Course Overview
This Study Guide is an extensive outline of content that is taught in the American Heart Association Advanced Life Support (ACLS) Course. It is intended to summarize important content, but since all ACLS content cannot possibly be absorbed in a class given every two years, it is expected that the student will have the 2005 Updated AHA ECC Handbook readily available for review and as a reference. The student is also required to have the AHA ACLS Textbook available for reference and study for more in depth content.

Agenda
Welcome, Introduction, Overview
Lethal Rhythms
Rhythm Practice
Primary Survey Approach to ECC
Secondary Survey Approach to ECC
ACLS Algorithms
Skills Stations/ Mega Code Practice
Mega Code Skills Evaluation
Written Evaluation

Evidence Based Updates
Approximately every 5 years the AHA updates the guidelines for CPR and Emergency Cardiovascular Care. These updates are necessary to ensure that all AHA courses contain the best information and recommendations that can be supported by current scientific evidence. Evidence based guidelines were developed, documented, debated and then evaluated by scientific experts from inside and outside the United States and outside the AHA. The guidelines were then classified as to the strength of evidence that supports the recommendation.

Objectives
Upon the completion of this ACLS course the participant will be able to:

- Identify lethal rhythms
- Verbalize the primary survey approach to Emergency Cardiovascular Care (ECC)
- Verbalize the secondary survey approach to ECC
- Verbalize treatment algorithms for each of the following lethal rhythms:
  1. Pulseless Arrest
  2. Bradycardia
  3. Tachycardia
  4. Acute Coronary Syndrome
- Verbalize steps to determine ischemic stroke and subsequent treatment
- Stimulate confirmation of endotracheal tube placement
- Describe the difference between monophasic and biphasic defibrillation
- Describe the difference between defibrillation and cardioversion
**Normal Anatomy Review**

In order to understand Advanced Cardiovascular Life Support, it is essential to understand normal cardiac function. By understanding the normal electrical pathways in the heart, it will be easier to understand abnormal function. When blood enters the atria of the heart, an electrical impulse is sent out from the Sinoatrial (SA) node that conducts through the atria causing them to contract. The atrial contraction registers on an EKG strip as the P wave. This impulse then travels to the Atrioventricular (AV) node that in turn sends out an electrical impulse that travels through the Bundle of His, bundle branches, and into the Purkinje fibers of the ventricles causing them to contract. The ventricular contraction registers on the EKG strip as the QRS complex. Following ventricular contraction, the ventricles rest and repolarize that is registered on the EKG strip as the T wave. The atria repolarize also, but this coincides with the QRS complex and therefore cannot be observed on the EKG strip. Together a P wave, QRS complex, and T wave indicate a Sinus Rhythm.
In general, narrow QRS complexes originate at the junction of the heart or near the AV node. Wide QRS complexes indicate that a rhythm is originating below the Bundle of His or in the ventricles.

Typically, when looking at an EKG strip, a patient will be hooked up to a monitor or a printout will read the heart rate. However, this is not always the case. It is important to be able to determine a heart rate when the monitor or printout rate is not given. There are two ways that will be discussed to determine the heart rate.

1. The most common way to determine heart rate is to count the QRS complexes on a six second strip and then multiply by 10 to give a rate per minute.
2. The second way works especially well in patients without a 6 second strip and in tachycardia patients. In tachycardia patients, it can be time consuming to count the number of QRS complexes on a six second strip. A better method is to memorize the numbers 300 – 150 – 100 – 75 – 60 – 50, as is shown in the diagram above. One suggestion is to memorize them in triplets “300-150-100” “75-60-50.” It has a nice rhythm. Starting from a QRS complex that falls on a heavy line, count 300 on the next heavy line, then 150 on the next heavy line, and so on until the next QRS complex is reached. This will give a range as to the heart rate with accuracy enough to determine tachycardia or normal rhythm. For bradycardia, counting the QRS complexes on a six second strip will be faster.
### Cardiac Arrhythmias

#### Pulseless Rhythms

**Ventricular Fibrillation**

Ventricular Fibrillation (V-Fib or VF) is the most common rhythm that occurs immediately after cardiac arrest. In this rhythm, the ventricles quiver and are unable to uniformly contract to pump blood. It is for this reason that early defibrillation is so imperative. A victim’s chance of survival diminishes rapidly over time once the heart goes into V-Fib, therefore, each minute counts when initiating defibrillation.

\[ \text{V-Fib} = \text{Defibrillation} \]

Defibrillation stops the heart, like rebooting a computer, and allows it to restart with a corrected rhythm (hopefully).

There are two types of VF, fine and coarse VF. Coarse VF usually occurs immediately after a cardiac arrest and has a better prognosis with defibrillation. Fine VF, in which the waves flatten and nearly look like Asystole, often develops after more prolonged cardiac arrest and is much more difficult to correct. Caution: Sometimes artifact can look like VF, but we know to always check our patient.

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**Ventricular Tachycardia**

1. Stable vs. Unstable
2. Pulse vs. No pulse

Since this section is about pulseless rhythms, we are looking at Ventricular Tachycardia (VT) without a pulse. Ventricular Tachycardia will be discussed in more detail later. When a VT is present and the victim has no pulse, the treatment is the same as with VF. High dose shocks for defibrillation will give the best chance for converting the patient out of pulseless VT.

**Polymorphic VT is also treated like VT.**
Pulseless Electrical Activity

Pulseless Electrical Activity (PEA) occurs when the heart is beating and has a rhythm, it can be any rhythm, but the patient does not have a pulse. *Always treat the patient, not the rhythm strip.* The number one question in this situation is, “Why?”

- **P** = Problem or Possible correctable causes*
- **E** = Epinephrine 1 mg 1:10,000*
- **A** = Atropine 1 mg (if the PEA is slow, which most times it is)

The possible causes are referred to as “H’s & T’s” and are the following:

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

In order to treat pulseless rhythms, bradycardias, and tachycardias, identification of the possible underlying causes is essential. If a cause is not identified, all of the drugs in the world will not cure the problem. For example, if a patient is hypovolemic, unless he or she gets more fluids, it will be impossible to correct the problem.

Asystole*

Asystole is when there is no detectable cardiac activity on EKG. It may occur immediately after cardiac arrest or may follow VF or PEA. Asystole may also follow a third degree heart block. *Treatment of Asystole is the same as PEA.* The American Heart Association recommends that if a patient is in sustained Asystole for 15 minutes, it is reasonable to call the code, but involve the family in the decision if they are available.
**Bradycardia**

Bradycardia occurs when the heart is beating too slowly—less that 60 beats per minute. If symptomatic, provide oxygen, give Atropine 0.5mg, and call for the transcutaneous pacemaker.

**Sinus Bradycardia***

In Sinus Bradycardia, the SA node fires at a rate slower than normal for a person’s age. Athletes may have heart rates less than 60 due to their physical conditioning. Obviously, they would not need treatment. Some patients may have heart rates less than 60 and be asymptomatic. However, if a patient with a heart rate less than 60 has signs of poor perfusion; begin treatment with oxygen and Atropine 0.5 mg*

![EKG of Sinus Bradycardia](image1)

**First-Degree AV Block—All P waves conducted but delayed**

First-Degree AV block = prolonged PR interval (> 0.20 seconds or 5 small boxes on the EKG strip)

In First-Degree AV Block, all of the components of the EKG strip are normal except the PR interval. What happens in this situation is that the impulse from the SA node is delayed at the AV node. All impulses are, however, conducted following the delay.

![EKG of First-Degree AV Block](image2)

**Second-Degree AV Block Type I, (Mobitz I, Wenckebach)—Some P waves conducted, others blocked**

Second-Degree AV Block Type I (Mobitz I, or Wenckebach) = progressive lengthening of the PR interval with dropped QRS complexes.

The delay in Second-Degree AV Block Type I occurs at the AV node. The delay produces progressively lengthening PR intervals and then there will be a P wave that is not followed by a QRS complex. Following this event, the cycle starts over again with progressively lengthening PR intervals followed by a dropped QRS.

![EKG of Second-Degree AV Block Type I](image3)
Each repeating Wenckebach series has a consistent P:QRS ratio like 3:2, 4:3, 5:4, etc. (one less QRS than P’s in the series).

AV block location differentiates Second-Degree AV Type I blocks from Type II blocks.

**Second-Degree AV Block Type II (Mobitz II)—Some P waves conducted, others blocked**
Second-Degree AV Block Type II = PR interval stays the same, but there are dropped QRS complexes.

The delay in Second-Degree AV Blocks occurs **below** the AV node at the Bundle of His or bundle branches. They usually produce a series of cycles consisting of one normal P-QRS-T cycle preceded by a series of paced P waves that fail to conduct through the AV node resulting in no QRS. Each repeating Mobitz series has a consistent P:QRS ratio, like 3:1, 4:1, 5:1, etc. This is a much more serious rhythm than Wenckebach and transcutaneous pacing is usually recommended.

**Third-Degree or Complete AV Block—No P waves conducted**
Third-Degree or Complete AV Block = no communication in the heart between SA and AV nodes.

In Third-Degree or Complete AV block, the impulse originating in the SA node is completely blocked. This block may occur at the AV node, Bundle of His or bundle branches. In response to this situation, the heart may develop a secondary pacemaker (either junctional or ventricular) in order to stimulate the ventricles to contract. The location of this “escape pacemaker” will determine if the QRS complexes are wide or narrow. A junctional (narrow QRS complex) escape pacemaker rhythm may possibly be stable with a ventricular rate of more than 40 bpm. However, a ventricular (wide QRS complex) escape pacemaker rhythm is usually unstable with a heart rate of less than 40 bpm.

Often heart blocks occur due to cardiac damage following a MI. In high degree blocks (Type II Second- and Third-Degree AV Blocks), characterized by poor perfusion, transcutaneous pacing is recommended.

**Tachycardia**

There are 3 basic groups of tachycardias: Sinus Tachycardia, Supraventricular Tachycardia (including Atrial Tachycardia), and Ventricular Tachycardia. Fortunately, there is only one algorithm to treat all of them. The key factors are: STABLE vs. UNSTABLE and PULSE vs. NO PULSE. Additional factors are NARROW QRS vs. WIDE QRS and REGULAR vs. IRREGULAR.
**Sinus Tachycardia**

Sinus Tachycardia occurs when the SA node is firing at a rate that is faster than normal for a person’s age. The rate is usually 101 to 180 bpm. All components of a normal EKG are present, P waves, QRS complexes, and T waves. Sinus Tachycardia generally starts and stops gradually. There is often a cause such as pain, fever, or agitation that can be identified and treated.*

![EKG of Sinus Tachycardia](image)

**Supraventricular Tachycardia**

Supraventricular Tachycardia (SVT) includes any rhythms that begin above the bundle branches. This includes rhythms that begin in the SA node, atrial tissue, or the AV junction. Since the rhythms arise from above the bundle branches, they are characterized by narrow QRS complexes. A Supraventricular Tachycardia is not the name of a specific arrhythmia. It is a term used to describe a category of regular arrhythmias that cannot be identified more accurately because they have indistinguishable P waves due to their very fast rate—usually greater than 150 bpm. The P waves are often indistinguishable because they run into the preceding T waves. The most common SVT rhythms are Atrial Tachycardia and Junctional Tachycardia, although Sinus Tachycardia and Atrial Flutter can sometimes also fit into this category with indistinguishable P waves.

![EKG of Supraventricular Tachycardia](image)

**Treatment Question #1** = Stable vs. Unstable
   - If Unstable, Cardiovert
   - If Stable, answer question #2

**Treatment Question #2** = Regular vs. Irregular Rhythm
   - Regular (STV or Junctional) = Vagal maneuvers & Adenosine
   - Irregular (A-Fib, A-flutter, Multifocal A-Tach) = Calcium Channel Blockers or Beta Blockers
     - A = A-Fib, A-flutter
     - B = Beta Blockers
     - C = Calcium Channel Blockers (usually used 1st to slow the rate)
Atrial Tachycardia
The SA node and AV nodes are the primary pacemakers of the heart. However, there are other “automaticity foci” (sometimes called “ectopic” foci) that are potential pacemakers capable of taking over the pacemaker function in emergency situations. In Atrial Tachycardia, a very irritable automaticity focus may begin firing leading to a very rapid heart rate. This often begins suddenly. A rhythm that starts suddenly is termed “Paroxysmal”. Therefore, when an Atrial Tachycardia arises suddenly from a very irritable automaticity focus, it is termed Paroxysmal Supraventricular Tachycardia (PSVT). The Atrial Tachycardia may be termed “ectopic” or “multifocal”, arising from one or more automaticity foci. Multifocal Atrial Tachycardia is a chaotic and irregular rhythm due to multiple foci, each with their own rates, stimulating the atria.

Junctional Tachycardia
In Junctional Tachycardia, the AV junction becomes irritable and begins firing rapidly leading to a very rapid heart rate. If P waves are present (which they would not be in SVT), they would be inverted. The reason that P waves are inverted in junctional rhythms is because the impulse is being conducted backwards through the atria. This is more properly described as the atria being depolarized via retrograde conduction.

Atrial Fibrillation
In Atrial Fibrillation (A-Fib or AF), the atria quiver. This causes blood to pool in the heart where a blood clot can form. This is very critical in treatment of AF. The AF rhythm can be very fast when a significant number of the 400-600 impulses per minute trigger the AV node. Since the impulses being fired in AF are not at a regular rate, the rate of AF is usually irregular and can vary significantly in the ventricular rate generated. Calcium channel blockers are used to decrease the automaticity and slow the heart.
**Atrial Flutter**
In Atrial Flutter, the impulse circles around a large area of atrial tissue creating multiple P waves. If the AV node blocks the rapid impulses coming to it, and fires at a regular rate, the resulting rhythm will be regular. If the AV node blocks the rapid impulses coming to it at an irregular rate, the rhythm will be irregular.

![ECG waveform for Atrial Flutter](image)

**Ventricular Tachycardia***
Ventricular Tachycardia (VT) occurs when an irritable automaticity focus in either ventricle begins firing. This ventricular focus fires at a tachycardia rate and overrides the higher pacemaker sites and takes over control of the heart. It is basically a run of Premature Ventricular Complexes (PVCs). In PVCs or in VT, the ventricles fire prematurely and in an abnormal manner. Because the rhythm is originating in the ventricles, the QRS complex is wide.

Treatment Question #1 = Stable vs. Unstable
- If Stable, Cardiovert
- If Unstable, answer question #2

Treatment Question #2 = Pulse vs. No Pulse
- If Stable or Unstable with a pulse = Amiodarone
- If No Pulse = Defibrillate

**Monomorphic Ventricular Tachycardia**
In Monomorphic VT, the QRS complexes are of the same shape and amplitude. In ACLS, we want to know if the patient is stable or unstable. If unstable, we want to know if there is a pulse or no pulse. If NO pulse, we treat Monomorphic VT the same as VF.

![ECG waveform for Monomorphic Ventricular Tachycardia](image)
**Polymorphic Ventricular Tachycardia**
In Polymorphic VT, the QRS complexes are of different shape and amplitude. In ACLS we treat Polymorphic VT the same as VF = Defibrillation

![Polymorphic Tachycardia = irregular size QRS](image)

**Torsades de Pointes**
In Torsades de Pointes VT, the QRS complexes are of different shape and amplitude. The name means “twisting of points”, and in fact, what differentiates this rhythm from others is that it looks like a twisting party streamer with upward-pointing and then downward-pointing QRS complexes in an alternating pattern. This rhythm can be caused by an electrolyte imbalance. ACLS recommendations include treatment with Magnesium.

![Torsades de Pointes](image)
ST-Elevation MI (STEMI)

A person who is having a MI will typically exhibit certain characteristics on their ECG. If necrosis or damage has occurred in the heart, a Q wave will be present. Once the damage has occurred, The Q wave will always be present. Therefore, based upon the presence of a Q wave, it is impossible to determine if the MI is acute or occurred in the past. However, the ST-Segment may become elevated during an acute MI and is usually one of the earliest signs of a MI. Therefore, evaluating the ST-Segment is a key part of assessment of a person exhibiting signs of a MI. In order to determine if there is a ST-Elevation, the isoelectric line must be evaluated. The isoelectric line is an imaginary line that goes from the beginning of to the end of the QRS complex. If this line is level, the isoelectric line is normal. If, however, the ST-Segment exits the complex prior to returning to the isoelectric line, the ST-Segment is elevated. This indicates acute injury occurring in the heart. Sometimes an ST-Segment depression may occur as well. Following the injury, the ST-Segment will return to normal. However, if the event caused significant ischemia, a Q wave will develop. If the injury resulting from a MI is milder, the ST-Segment may not be elevated, even though a MI has occurred. This is called a Non-ST-Elevation MI (NSTEMI). One other factor that can indicate ischemia during an acute MI is the inversion of the T wave.

Q = Infarction
ST-Elevation (or depression) = Acute injury
T wave inversion = Ischemia

Adult Chain of Survival

Early Access ♥ Early CPR ♥ Early Defibrillation ♥ Early Advanced Care
**BLS Review**

*(Primary Survey Approach to ECC)*

Performing high quality CPR is the most critical component for successful resuscitation of a patient in cardiac arrest. The goal of intervention for a patient in respiratory or cardiac arrest is to restore effective oxygenation, ventilation and circulation.

- **A** = Airway
- **B** = Breathing
- **C** = Circulation
- **D** = Defibrillation

**Rescue Techniques – ABC and D**

**Unresponsiveness:** After determining that the scene is safe, check to see if victim is responsive. If the adult victim is unresponsive, send someone to activate the emergency response system (EMS) – phone 911 and get the AED.

**IF IN THE HOSPITAL, CALL THE CODE!**

“Phone FIRST” versus “Phone FAST” if rescuer is ALONE

If **alone** the rescuer phones 911 immediately after discovering an unresponsive adult victim and then returns to begin CPR. The goal of the “phone first” approach is fast arrival of EMS professionals able to attach and use a defibrillator.

EXCEPTION: if asphyxial arrest likely, call after 5 cycles of CPR (2 minutes).

**Airway:** Open the Airway.

- The **head tilt-chin lift** is the best way to open unresponsive victim’s airway when you do NOT suspect cervical spine injury.
- The **jaw-thrust** with cervical spine immobilization is used for opening airway without tilting the head or moving the neck if a neck injury is suspected (this includes drowning victims)—after two unsuccessful attempts, use **head tilt-chin lift**.

**Breathing:** Check for Breathing.

- Look, Listen, and Feel for breathing. Check for breathing by looking to see chest rise and fall when the victim breathes, listening and feeling for airflow through victim’s nose and mouth.
- Next, pinch the victim’s nose closed, or for an infant place your mouth over the infant’s nose and mouth, and give 1 breath (blow for 1 second), watching for the chest to rise. If the chest does not rise, make a second attempt to open the airway with a head tilt-chin lift. Then give 1 breath (blow for 1 second) and watch for the chest to rise. Of course, if using a mask barrier device or bag mask ventilation, there is no need to pinch the nose. Only provide enough air to see the chest rise and fall. If using a bag mask, there is no need to compress the bag completely.
- Do not over-inflate the lungs. The positive pressure in the chest that is created by rescue breaths will decrease venous return to the heart. This limits the refilling of the heart, so it will reduce cardiac output created by subsequent chest compressions.
- Some victims may continue to demonstrate agonal or gasping breaths for several minutes after a cardiac arrest, but these breaths are too slow or too shallow and will not maintain oxygenation. Perform rescue breathing.

**Circulation:** Check for a Pulse.


- The best location for performing a pulse check for an adult is the carotid artery of the neck.
You should start cycles of chest compressions and breaths when the victim is unresponsive, is not breathing adequately, and does not have a pulse.

The compression to ventilation ratio is 30:2

Proper compression technique requires the right rate and depth of compressions, as well as full chest recoil. Take your weight off your hands and allow the chest to come back to its normal position. Full chest recoil maximizes the return of blood to the heart after each compression.

The rate of performing chest compressions for a victim of any age (adult, child and infant) is at a rate of 100 compressions per minute.

Compressions on the adult: two hands are placed in the center of the chest between the nipples on the lower half of the sternum.

Rotation of two-rescuer CPR is every 2 minutes (5 cycles of 30:2).

Minimizing interruptions in chest compressions will increase the victim’s chance of survival.

Defibrillation: Attach the Automated External Defibrillator (AED).

The probability of successful defibrillation diminishes rapidly over time. Immediate CPR and defibrillation within no more than 3 to 5 minutes give an adult in sudden cardiac arrest the best chance of survival.

The AED is used on an adult victim and may be used on a child victim over the age of 1 yr.

Only use adult AED pads when performing defibrillation on an adult.

Child victim: Rescuer should use pediatric pads when available for children ages 1 to 8 yr. If not available use adult pads making sure that they do not touch each other.

Adult or Child victim: Place one pad on the victim’s upper right chest just below the collar bone and right of the sternum and the other pad on the left side and below the nipple, being careful that the pads do not touch.

The four common steps of operating an AED are:
1. Power on the AED.
2. Attach pads to the victim.
3. Clear the victim and allow the AED to analyze the rhythm.
4. Clear the victim and deliver shock, if advised.

Make sure to clear the victim before shocking, so that you and others helping do not get shocked.

If no shock is advised, leave the AED pads on the victim and continue CPR, beginning with compressions.

CPR alone may not save the life of a sudden cardiac arrest victim. Early defibrillation is needed.

Foreign Body Airway Obstruction - Choking

The best way to relieve severe choking in a responsive adult or child: Perform abdominal thrusts.

When a choking victim becomes unresponsive (adult, child, or infant): Begin CPR. When you open the airway, look for and remove the object (if seen) before giving rescue breaths.
Analyze the Problem and Treat the Rhythm
(Secondary Survey Approach to ECC)

Airway
Look for any signs of airway obstruction. Make sure that the airway is adequate and protected. Secure the airway with:
   - Nasopharyngeal Airway – semi-conscious
   - Oral pharyngeal Airway -- unconscious
   - Laryngeal Mask Airway (LMA) – recommended if provider is inexperienced with ET tube
   - Combitube – if provider inexperienced with ET tube
The ET tube is placed using a laryngoscope, looking for the triangular vocal cords, and placing the ET tube through them.
Cricoid pressure may be helpful during intubation. This is performed by pressing firmly (may require about 10 lbs of pressure) on the cricoid cartilage.
Attempts should be limited to 30 seconds
Insertion of an advanced airway may be deferred until several minutes into the attempted resuscitation, since airway insertion requires an interruption in chest compression for many seconds.

Breathing (oxygen)
   - Oxygen = #1 drug – give oxygen as soon as it is available
   - Provide oxygen: Room air has 21% oxygen.
   - Nasal Cannula (1 to 5 liters) = increases oxygen by 4% for each liter
   - Face Mask without reservoirs = increases oxygen by 10% for each liter not recommended to give more than 40 – 60% without a reservoir
   - Face Mask with reservoir = ability to provide 100% oxygen
   - Always monitor a patient with pulse oximetry.
Endotracheal (E.T.) Tube
   - Confirm E.T. tube placement:
     1. Mist in the tube
     2. Auscultation of lungs for bilateral breath sounds
     3. Auscultation of the gastric area—no gurgling should be heard that would indicate intubation of the esophagus
   - Confirmation with CO2 Detector or Esophageal Detector (now one or the other is required for primary confirmation
   - Secure the airway with a commercial device.
   - Once an advanced airway is in place, there is no need to pause chest compressions for ventilations.
     - Provide 100 compressions per minute and 1 breath every 6 to 8 seconds which is equal to 8-10 ventilations per minute.*
   - Assess for pneumothorax. If necessary, decompress.

Circulation
   - Establish IV/IO access in order to: give fluids as necessary, give medications, flush drugs in with a fluid bolus. ET administration of drugs is not preferred
   - Treat heart rate and rhythm
   - Check blood pressure
   - Send blood samples to lab for T & C and analysis

Differential Diagnosis – H’s and T’s
“Thinking it Through” Unless the cause of an arrhythmia is correctly identified, it will be impossible to treat. A hypovolemic person in PEA will not be helped by all of the epinephrine in the world. H’s and T’s are essential to nearly every algorithm.
   - Hypovolemia – Give fluids
• Hypoxia – Give oxygen, check E.T. tube
• Hydrogen ion (acidosis) – Sodium bicarbonate
• Hypo-/Hyperkalemia – Potassium or sodium bicarbonate
• Hypoglycemia – Glucose
• Toxins – Drug overdose = Give Narcan
• Tamponade, cardiac – Pericardiocentesis
• Tension pneumothorax – Needle decompression
• Thrombosis (coronary or pulmonary) – ? surgeon
• Trauma – assess & treat

Disability
  Mental status, pupil response
  Glasgow Coma Scale
  Stroke screen as indicated

Other Emergency Considerations
  Expose extremities, examine skin
  Fingers (rectal, vaginal exam), Foley, flip (check back)
  Gastric tube
  History

Team Work

Each team member in resuscitation must be proficient in skills according to his or her scope of practice.*

There are several elements of team dynamics that will make any code run more smoothly. These elements include:
  Closed loop communication by the team leader
  Delivery of clear messages
  Having clear roles and responsibilities
  Knowing one’s limitations
  Knowledge sharing
  Constructive interventions
  Reevaluation and summarizing
  Mutual respect
Algorithms to Treat the Rhythm

Pulseless Arrest

Pulseless Arrest includes:
1. Ventricular Fibrillation and Pulseless Ventricular Tachycardia
2. Asystole and Pulseless Electrical Activity

V-Fib and Pulseless VT ARE shockable
Asystole and PEA ARE NOT shockable

IF SHOCKABLE (V-Fib and Pulseless VT):

Defibrillation can be performed using either monophasic or biphasic technology. Biphasic, the newer technology uses about ½ the energy of a monophasic shock.

Monophasic = maximum 360 J
Biphasic = maximum 150 J to 200 J (If unsure, use 200 J)

The first shock eliminates VF more than 85% of the time.

Steps for defibrillation:
1. When the AED or defibrillator arrives, turn it on
2. Select energy level
3. Position appropriate pads or electrodes (apply conductive paste if using paddles)
4. Analyze the rhythm (do not touch the victim during this phase) if the rhythm is V-Fib or Pulseless VT (or if the AED recommends a shock), prepare to shock
5. Prepare to shock by selecting the appropriate # of Joules and selecting defibrillate mode
6. Press the charge button—announce that you are doing this—continue CPR while charging
7. Clear: I’m clear (you are not touching the patient or bed), You’re clear—includes making sure that the oxygen is away from the patient, Everybody’s clear (no one is touching patient, or bed)
8. Press the shock button and wait for shock discharge

- Immediately following the shock, resume CPR starting with chest compressions.
- Perform CPR for 2 minutes (5 cycles of 30 compressions to 2 breaths)
- After 2 minutes of CPR, stop compressions just long enough to check the rhythm and check for a pulse
- If another shock is needed, prepare to shock and give a medication as close to the shock as possible
- Repeat this sequence until the rhythm is not shockable
Reasons for CPR immediately after the shock:

- If the first shock fails, CPR will circulate the blood and bring more oxygen to the heart, making a subsequent shock more likely to be successful.
- Even when a shock eliminates VF, it often takes several minutes for a normal heart rhythm to return and more time for the heart to create blood flow. Chest compressions can deliver oxygen and sources of energy to the heart, increasing the likelihood that the heart will be able to effectively pump blood after the shock.

Drug delivery should not interrupt CPR. Rescuers should prepare the next drug dose before it is time for the next rhythm check so that the drug can be administered as soon as possible after the rhythm check. The timing of the drug is less important than minimizing interruptions in chest compressions.

A drug may be administered
1. During the CPR
2. While the defibrillator is charging
3. Immediately after the shock

Medication Sequence:
- Epinephrine 1 mg IV/IO 1:10,000 every 3 to 5 minutes
- Amiodarone 300 mg IV/IO push followed by
- Amiodarone 150 mg IV/IO push in 3 to 5 minutes

Note: The 2005 Guidelines suggest giving one dose of Vasopressin 40 U as a substitute for either the first or second dose of Epinephrine. Also, Lidocaine may be given instead of Amiodarone, but only if Amiodarone is not available.

One additional consideration is regarding a VF cardiac arrest that has been present for several minutes prior to CPR. The heart has probably used up most of the available oxygen needed to contract effectively. The VF is therefore, fine VF and defibrillation is not typically successful. If it is successful, the heart is unlikely to pump blood effectively for several seconds or even minutes after defibrillation. A period of CPR before shock delivery will provide some blood flow to the heart, delivering some oxygen and sources of energy to the heart muscle. This will make a shock more likely to eliminate the VF and will make the heart more likely to resume an effective rhythm and effective pumping function after shock delivery.

IF NOT SHOCKABLE (Asystole and PEA):

P = Possible causes = H’s & T’s, hypovolemia is most common and often easiest to treat
E = Epinephrine 1 mg of 1:10,000 IV/IO every 3 to 5 minutes*
A = Atropine 1 mg every 3 to 5 minutes (for a slow PEA or Asystole)

Note: Asystole should not be called “flat line”. Flat line indicates a lead is off or the gain and sensitivity need to be adjusted.
If a patient is in sustained Asystole for 15 minutes, it may be reasonable to consult the family and consider calling the code.
Bradycardia

Atropine 0.5 mg IV repeated every 3 to 5 minutes up to a maximum of 3 mg is the drug of choice. Have the transcutaneous pacemaker ready.

Atropine 0.5 mg if alive (pulse), 1.0 mg if dead (no pulse)

Atropine will probably not work in high-degree heart blocks (type II second-degree block or third-degree AV block). They will almost certainly require transcutaneous pacing.

- Steps for transcutaneous pacing:
  1. Consider sedation
  2. Attach pacing electrodes to the patient as shown on package (AP position preferred)
  3. Turn pacer on
  4. Set the pacing rate—start at 60 bpm and then can adjust higher if needed
  5. Look for electrical capture on the strip (turn up mA dial until capture is achieved—widening QRS & broad T waves)
  6. Assess mechanical capture by assessing right arm or right femoral pulses
  7. Once capture is achieved, set pacing at about 2 mA higher than the threshold of initial capture. Epinephrine or dopamine infusion may be considered while awaiting pacer or if pacing is ineffective.

Tachycardia

#1 Question = STABLE vs. UNSTABLE (Unstable signs include altered mental status, ongoing chest pain, hypotension or other signs of shock)

STABLE = Medication
**Narrow QRS Regular Rhythm (SVT)**

1. Try Vagal maneuvers
2. Adenosine 6 mg - 12 mg - 12 mg RAPID IVP
   Note: A brief period of Asystole may follow the injection

**Narrow QRS Irregular Rhythm (A-Fib, A-flutter, etc) or does not convert with Adenosine**

Control the rate with Calcium Channel Blockers (Diltiazem) or Beta Blockers
A = A-Fib, A-flutter
B = Beta Blockers
C = Calcium Channel Blockers

If a person in A-fib does not convert with Calcium Channel Blockers, cardioversion may be necessary. Since the atria are not pumping effectively, blood clots may form in the atria due to blood pooling. If a person has been in A-fib for more than 48 hours, anticoagulant therapy is indicated for several days prior to cardioversion. Always seek expert consultation from the cardiologist.

**Wide QRS (VT with pulse)**

Amiodarone 150 mg IV over 10 minutes
May need synchronized cardioversion
Polymorphic VT -- defibrillate

**Wide QRS (torsades de pointes)**

Magnesium load with 1-2 g/IV over 5-60 minutes, then infusion
UNSTABLE (WITH PULSE) = SYNCHRONIZED CARDIOVERSION

Prepare for IMMEDIATE cardioversion. While preparing, you may try an appropriate medication (Adenosine or Amiodarone) if there is time. Also, sedate the patient if possible.

Use 100 J, 200 J, 300 J, 360 J monophasic, depending upon the acuity of the patient (or a clinically equivalent biphasic energy dose. Optimal biphasic doses have not yet been established with certainty. An initial dose of 100 J-200 J is a usual starting dose for A-Fib and has been shown to be 80%-85% effective. This information can be used to extrapolate biphasic cardioversion does to other tachycardias. A-Flutter and SVTs generally require lower doses. Initial energy of 50 J to 100 J (Monophasic) is often sufficient.

- Steps for cardioversion:
  1. Consider sedation
  2. Turn on defibrillator
  3. Attach monitor leads to patient
  4. Press “SYNC” mode button
  5. Look for markers on R waves indicating sync mode
  6. Select appropriate energy level
  7. Position appropriate pads or paddles
  8. Press the charge button—announce that you are doing this
  9. Clear: I’m clear, You’re clear—including making sure that the oxygen is away from the patient, Everybody’s clear
  10. Press the shock button and wait for shock discharge (this may take a few seconds while the machine looks for R waves and determines where to sync the shock)
  11. Analyze the rhythm again. If still in tachycardia, increase the joules and try again.

Note: Reset the sync mode after each synchronized cardioversion because most defibrillators default back to unsynchronized mode.

Hypothermia (Fortunately not a huge problem in Southern California)
- **Mild** = 34 to 36 degrees C
  - Treatment: Passive rewarming
  - Active external rewarming
- **Moderate** = 30 to 34 degrees C
  - Treatment: Passive rewarming
    - Active external rewarming of truncal areas only
- **Severe** = <30 degrees C
  - Treatment: Active internal rewarming

If NO pulse and NOT breathing:
- Start CPR
- Give 1 shock (same as defibrillation protocol)
- May give meds, but space at longer than standard intervals
- May repeat shock as core temperature rises
**Stroke**

Signs and symptoms of a stroke include:

- Sudden weakness or numbness of the face, arm, or leg on one side of the body
- Sudden dimness or loss of vision, particularly in one eye
- Loss of speech, or trouble talking or understanding speech
- Sudden, severe headache with no apparent cause
- Unexplained dizziness, unsteadiness or sudden falls, especial along with any of the previous symptoms

EMS must be notified immediately.

There is a **3 hour** window from onset of symptoms during which fibrinolytic therapy will be most effective.*

Within 3 hours, the following must be completed

- Support ABCs, give oxygen as needed
- Evaluate the prehospital assessments: Cincinnati Prehospital Stroke Scale or Los Angeles Prehospital Stroke Screen (LAPSS)
- Check blood sugar (severe hypo- or hyperglycemia can mimic a stroke)
- Establish time that the stroke occurred
- Transport to a stroke unit
- History, Physical, Neurological assessment
- Non-contract CT scan of the head – must be read by a radiologist*
- Monitor and treat BP (Systolic BP must be <185, Diastolic <110 to be eligible for fibrinolytic therapy)
- If all inclusion and exclusion criteria are acceptable, fibrinolytic therapy may be started
Acute Coronary Syndromes (ACS)
The primary symptom of an acute MI may be chest pain radiating to the jaw or left arm. However, various other milder symptoms may also signal varying degrees of coronary artery occlusion and ischemia resulting from atherosclerosis. Of particular concern are women, who tend to have more vague signs of a MI. This is further complicated when a woman has a history of diabetes. Diabetes can alter the pain perception due to neuropathy. Therefore, a diabetic woman can have ischemic symptoms that are overlooked because they are vague and not the characteristic chest pain.

Acute coronary syndromes can be divided into three groups
1. Unstable angina
2. Non ST-Segment Elevation MI (NSTEMI)
3. ST-Segment Elevation MI (STEMI)

Any of these may lead to sudden cardiac death.

Unstable Angina
In an artery that is partially occluded by a thrombus, there may be intermittent episodes of ischemia. The symptoms may be prolonged and may even occur at rest. These symptoms are better treated with aspirin, not fibrinolytic therapy, because fibrinolytic therapy, because fibrinolytics may cause the thrombus to be released and create full occlusion and subsequent necrosis.

Non ST-segment Elevation MI (NSTEMI)
A thrombus that intermittently occludes an artery may cause myocardial necrosis, producing a NSTEMI. This necrosis is usually in a smaller area and not as significant as necrosis that would cause ST-Segment Elevation. A Q wave may not develop following a NSTEMI.

ST-segment Elevation MI (STEMI)
When a thrombus occludes the coronary vessel for a prolonged period, the resulting necrosis usually produces a STEMI. A STEMI represents significant myocardial damage and usually results in a Q wave developing. This condition is best treated with heparin, fibrinolytic therapy (eg, Alteplase, Retepalce, Streptokinase, and Tenecteplase) and a trip to the cardiac cath lab.

Treatment
1. The first treatment consideration is to support the ABCs
2. If a person is unresponsive, prepare to defibrillate since VF is often the rhythm that follows a significant MI
3. If a person is stable, call in nurse MONA*
   M = Morphine (4) – Only if Nitro doesn’t relieve chest pain
   O = Oxygen (#1) – 4 liters per NC
   N = Nitroglycerin (#3) – 3 doses SL if NO Viagra in 24 hrs. & BP is OK
   A = Aspirin (#2) – may chew
   NOT in that order, however. See number above
4. Get a 12 lead ECG. Look for ST-Segment Elevation, Q wave or inverted T wave
5. Draw blood for cardiac markers
6. Monitor BP – Avoid hypotension with Nitroglycerin & Morphine, elevate hypertension that may exclude fibrinolytic therapy BP>185 systolic or > 110 diastolic
7. The goal is to start fibrinolytic therapy within 30 minutes (door-to-needle) of the MI, but it may be started up to 12 hours following the event
8. The goal is to perform percutaneous coronary intervention (PCI) within 90 minutes (door-to-balloon inflation), but this also may be considered up to 12 hours following the MI
**Post-Resuscitation Care**

Patients display a wide spectrum of responses to resuscitation. Following return of spontaneous circulation, patients may respond by becoming awake and alert with adequate spontaneous respirations and hemodynamic stability. Others will remain comatose with an unstable circulation and no spontaneous breathing. Many will require 24 to 48 hours of invasive hemodynamic monitoring for optimal management after resuscitation.

Your immediate goal is to provide cardio-respiratory support to optimize oxygenation and perfusion, particularly to the brain. This is accomplished by assessing and treating the primary and secondary ABCD surveys:

- **A**irway = Secure the airway and confirm tracheal tube with primary assessments and secondary assessment which must include a chest x-ray.
- **B**reathing = Administer 100% oxygen with mechanical ventilation and monitor with oxygen saturation levels and blood gas analyses. Mechanical ventilation often requires paralysis and sedation.
- **C**irculation = Administer normal saline IV and monitor urine output to reflect tissue perfusion. Insert nasogastric tube and initiate an infusion of an antiarrhythmic for secondary prophylaxis.
- **D**ifferential Diagnosis = Search for specific cause for the arrest. Review the chest x-ray, 12-lead ECG, history, and serum electrolytes.

Other Actions:
- Change IV lines placed without proper sterile technique.
- Replace deficient electrolytes
- Transport to higher level of care.

The following problems may develop:
- Hostile environment for the brain – control seizures that increase cerebral oxygen requirements. Elevate the head 30 degrees to decrease intracranial pressure.
- Hypotension – even mild hypotension can impair recovery of cerebral function.
- Recurrent VF/Pulseless VT – consider administration of an infusion of the antiarrhythmic used during resuscitation.
- Post-resuscitation of tachycardia – rapid SVTs that may develop in the immediate post-resuscitation period are best treated by leaving them alone.
- Post-resuscitation bradycardia – poor ventilation and oxygenation play a major role in post-resuscitation bradycardia.
- Post-resuscitation PVCs – improved oxygenation over time may eliminate the ectopic beats.

Post resuscitation care includes support of the myocardial function with anticipation that myocardial “stunning” may be present, requiring vasoactive support. A healthy brain is the primary goal of cerebral and cardiopulmonary resuscitation. This may be accomplished by allowing moderate hypothermia.

- Hypothermia—The 2005 Guidelines emphasize the importance of avoiding hyperthermia and the possible benefits of induced hypothermia (32°C to 34°C) for 12 to 24 hours for patients who remain comatose after resuscitation from cardiac arrest. Providers should monitor temperature and treat fever aggressively.
- Maintaining strict glucose control—Additional studies are needed to determine the precise blood glucose concentration that requires insulin therapy and the target range of blood glucose concentration. The 2005 guidelines recommend lowering of blood glucose in patients with acute ischemic stroke when serum glucose level is greater than 200 mg/dl.
10 Required Scenarios (With Key Points)

1. Respiratory Arrest – ABC’s if pulse, support, respiration, intubate if necessary
2. VF treated with CPR and AED – ABC’s, defibrillation. As soon as AED arrives, follow AED prompts
3. Ventricular Fibrillation/Pulseless VT – ABC’s, shock, resume CPR (2 minutes), give a drug, draw up, and repeat steps. Start with Epinephrine q 3-5 minutes, may substitute Vasopressin. X 1, then Amiodarone 300mg
4. Pulseless Electrical Activity – ABC’s, Possible causes, Epinephrine, Atropine
5. Asystole – Same as PEA
6. Acute Coronary Syndromes – MONA, STEMI, or not, Fibrinolytic therapy, cath lab
7. Symptomatic Bradycardia – Atropine, 2nd or 3rd Degree Blocks = transcutaneous pacing
8. Unstable Tachycardia – Cardiovert
9. Stable Tachycardia – Meds: Narrow Complex/regular = Vagal, then Adenosine, narrow/irregular Calcium Channel Blockers, wide complex = Amiodarone
10. Acute Stroke – Check glucose, non-contrast CT of head read by radiologist, fibrinolytics within hours if BP OK

*** Used throughout the course outline indicates that there is a test question related to the noted material.

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