2025 ACLS

Study Guide

Bulletin: **New resuscitation science and American Heart Association treatment guidelines were released**

October 25, 2025!

The new AHA Handbook of Emergency Cardiac Care (ECC) contains these 2025 Guidelines and is required study for this course. The 2025 ACLS Provider Manual is not yet available. This study guide will provide you with additional study information.

Self Assessment https://shopcpr.heart.org/acls-prework?srsltid=AfmBOooqiY-N8QS_nzaEtey-hg_r3yC9yQ2Es2Ypv66GCfLZrLrGzaVj

www.phsinstitute.com StudyGuide2025_ACLS_.pdf **(for study info. on rhythm review)**

What is required to successfully complete ACLS?

- **Completed ACLS Pre-test/ Self Assessment 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 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, Atrial Fibrillation, Atrial 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.

The AHA 2025 ACLS guidelines, released in late 2025, feature sharper, more evidence-based updates focused on precision, real-time feedback, and clearer decision-making, emphasizing earlier identification of cardiopulmonary compromise in bradycardia, tighter airway management with capnography, and consistent team communication, with key changes including atropine dosing, epinephrine timing, and refined post-cardiac arrest care, all aiming for improved outcomes in cardiac arrest and related emergencies.

Key Algorithm Updates

- Bradycardia: Earlier assessment for cardiopulmonary compromise (hypotension, AMS, shock) moved to the front of the algorithm;
- Cardiac Arrest (VF/pVT): Focus on precise timing for epinephrine and shock delivery, increased use of feedback devices (depth/rate), and less emphasis on double sequential defibrillation.
- Cardiac Arrest (PEA/Asystole): Stronger push for identifying reversible causes (Hs & Ts) and using capnography to monitor perfusion.
- Post-Cardiac Arrest Care: Refined protocols for airway (1 breath every 6s, avoid over-ventilation), managing hypotension (MAP < 65 mmHg), temperature management, and urgent 12-lead ECG for PCI consideration.

Airway & Oxygenation

- Advanced Airways: Waveform capnography is crucial for confirming placement and monitoring effectiveness.
- Ventilation: 1 breath every 6 seconds for patients with an advanced airway; avoid over-ventilation.

- Oxygen: 100% oxygen for arrest; titrate to 90-98% saturation post-ROSC.

Medications & Interventions

- Epinephrine: Administer ASAP in arrest (1mg q3-5min); high-dose not recommended.
- Vasopressin: Offers no advantage over epinephrine.
- Lidocaine/Beta-blockers: Insufficient evidence for routine use within the first hour post-ROSC.

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 CAB:*** **(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 Color** **O_2 is being inhaled:** expected. **Tube**
    - **change** on exhalation = **CO_2!!** **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 100 to 200 J. The initial monophasic dose for cardioversion of atrial fibrillation and atrial flutter is 200 J.

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 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

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

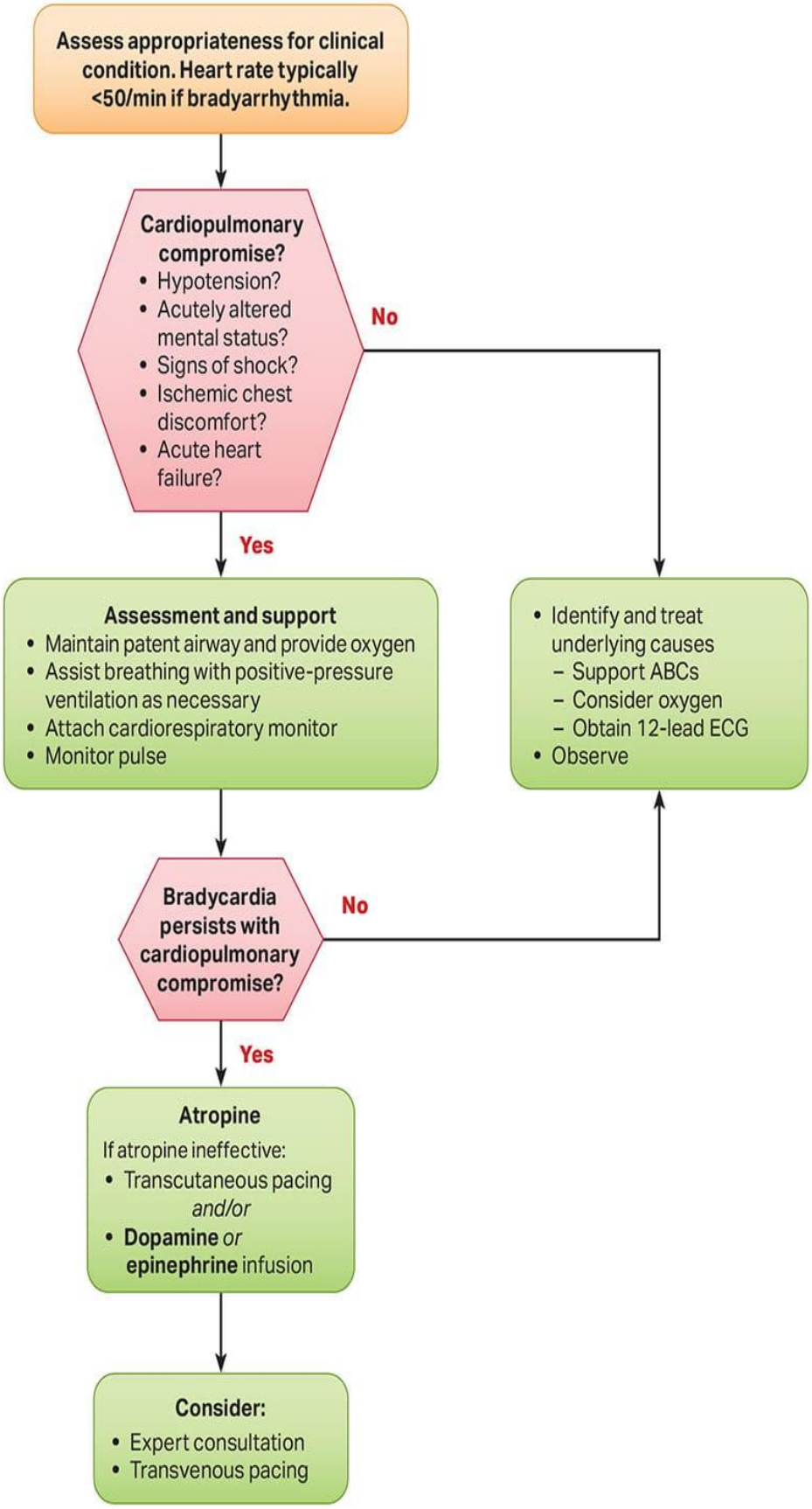
IV/IO: Increases blood pH helping to correct metabolic acidosis.
Moderate metabolic acidosis: 50 to 150 mEq sodium bicarbonate diluted in 1 L of D5W to be intravenously infused at a rate of 1 to 1.5 L/hour during the first hour.

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

Adult Bradycardia With a Pulse Algorithm



Doses/Details

Atropine IV dose:
First dose: 1 mg bolus.
Repeat every 3-5 minutes.
Maximum total dose: 3 mg.

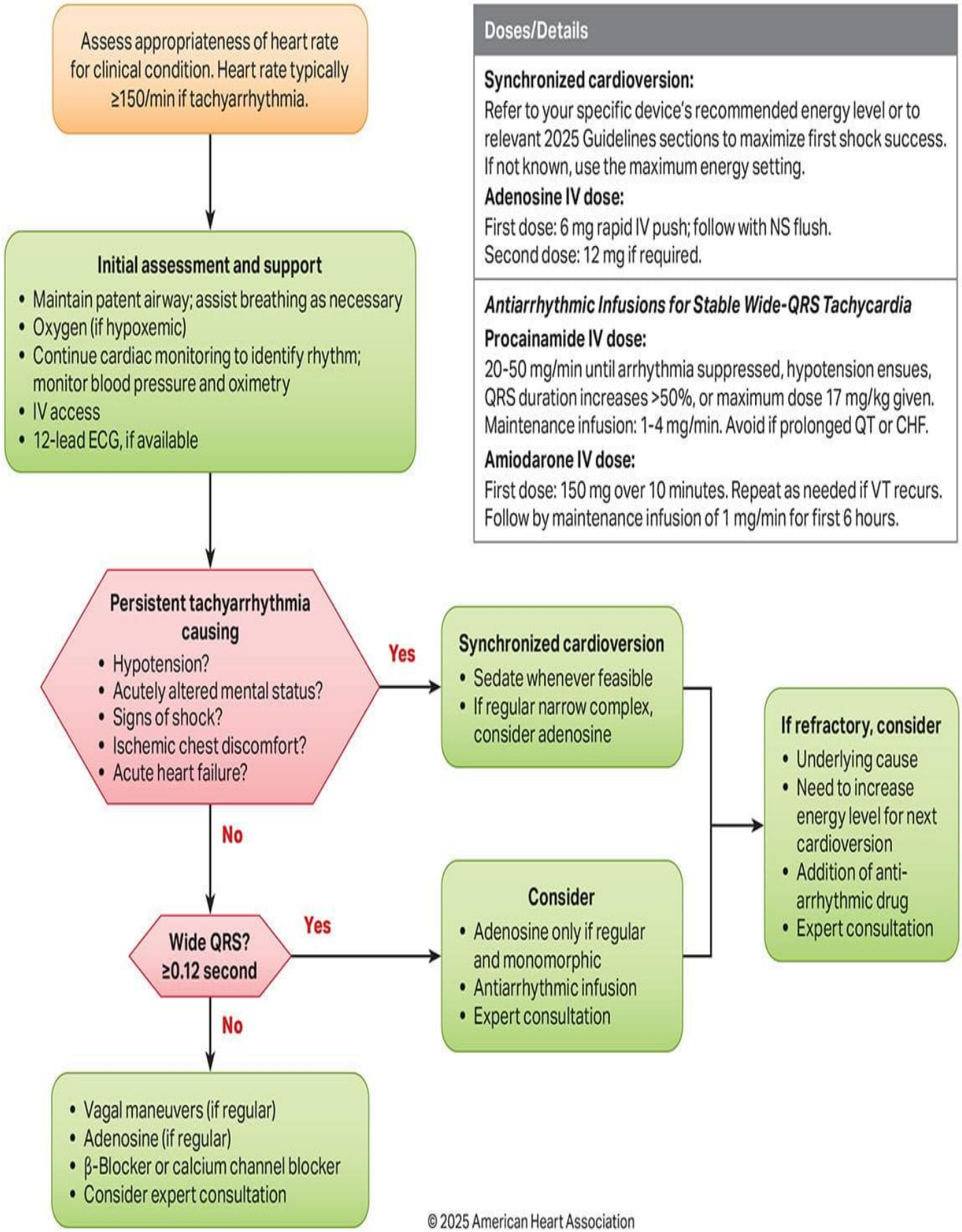
Dopamine IV infusion:
Usual infusion rate is 5-20 mcg/kg per minute.
Titrate to patient response; taper slowly.

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

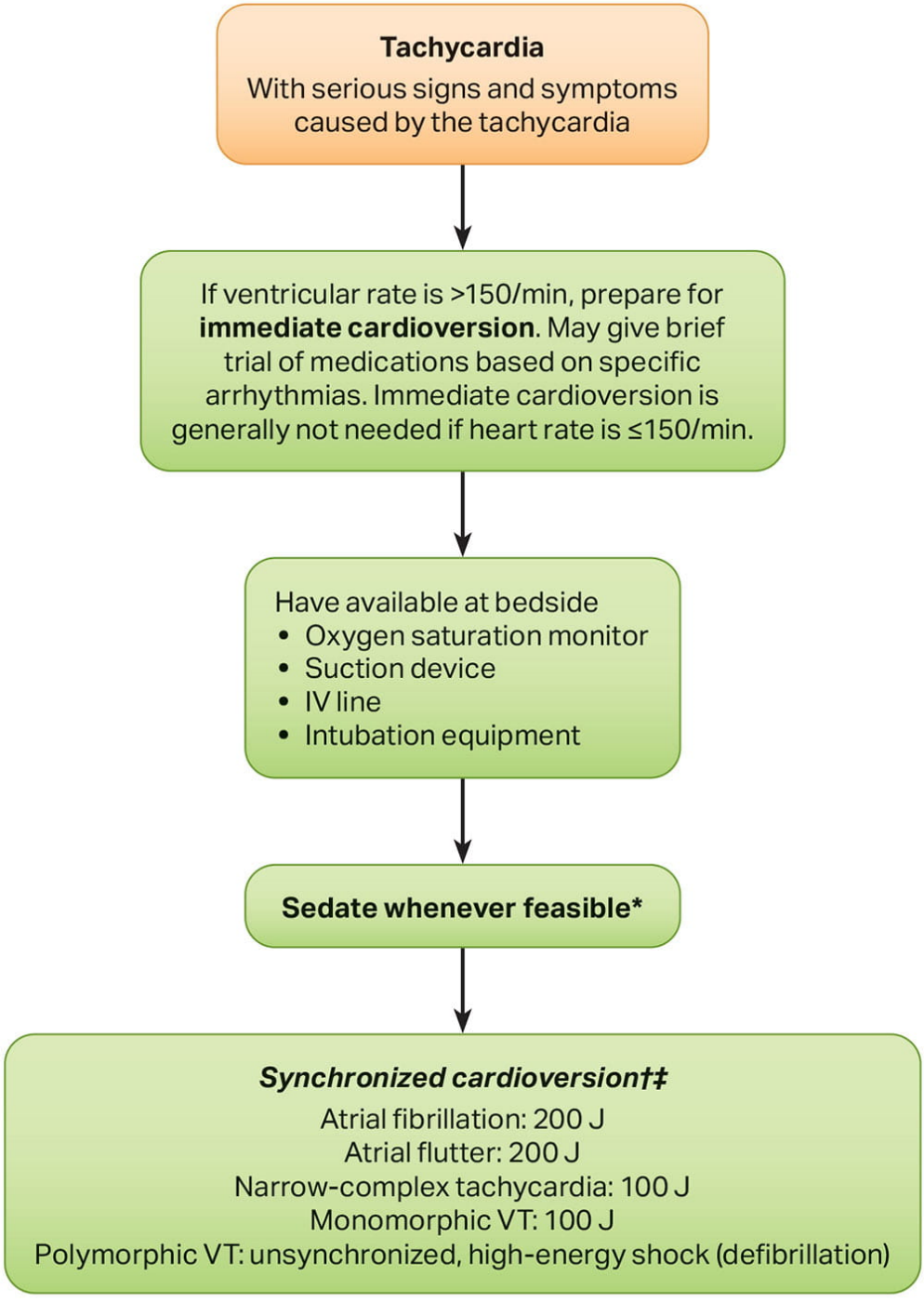
Possible Causes

- Myocardial ischemia/infarction
- Drugs/toxicologic (eg, calcium-channel blockers, β-blockers, digoxin)
- Hypoxia
- Electrolyte abnormality (eg, hyperkalemia)

Adult Tachyarrhythmia With a Pulse Algorithm



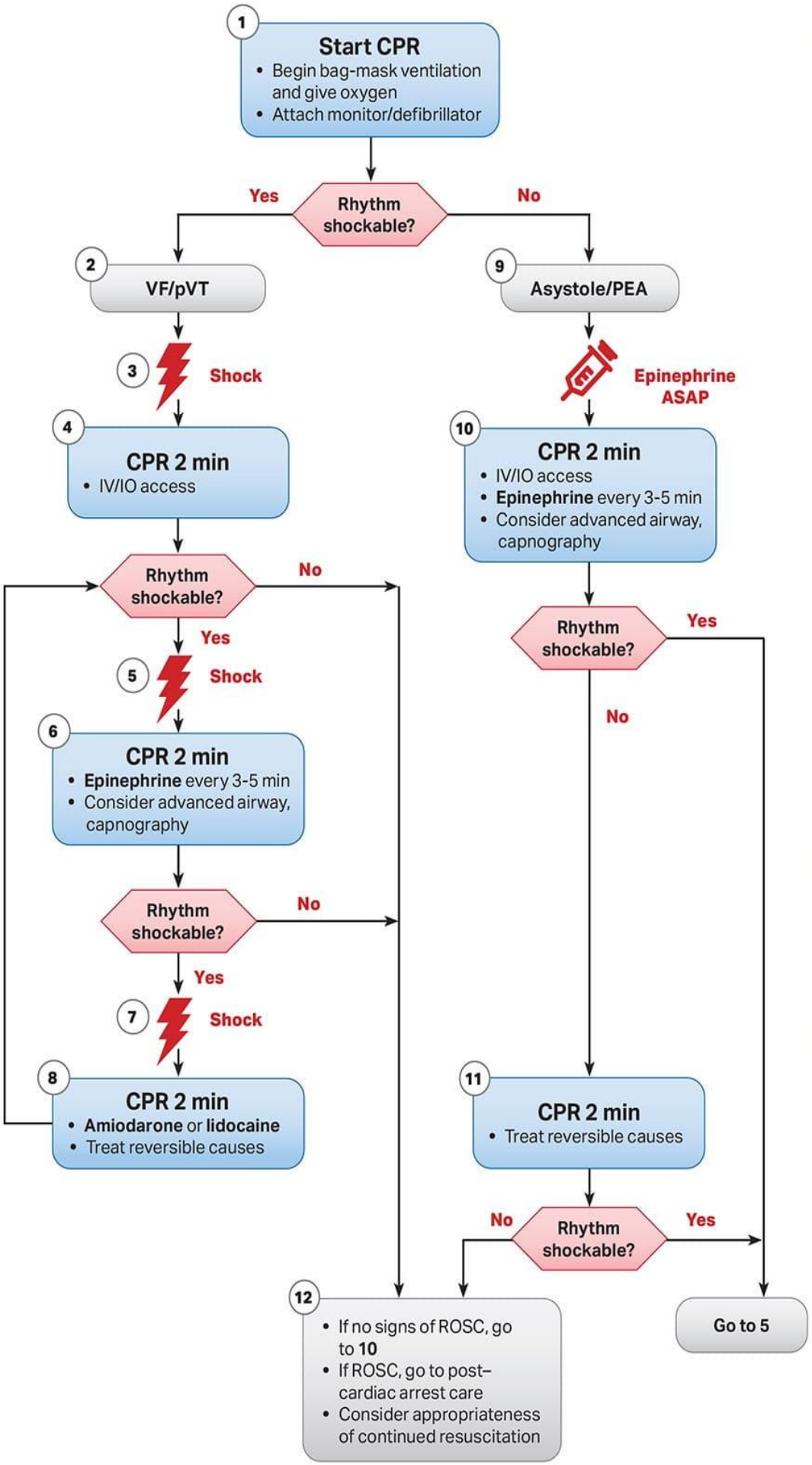
Electrical Cardioversion Algorithm



Notes

*Effective regimens have included a sedative **(eg, diazepam)** with or without an analgesic agent **(eg, fentanyl)**. Many experts recommend anesthesia if service is readily available.
†Note possible need to resynchronize after each cardioversion.
‡If delays in synchronization occur and clinical condition is critical, go immediately to unsynchronized shocks.

Adult Cardiac Arrest Algorithm (VF/pVT/Asystole/PEA)



High-Quality CPR

- Push hard (at least 2 inches [5 cm]).
- Push fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Change compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, use 30:2 compression-ventilation ratio.
- If advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions.
- Continuous waveform capnography
  - If $ETCO_2$ is low or decreasing, reassess 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 *or* **Lidocaine IV/IO dose:** First dose: 1-1.5 mg/kg Second dose: 0.5-0.75 mg/kg

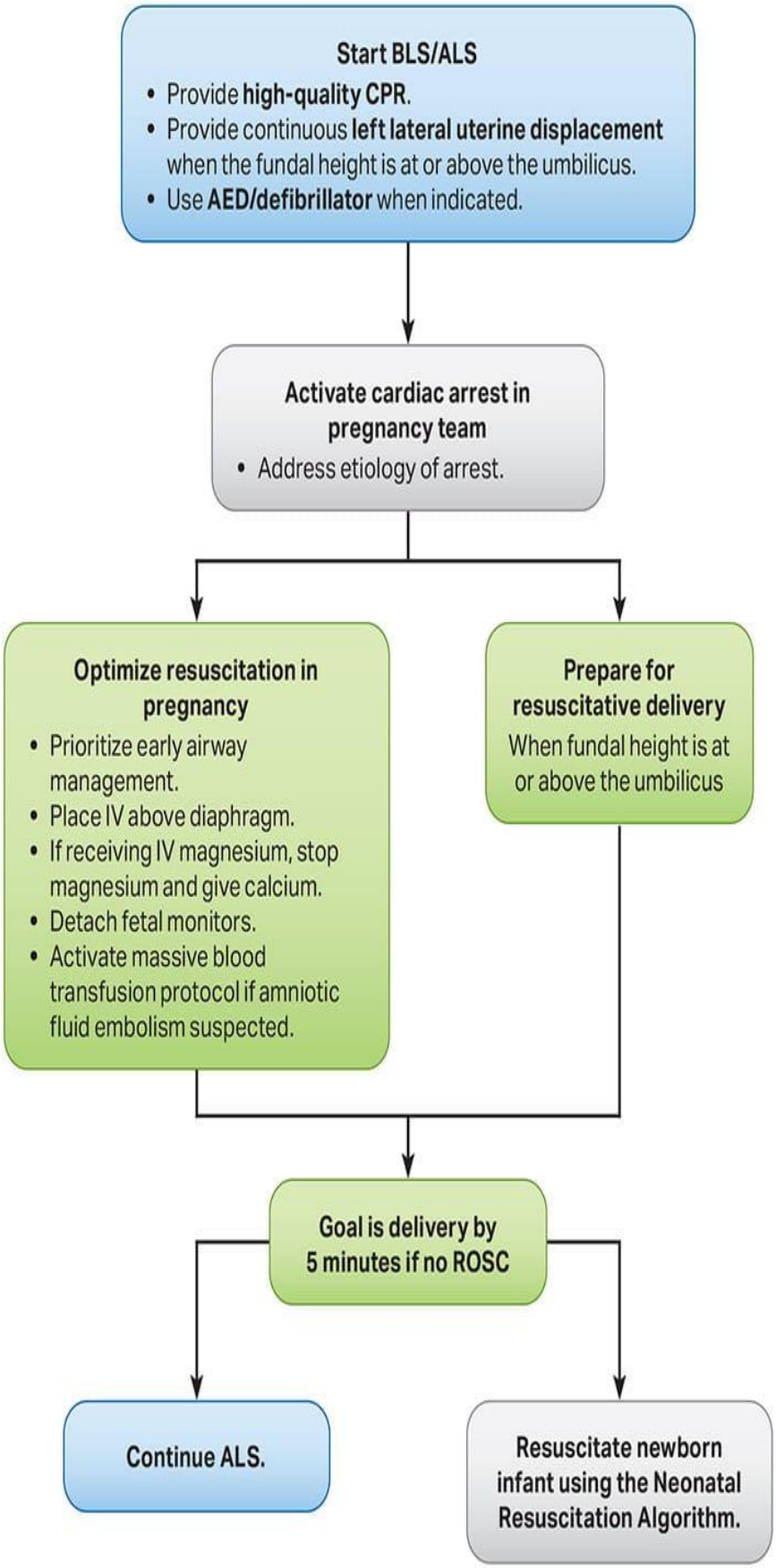
Advanced Airway

- ET intubation or supraglottic advanced airway
- Continuous waveform capnography or capnometry to confirm and monitor ET tube placement

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 in Pregnancy Algorithm



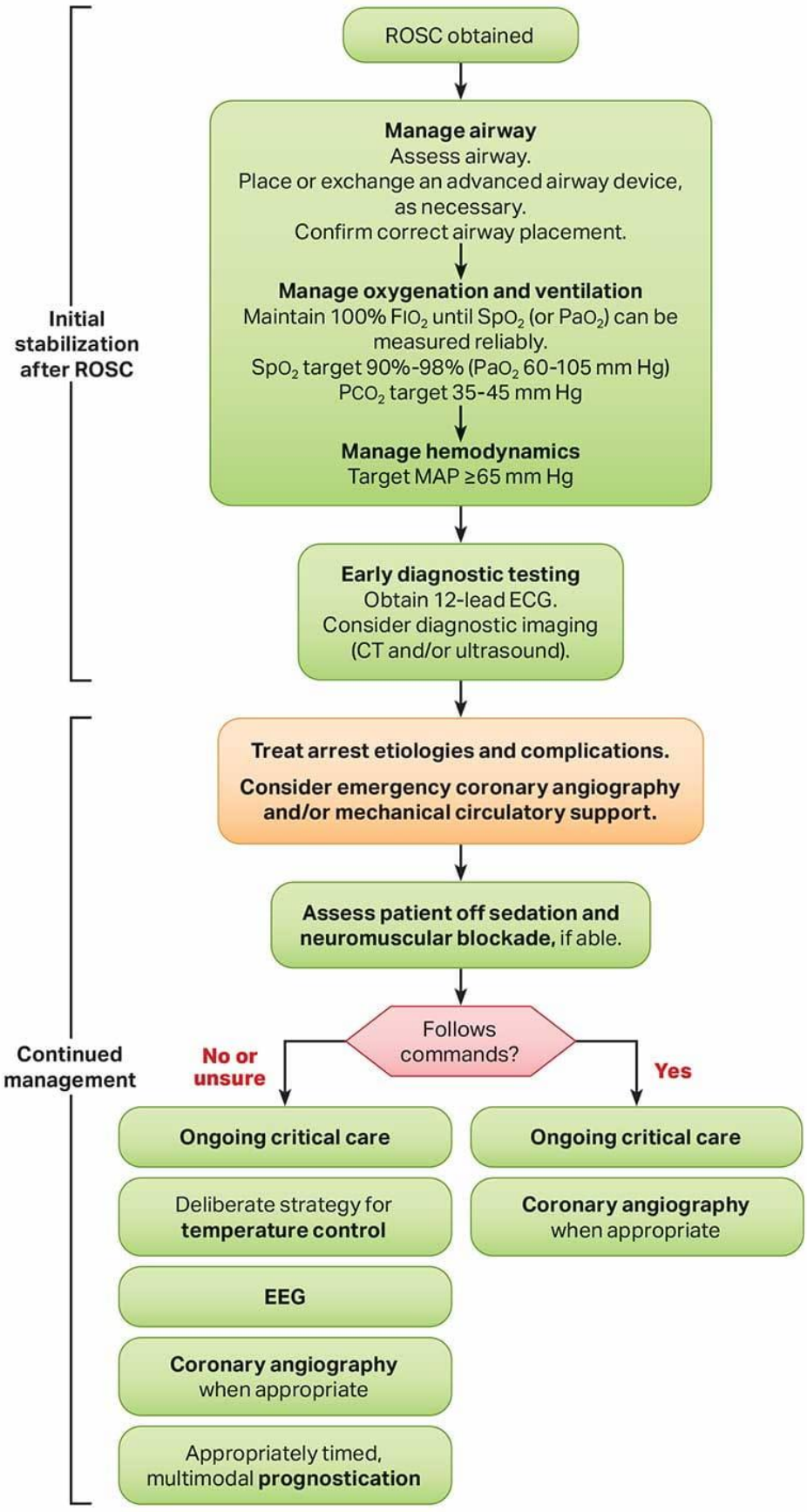
Explanation of Cardiac Arrest Interventions

- Cardiac arrest in pregnancy team will vary according to local resources but may include
  - Team Leader
  - Anesthesiologist
  - Obstetrician
  - Neonatologist
  - Nurses
  - Pharmacists
  - Other professionals
- The goal of left lateral uterine displacement is to relieve aortocaval compression and to facilitate effective chest compressions.
- The goal of resuscitative delivery is to improve the pregnant patient's outcome, and when feasible, the newborn infant's outcome.
- Ideally, perform resuscitative delivery by 5 minutes, depending on local resources.
- In pregnancy, difficult airway is common and is managed (eg, endotracheal intubation or supraglottic airway) by the most experienced professional.

Etiologies of Cardiac Arrest

A Anesthetic complications
B Bleeding
C Cardiovascular
D Drugs
E Embolic (amniotic fluid or pulmonary embolism)
F Fever
G General causes (H's and T's)
H Hypertension (eg, preeclampsia)

Adult Post–Cardiac Arrest Care Algorithm



Initial Stabilization After ROSC

Resuscitation is ongoing during the post-ROSC phase, and many of these activities can occur concurrently.

Manage airway: Assess and consider placement or exchange of an advanced airway device (usually endotracheal tube or supraglottic device). Confirm correct placement of an advanced airway. This generally includes the use of waveform capnography or capnometry.

Manage oxygenation and ventilation: Titrate FIO_2 for SpO_2 90%-98% (or PaO_2 60-105 mm Hg). Adjust minute ventilation to target PCO_2 35-45 mm Hg in the absence of severe acidemia.

Manage hemodynamics: Initiate or adjust vasopressors and/or fluid resuscitation as necessary for goal MAP ≥65 mm Hg.

Early diagnostic testing: Obtain 12-lead ECG to assess for ischemia or arrhythmia. Consider CT head, chest, abdomen, and/or pelvis to determine cause of arrest or assess for injuries sustained during resuscitation. Point-of-care ultrasound or echocardiography may be reasonable to identify clinically significant diagnoses requiring intervention.

Continued Management

Treat arrest etiologies and complications.

Consider emergency cardiac intervention:

- Persistent ST-segment elevation present
- Cardiogenic shock
- Recurrent or refractory ventricular arrhythmias
- Severe myocardial ischemia

Temperature control: If patient is not following commands off sedation and neuromuscular blockade or is unable to assess, initiate a deliberate strategy of temperature control with goal 32 °C-37.5 °C as soon as possible.

Evaluate for seizure: Evaluate for clinical seizure and obtain EEG to evaluate for seizure in patients not following commands.

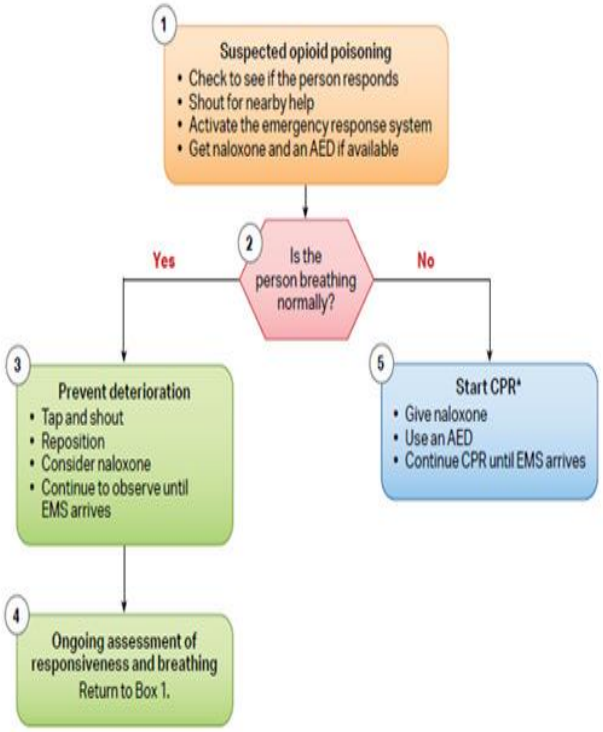
Prognostication: Multimodal approach with delayed impressions (≥72 hours from ROSC or achieving normothermia).

Ongoing critical care includes the following:

- Target PaO_2 60-105 mm Hg, PCO_2 35-45 mm Hg (unless severe acidemia); avoid hypoglycemia (glucose <70 mg/dL) and hyperglycemia (glucose >180 mg/dL); target MAP ≥65 mm Hg.
- Consider antibiotics.

A

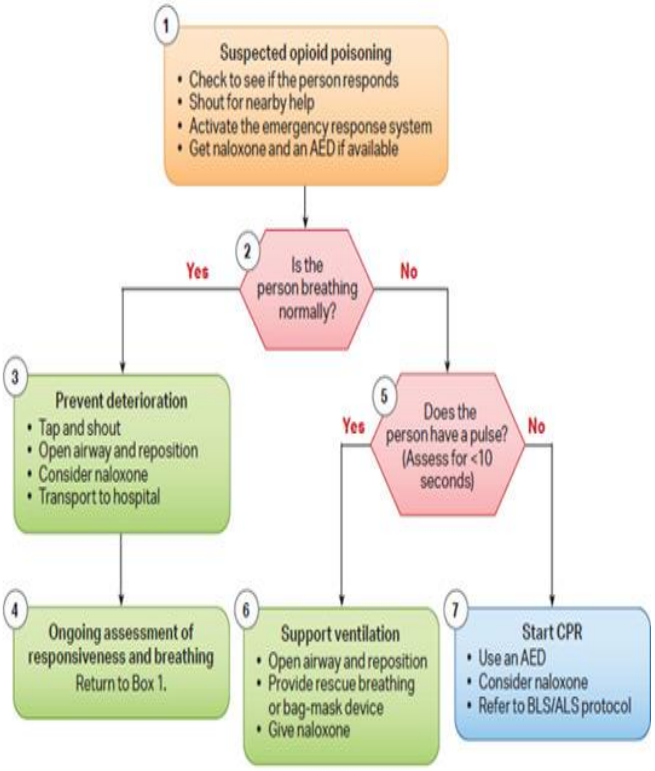
Opioid-Associated Emergency for Lay Responders Algorithm



*Responders should perform compressions and rescue breaths for opioid-associated emergencies if they are trained. Responders without formal training should perform Hands-Only CPR.

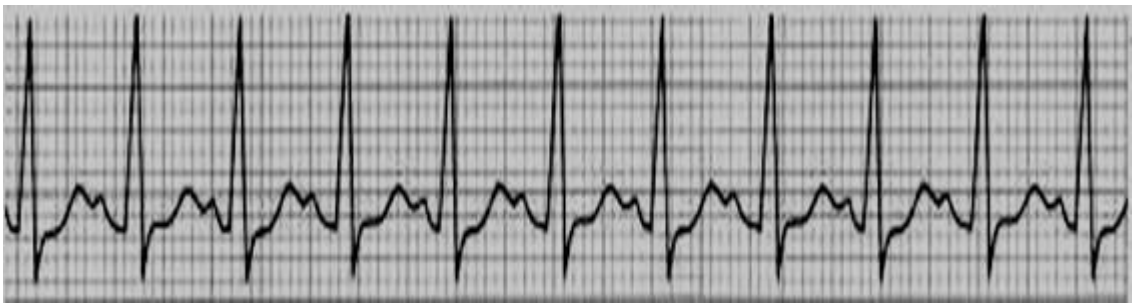
B

Opioid-Associated Emergency for Healthcare Providers Algorithm



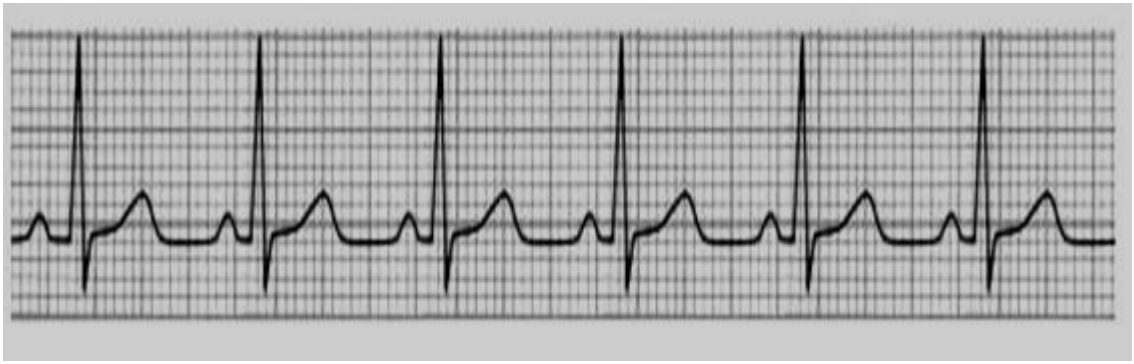
ECG REVEIW

1



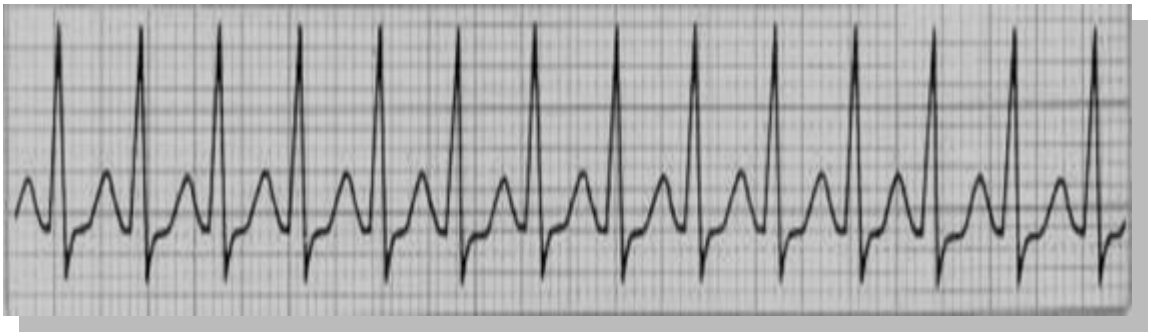
Rhythm **SINUS TACH**

2

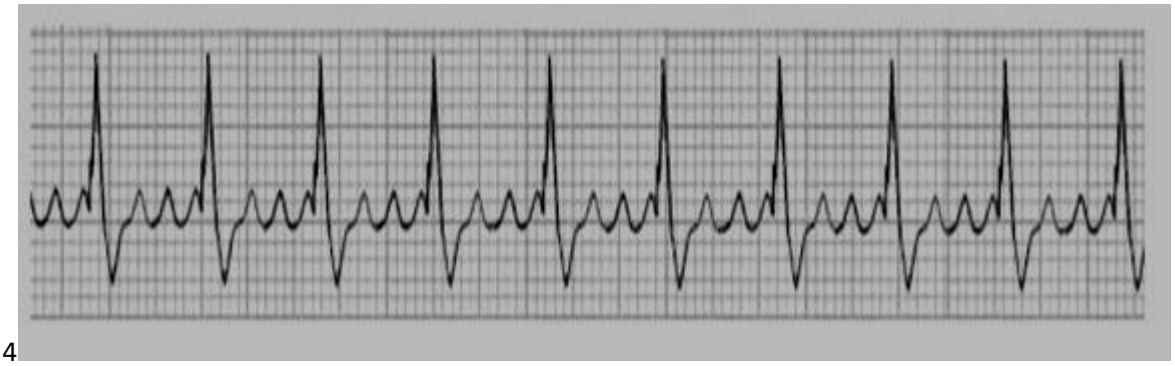


a. Rhythm Sinus Rhythm

3

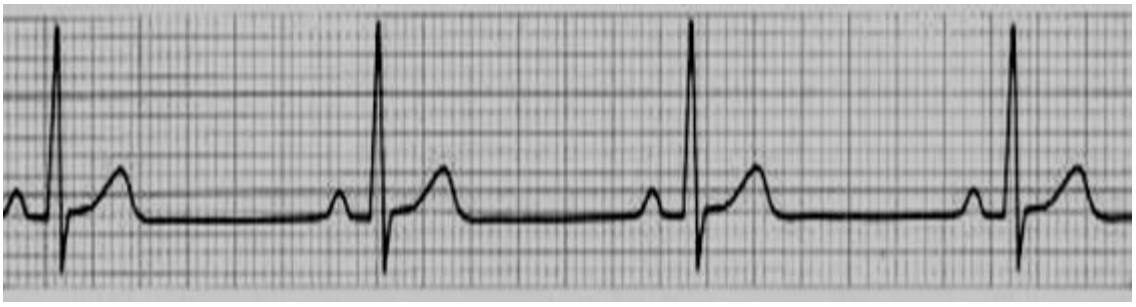


Rhythm <u>SVT</u>

4  **a.**

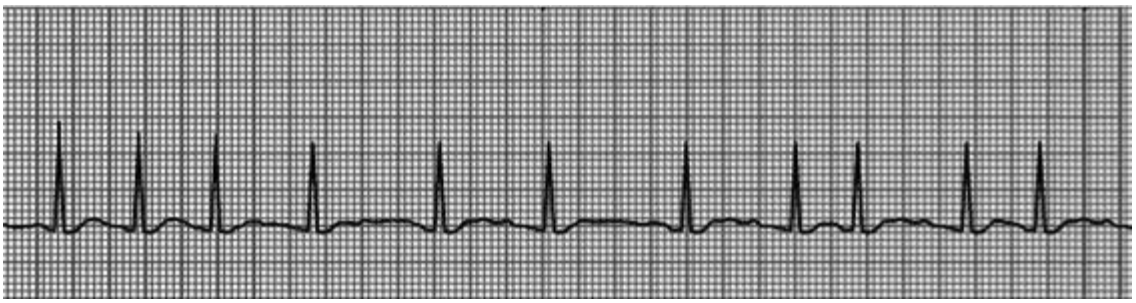
Rhythm : Atrial Flutter

5



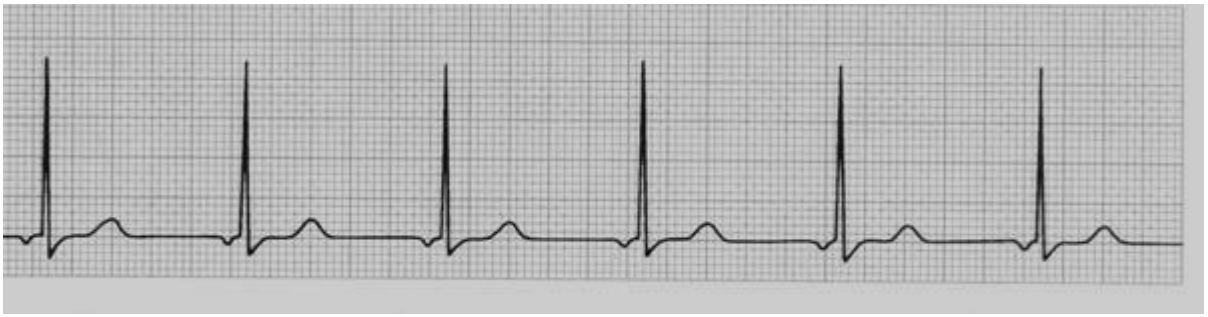
a. Rhythm: Sinus Brady

6



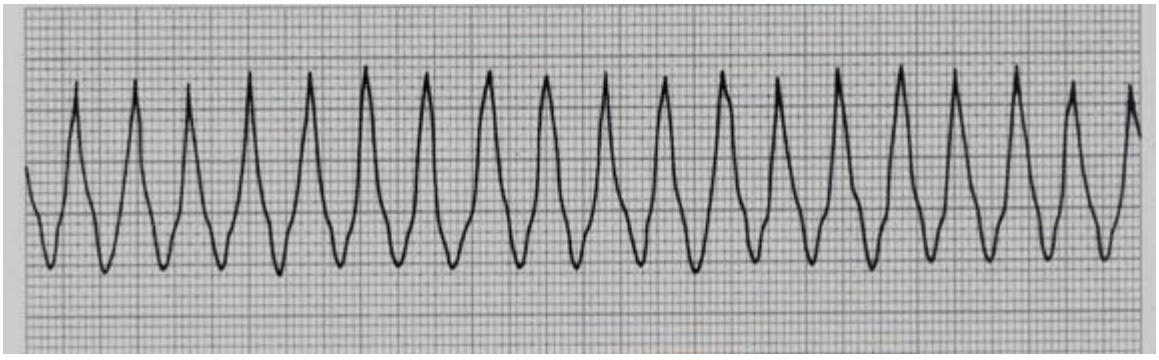
Rhythm : Atrial Fibrillation (No regular Ps, variable rate and fibrillatory baseline)

7



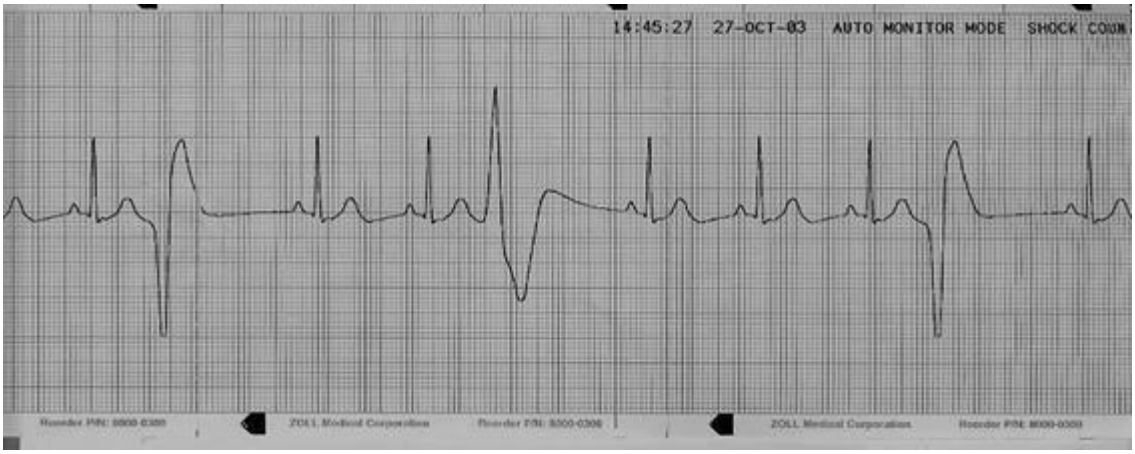
Rhythm : Junctional Rhythm.~ 60 bpm

8



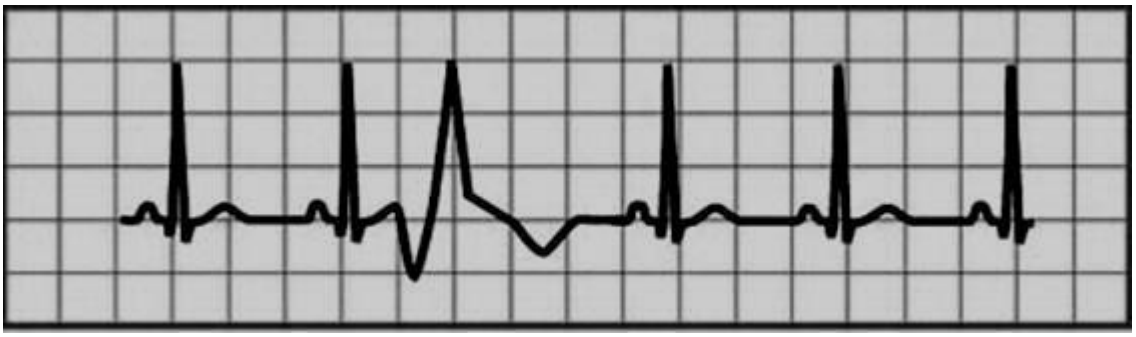
Rhythm : Monomorphic V-Tach

9



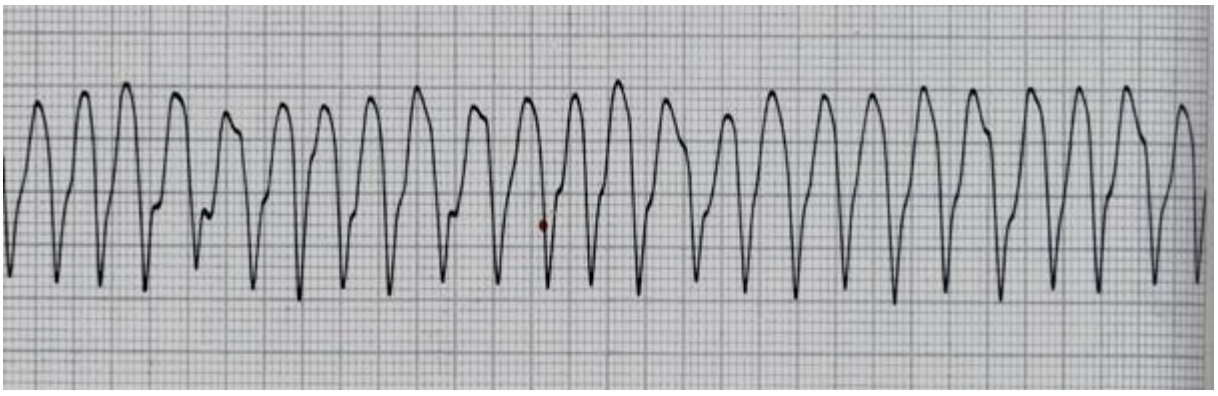
Rhythm : Sinus Rhythm W/ multifocal PVC's

10



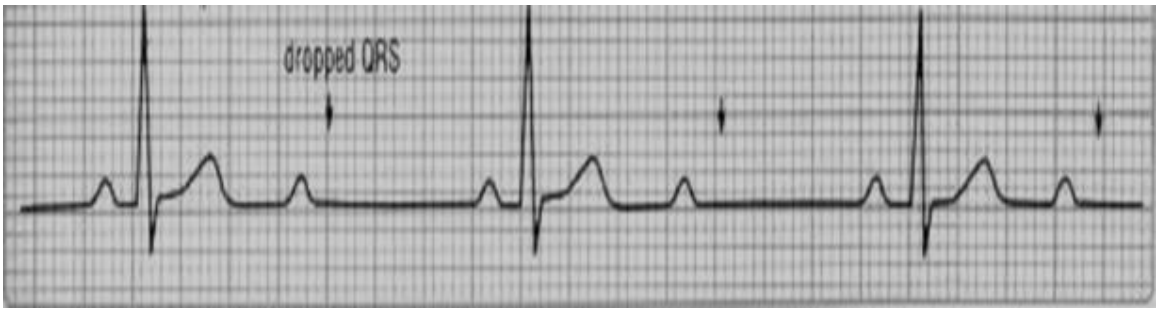
Rhythm: Sinus Rhythm W/ PVC

11



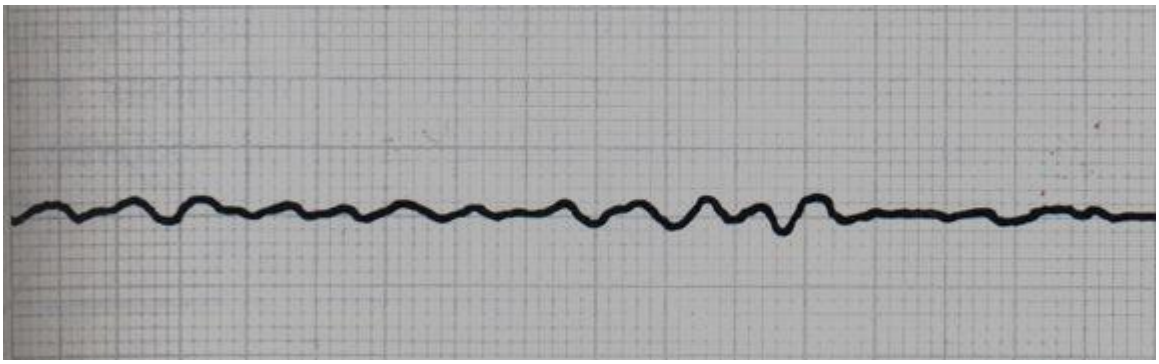
Rhythm : Polymorphic V-Tach (Probably normal QT)

12



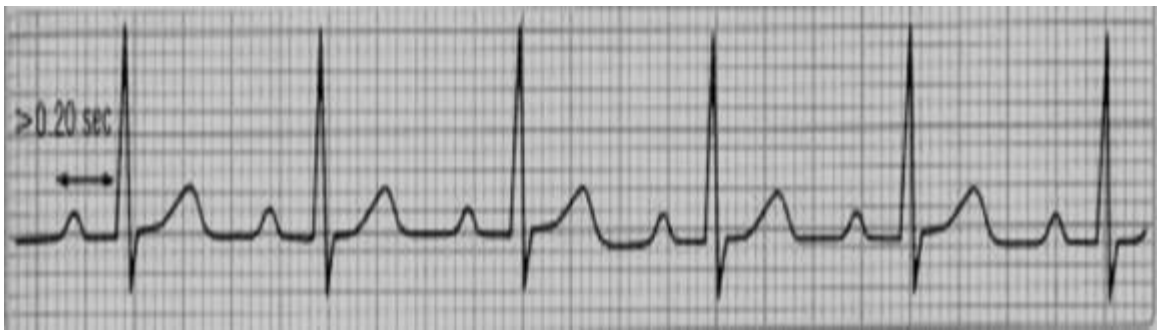
a. Rhythm: 2nd Degree type II

13



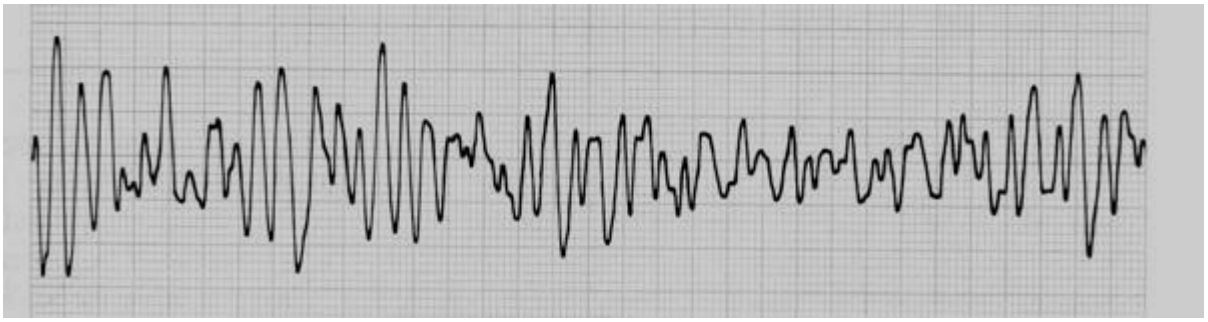
Rhythm : Fine V-Fib

14



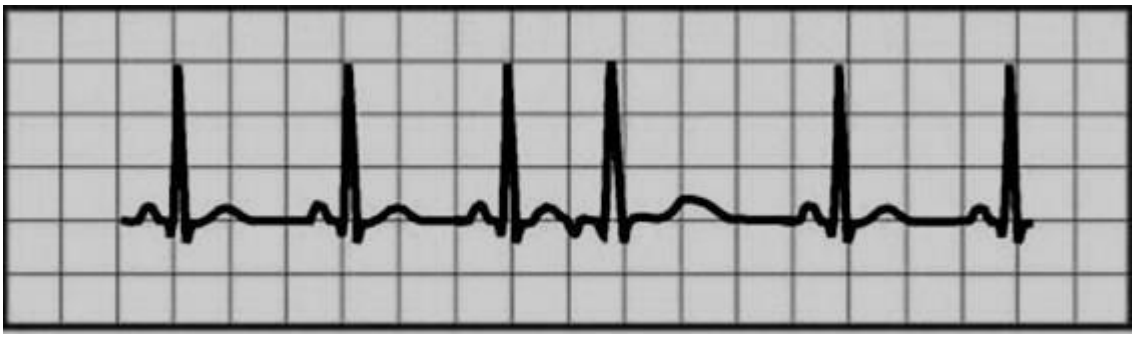
a. Rhythm : 1 Degree AVB

15



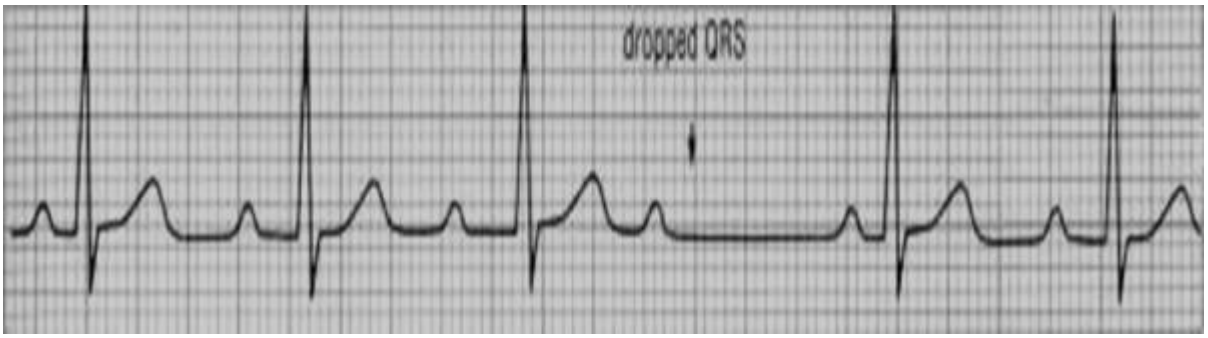
Rhythm: Coarse V-Fib

16



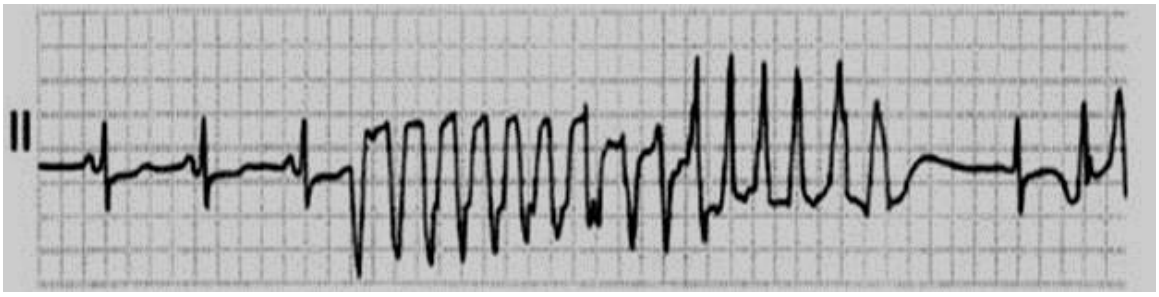
Rhythm : Sinus Rhythm W/PAC

17



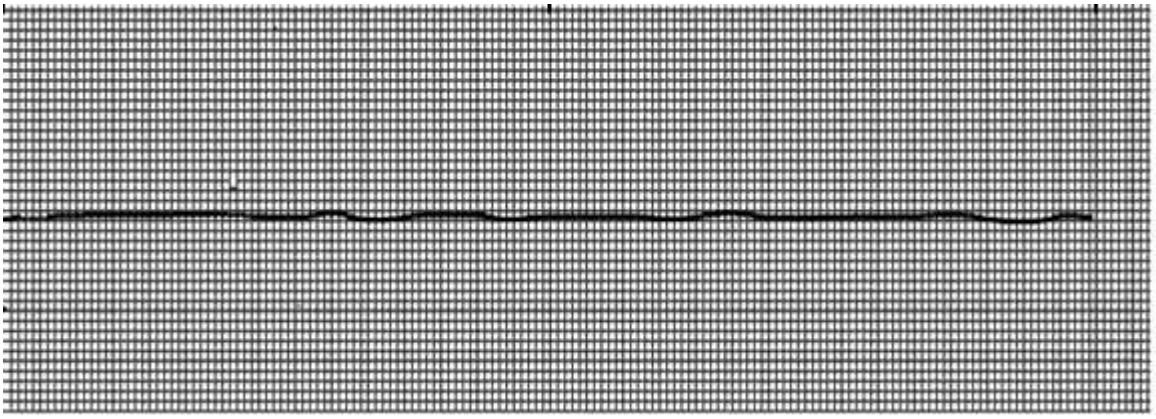
Rhythm: 2nd Degree type I

18



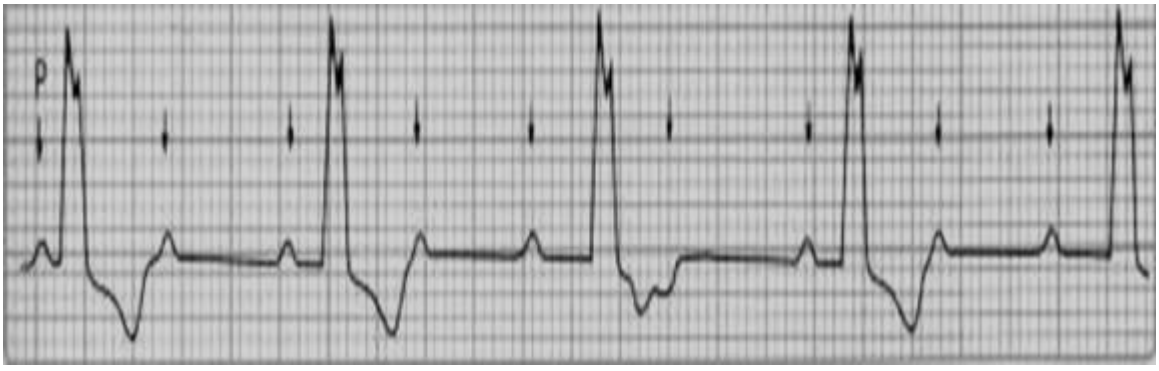
Rhythm: Polymorphic V-Tach / Torsades de Points

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

Recommendation-Specific Supportive Text

1.

Regardless of the underlying QT interval, all forms of **polymorphic VT are considered hemodynamically and electrically unstable**. Episodes of polymorphic VT may repeatedly recur and remit spontaneously, become sustained, or degenerate to VF. When the QRS complex of a VT is of uniform morphology, electric cardioversion synchronized to the QRS complex minimizes the risk of provoking VF during the vulnerable period of the cardiac cycle (T wave). In contrast, polymorphic VT cannot be synchronized reliably because of the differing characteristics of each QRS complex and requires high-energy (maximum manufacturer's setting) unsynchronized shock. Assessment of a patient's mental status is important when the appropriateness of sedation is considered before defibrillation. While effective in terminating polymorphic VT, defibrillation may not prevent its recurrence, for which pharmacological therapies are often required and the primary focus of subsequent recommendations.

2.

Torsades de pointes typically presents in a recurring pattern of self-terminating, hemodynamically unstable polymorphic VT in context of a known or suspected long QT abnormality, often with an associated bradycardia. Termination of torsades by defibrillation may not prevent its recurrence, which requires additional pharmacological interventions. In small case series, IV magnesium has been effective in suppressing and preventing recurrences.[10–13] Magnesium is believed to suppress fluctuations in the myocardial action potential that can trigger torsades.[14] Correcting electrolyte abnormalities, particularly hypokalemia, is also advisable.

3.

Polymorphic VT that is clearly not associated with QT prolongation, is most often triggered by acute myocardial ischemia or infarction and often rapidly degenerates into VF.[3,5] Polymorphic VT may be difficult to differentiate from VF initially, but, as with other ventricular arrhythmias (VT and VF), is terminated with immediate defibrillation. However, termination of polymorphic VT with defibrillation may not prevent its recurrence, requiring additional pharmacological measures. No RCTs have been performed to determine the optimal pharmacological management of polymorphic VT. Treatment of causative myocardial ischemia (eg, β-adrenergic blockers or emergent coronary intervention) as well as lidocaine and amiodarone,[16–22] in concert with defibrillation, may be synergetic when the arrhythmia is sustained. β-Adrenergic blockers have also been shown to reduce the incidence of ventricular arrhythmias in acute coronary syndromes.[23,24]

Expert consultation is advisable when alternative causes of polymorphic VT are suspected, for which β-adrenergic blockers and antiarrhythmics may also have efficacy.[6,25] Since the 2018 AHA Focused Update on Advanced Cardiovascular Life Support Use of Antiarrhythmic Drugs During and Immediately After Cardiac Arrest,[26] no new evidence has been identified.

4.

In the absence of long QT, magnesium has not been shown to be effective in the treatment of polymorphic VT[10] or other ventricular tachyarrhythmias.[12] A single case series with 5 patients showed no benefit of magnesium administration for polymorphic VT with normal QT. Since the 2018 AHA focused update on antiarrhythmic drugs during and immediately after cardiac arrest no new evidence has been identified.

For In formation:

Part 9: Adult Advanced Life Support: 2025 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care | Circulation

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

Key Words:

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