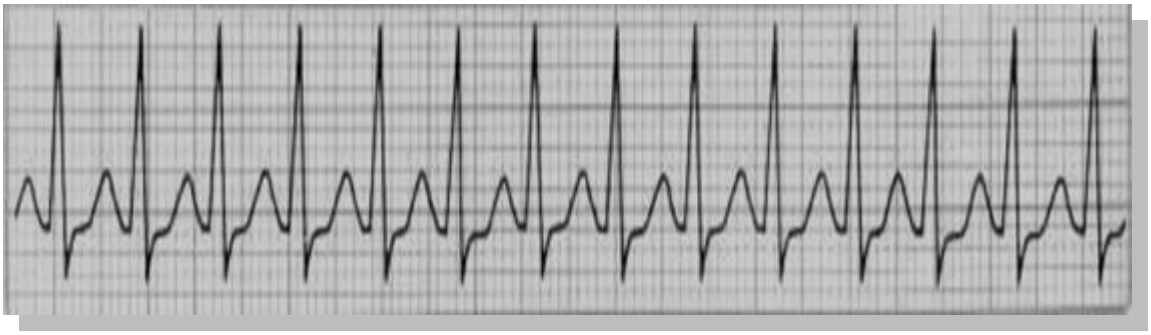
1

Rhythm **SINUS TACH**

2

a. **Rhythm Sinus Rhythm**

3



Rhythm <u>SVT</u>

4

a. **Rhythm : Atrial Flutter**

5

a. **Rhythm: Sinus Brady**

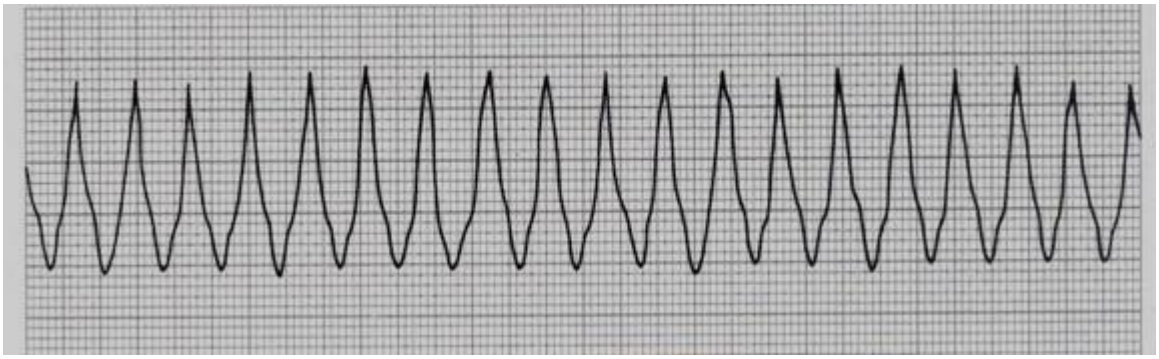
6

Rhythm : Atrial Fibrillation (No regular Ps, variable rate and fibrillatory baseline)

7

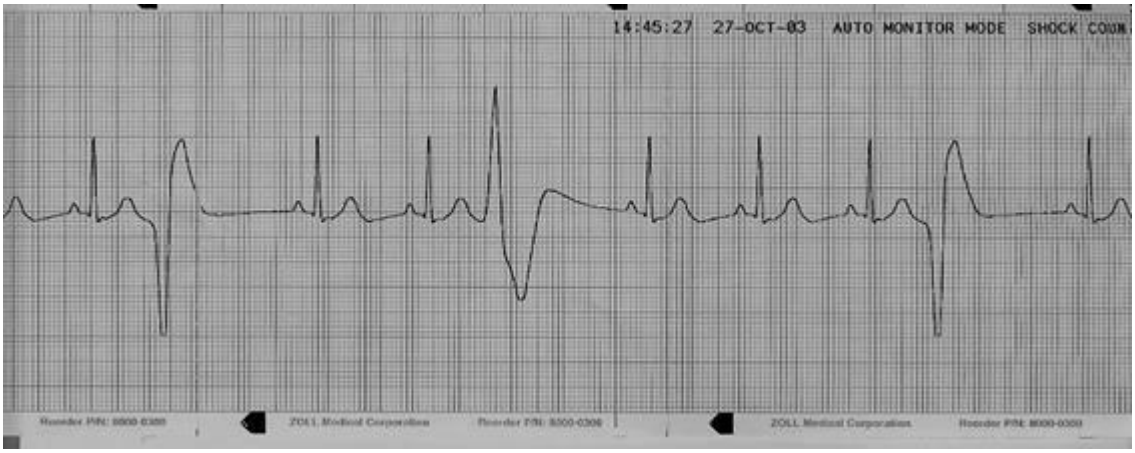
Rhythm : Junctional Rhythm.~ 60 bpm

8



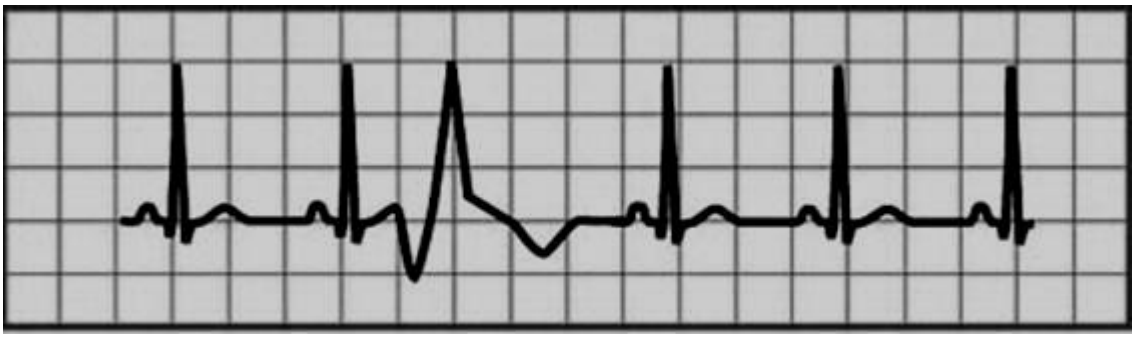
Rhythm : Monomorphic V-Tach

9



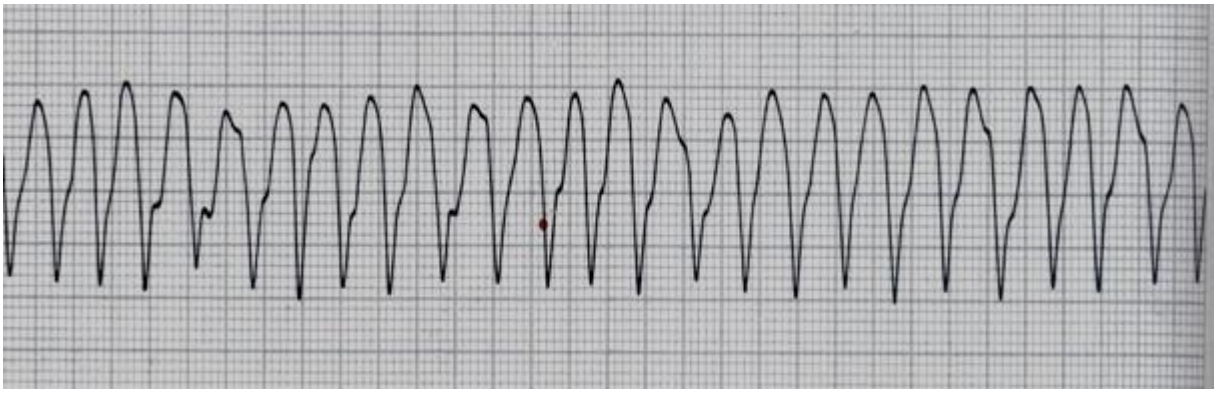
Rhythm : Sinus Rhythm W/ multifocal PVC's

10



Rhythm: Sinus Rhythm W/ PVC

11

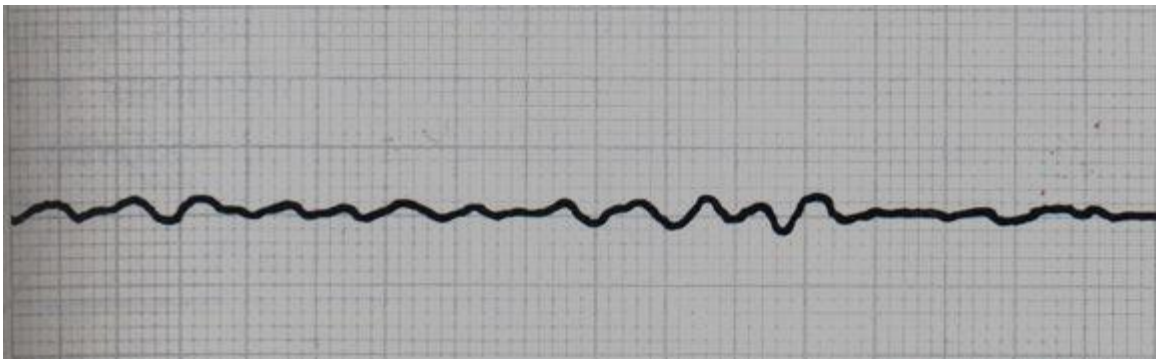


Rhythm : Polymorphic V-Tach (Probably normal QT)

12

a. **Rhythm: 2nd Degree type II**

13



Rhythm : Fine V-Fib

14

a. Rhythm : 1 Degree AVB

15

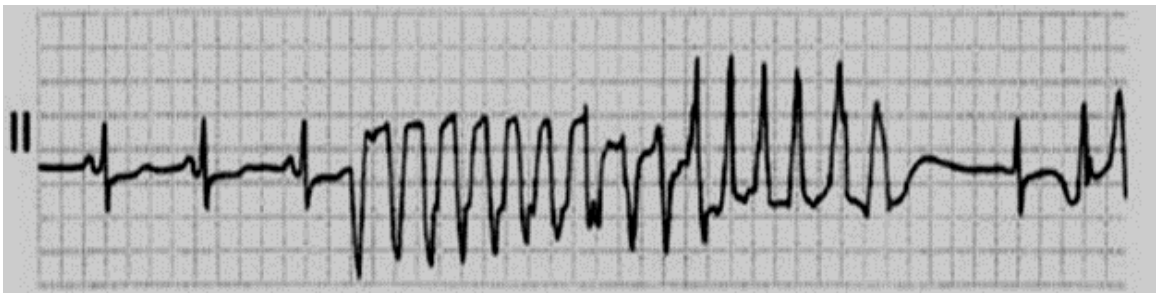
Rhythm: Coarse V-Fib

16

Rhythm : Sinus Rhythm W/PAC

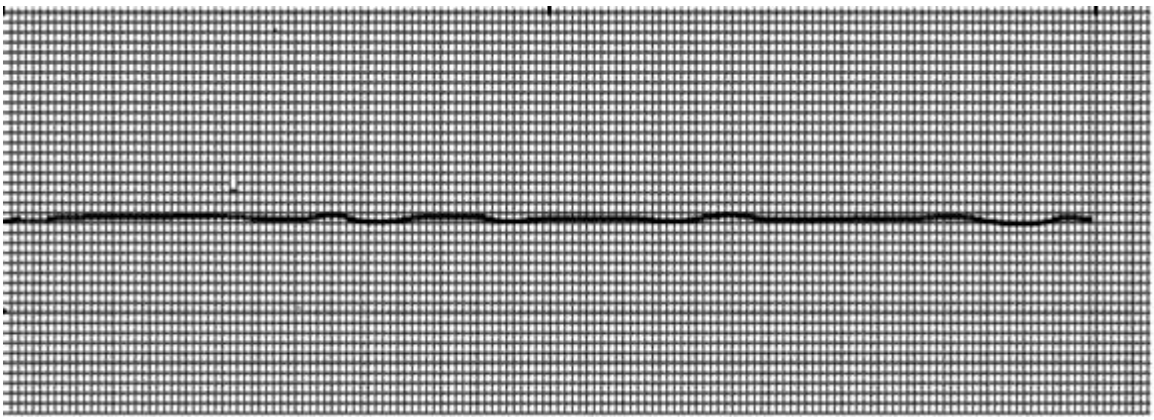
Rhythm: 2nd Degree type I

18



Rhythm: Polymorphic V-Tach / Torsades de Points

19



Rhythm: Asystole

20

Rhythm: 3rd Degree

How to use the H's and T's.

THE H's and T's – POTENTIALLY REVERSIBLE CAUSES

You must use these on all cardiac arrests and near cardiac arrests.

H's	T's
• **Hypovolemia** • **Hypoxia** • **Hydrogen ion – acidosis** • **Hyperkalemia / Hypokalemia** • **Hypothermia** • **Hypoglycemia and other metabolic disorders**	• **Tablets (drug OD, accidents)** • **Tamponade (cardiac)** • **Tension pneumothorax** • **Thrombosis, coronary (ACS)** • **Thrombosis, pulmonary (embolism)** • **Trauma**

Hypovolemia (is this pt hypovolemic?)

1. Look for obvious fluid/blood loss.
2. Secure IO/IV access
3. Give fluid boluses and reassess
4. Check mark for Hypovolemia

Hypoxia (is this person hypoxic?)

1. Confirm chest rise and bilateral breath sounds with each ventilation
2. Check O2 source (trace from bag to flow meter)
3. Check mark for hypoxia

Hydrogen Ion Acidosis (is this pt acidotic?) (Respiratory or metabolic)

1. Respiratory acidosis ensure adequate ventilation (don't hyperventilate!)
2. Metabolic acidosis give sodium bicarbonate
3. Check mark for acidosis

Hyper /Hypokalemia (is there any evidence hyper/hypokalemia in this pt?)

1. For elevated S-T's and tall peaked T waves (hyperkalemia) give calcium chloride 10ml of 10% over 5 minutes
2. Hypokalemia, (flat T-waves & U waves) give potassium 20 to 30 meq/hour, Magnesium 1 to 2 g (2 to 4 ml of 50% solution) diluted in 10 ml of D5W IV/IO
4. If no signs of hyper/hypokalemia move to the next H.
5. Checkmark for hyper/hypokalemia

Hyper/Hypothermia (take a temp)

1. If too hot, cool down
2. If too cold, warm up
3. If normothermic or mildly hypothermic go to the next H.
4. Check mark for Hyper/hypothermia

Hypo/Hyperglycemia

1. Accu-check and correct if needed.
2. If normoglycemic move to the T's Checkmark for Hypo/hyperglycemia

Tablets (drug OD, accidents)

1. Support circulation while you find an antidote or Reversal drug- (Poison control)
2. If no drug OD suspected, move on to the next T. Check mark for tablets

Tamponade (chest trauma, chest malignancy, recent central line insertion, JVD, narrow pulse pressure, electrical alternans etc...)

1. Pericardial centesis

If no history or ruled out move on to the next T and check mark for Tamponade

Tension Pneumothorax (chest asymmetry, tympani, diminished breath sounds, high peak pressures, JVD, tracheal deviation, severe respiratory distress etc...)

1. Vent tension in chest
2. Support ventilation and oxygenation with BVM and intubate as necessary
3. If no history or ruled out move on to the next T and check mark for pneumothorax

Thrombosis (coronary or pulmonary)

1. Consider fibrinolysis for suspected coronary or pulmonary embolus.
2. CPR is not an absolute contraindication for fibrinolysis.
3. If no history or ruled out move on to the next T and check mark for thrombosis

Trauma Inspect body completely. Remove all clothes.

1. Secure airway
2. Control external bleeding with tamponade while concurrently delivering volume with isotonic crystalloids and blood products.
3. Look for internal bleeding (tap the abdomen if suspicious for internal bleed)and take to OR within a couple of minutes.
5. If no history or ruled out move on to the next check mark for trauma Etc...

By Terry White Oct 2012

ADENOSINE
indications for use

*First drug for most forms of stable narrow complex SVT.
*Effective in terminating those due to reentry involving AV node or sinus node.

AMIODARONE
indications for use

*VF/pulseless VT unresponsive to shock delivery, CPR, and a vasopressor.
*Recurrent, hemodynamically unstable VT

ATROPINE SULFATE
indications for use

*First drug for symptomatic bradycardia
*May be beneficial in presence of AV nodal block
*Organophosphate poisoning

DOPAMINE
indications for use

*Second line drug for symptomatic bradycardia
*For hypotension with signs and symptoms of shock

EPINEPHRINE
indications for use

*Cardiac arrest: VF, pulseless VT, asystole, PEA
*Symptomatic bradycardia
*Severe hypotension
*Anaphylaxis, severe allergic reactions

LIDOCAINE
indications for use

*Alternative to amiodarone in cardiac arrest from VF/VT
*Stable monophasic VT with preserved ventricular function
*Stable polymorphic VT with normal baseline QT interval & preserves LV function
*Stable polymorphic VT with baseline QT-interval prolongation if torsades suspected

MAGNESIUM SULFATE
indications for use

*For use in cardiac arrest only if torsades-de-pointes or suspected hypomagnesemia present
*Life threatening ventricular arrhyhmias due to digitalis toxicity

Vasopressors for Resuscitation: Epinephrine

It may be reasonable to administer epinephrine as soon as feasible after the onset of cardiac arrest due to an initial nonshockable rhythm.

A very large observational study of cardiac arrest with nonshockable rhythm compared epinephrine given

at 1 to 3 minutes with epinephrine given at 3 later time intervals (4 to 6, 7 to 9, and greater than 9 minutes). The study found an association between early administration of epinephrine and increased ROSC, survival to hospital discharge, and neurologically intact survival.

$ETCO_2$ for Prediction of Failed Resuscitation

In intubated patients, failure to achieve an ETCO of greater than 10 mm Hg by waveform capnography after 20 minutes of CPR may be considered as one component of a multimodal approach to decide when to end resuscitative efforts but should not be used in isolation.
2

Failure to achieve an ETCO of 10 mm Hg by waveform capnography after 20 minutes of resuscitation has been associated with an extremely poor chance of ROSC and survival. However, the studies to date are limited in that they have potential confounders and have included relatively small numbers of patients, so it is inadvisable to rely solely on ETCO in determining when to terminate resuscitation.
2
2

Extracorporeal CPR

ECPR may be considered among select cardiac arrest patients who have not responded to initial conventional CPR, in settings where it can be rapidly implemented.

Although no high-quality studies have compared ECPR to conventional CPR, a number of lower-quality studies suggest improved survival with good neurologic outcome for select patient populations. Because ECPR is resource intensive and costly, it should be considered only when the patient has a reasonably high likelihood of benefit—in cases where the patient has a potentially reversible illness or to support a patient while waiting for a cardiac transplant.

Post–Cardiac Arrest Drug Therapy: Lidocaine

There is inadequate evidence to support the routine use of lidocaine after cardiac arrest. However, the initiation or continuation of lidocaine may be considered immediately after ROSC from cardiac arrest due to VF/pVT.

While earlier studies showed an association between giving lidocaine after myocardial infarction and increased mortality, a recent study of lidocaine in cardiac arrest survivors showed a decrease in the incidence of recurrent VF/pVT but did not show either long-term benefit or harm.

Post–Cardiac Arrest Drug Therapy: ß-Blockers

There is inadequate evidence to support the routine use of a ß-blocker after cardiac arrest. However, the initiation or continuation of an oral or IV ß-blocker may be considered early after hospitalization from cardiac arrest due to VF/pVT.

In an observational study of patients who had ROSC after VF/pVT cardiac arrest, ß-blocker administration was associated with higher survival rates. However, this finding is only an associative relationship, and the routine use of ß-blockers after cardiac arrest is potentially hazardous because ß-blockers can cause or worsen hemodynamic instability, exacerbate heart failure, and cause bradyarrhythmias. Therefore, providers should evaluate patients individually for their suitability for ß-blockers.

2 Introduction - Updated

These Web-based Integrated Guidelines incorporate the relevant recommendations from 2010 and the new or updated recommendations from 2015.

Basic life support (BLS), advanced cardiovascular life support (ACLS), and post–cardiac arrest care are labels of convenience that each describe a set of skills and knowledge that are applied sequentially during the treatment of patients who have a cardiac arrest. There is overlap as each stage of care progresses to the next, but generally ACLS comprises the level of care between BLS and post–cardiac arrest care.

ACLS training is recommended for advanced providers of both prehospital and in-hospital medical care. In the past, much of the data regarding resuscitation was gathered from out-of-hospital arrests, but in recent years, data have also been collected from in-hospital arrests, allowing for a comparison of cardiac arrest and resuscitation in these 2 settings. While there are many similarities, there are also some differences between in-

and out-of-hospital cardiac arrest etiology, which may lead to changes in recommended resuscitation treatment or in sequencing of care. The consideration of steroid administration for in-hospital cardiac arrest (IHCA) versus out-of-hospital cardiac arrest (OHCA) is one such example discussed in this Part.

The recommendations in this *2015 American Heart Association* (AHA) *Guidelines Update for Cardiopulmonary Resuscitation* (CPR) *and Emergency Cardiovascular Care* (ECC) are based on an extensive evidence review process that was begun by the International Liaison Committee on Resuscitation (ILCOR) after the publication of the ILCOR *2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular*

Care Science With Treatment Recommendations and was completed in February 2015.[1]
In this in-depth evidence review process, the ILCOR task forces examined topics and then generated prioritized lists of questions for systematic review. Questions were first formulated in PICO (population, intervention, comparator, outcome) format, and then a search strategy and inclusion and exclusion criteria were defined and a search for relevant articles was performed. The evidence was evaluated by using the standardized methodological approach proposed by the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) Working Group.
[2]
[3]
The quality of the evidence was categorized based on the study methodologies and the 5 core GRADE domains of risk of bias, inconsistency, indirectness, imprecision, and other considerations (including publication bias). Then, where possible, consensus-based treatment recommendations were created.
To create the 2015 Guidelines Update, the AHA formed 15 writing groups, with careful attention to avoid or manage conflicts of interest, to assess the ILCOR treatment recommendations and to write AHA treatment recommendations by using the AHA Class of Recommendation and Level of Evidence (LOE) system.
The recommendations made in this 2015 Guidelines Update are informed by the ILCOR recommendations and GRADE classification, in the context of the delivery of medical care in North America. The AHA ACLS writing group made new recommendations only on topics specifically reviewed by ILCOR in 2015. This chapter delineates any instances where the AHA writing group developed recommendations that are substantially different than the ILCOR statements. In the online version of this document, live links are provided so the reader can connect directly to the systematic reviews on the Scientific Evidence Evaluation and Review System (SEERS) website. These links are indicated by a superscript combination of letters and numbers (eg, ALS 433).
This update uses the newest AHA COR and LOE classification system, which contains modifications of the Class III recommendation and introduces LOE B-R (randomized studies) and B-NR (nonrandomized studies) as well as LOE C-LD (limited data) and LOE C-EO (consensus of expert opinion). All recommendations made in this 2015 Guidelines Update, as well as in the 2010 Guidelines, are listed in the Appendix. For further information, see "Part 2: Evidence Evaluation and Management of Conflicts of Interest." The ILCOR ACLS Task Force addressed 37 PICO questions related to ACLS care (presented in this Part) in 2015. These questions included oxygen dose during CPR, advanced airway devices, ventilation rate during CPR, exhaled carbon dioxide (CO_2) detection for confirmation of airway placement, physiologic monitoring during CPR, prognostication during CPR, defibrillation, antiarrhythmic drugs, and vasopressors. The 2 new topics are steroids and hormones in cardiac arrest, and extracorporeal CPR (ECPR), perhaps better known to the inpatient provider community as extracorporeal life support (ECMO). The 2010 Guidelines Part on electrical therapies (defibrillation and emergency pacing) has been eliminated, and relevant material from it is now included in this ACLS Part.
The major changes in the 2015 ACLS guidelines include recommendations about prognostication during CPR based on exhaled CO_2 measurements, timing of epinephrine administration stratified by shockable or nonshockable rhythms, and the possibility of bundling treatment of steroids, vasopressin, and epinephrine for treatment of in-hospital arrests. In addition, the administration of vasopressin as the sole vasoactive drug during CPR has been removed from the algorithm.

3 Adjuncts to CPR - Updated

3.1 Oxygen Dose During CPR - Updated ALS 889

The 2015 ILCOR systematic review considered inhaled oxygen delivery both during CPR and in the post–cardiac arrest period. This 2015 Guidelines Update evaluates the optimal inspired concentration of oxygen during CPR.
The immediate goals of CPR are to restore the energy state of the heart so it can resume mechanical work and to maintain the energy state of the brain to minimize ischemic injury. Adequate oxygen delivery is necessary to achieve these goals. Oxygen delivery is dependent on both blood flow and arterial oxygen content. Because