# Advanced Cardiac Life Support (ACLS) Review

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Advanced cardiac life support (ACLS) is a two-day course that teaches students to recognize and treat cardiac arrest, arrhythmias, acute coronary syndromes, stroke, cardiac arrest in pregnant women, and cardiac arrest in situations involving drug overdose, drowning, anaphylaxis, hypothermia, and electrocution. The ACLS course involves lectures, self-study, and simulations of medical emergencies. Drugs, advanced airways, defibrillation, cardioversion, and cardiac pacing are all covered.

## OBJECTIVES

When the student has finished this module, he/she will be able to:

1. Identify VF, pulseless VT, and PEA.
2. Know the indications for using amiodarone.
3. Identify the basic principles of stroke care.
4. Recognize the components of a basic stroke assessment.
5. Identify the appropriate treatment for STEMI.
6. Identify the indications for using adenosine.
7. Correctly identify aspects of the ACLS algorithm for tachycardia.
8. Recognize the indications/safety measures of using defibrillation, cardioversion.
9. Identify a basic method of arrhythmia recognition.
10. Identify the indications for using vasopressin
11. Recognize three conditions for which epinephrine is a first-line treatment.
12. Identify the ECG characteristics of third-degree heart block.
13. Identify the antidote for digoxin toxicity.
14. Identify the primary use for atropine, and the correct dose.
15. Recognize treatments for cardiac arrest caused by drowning, hypothermia
16. Recognize treatments for cardiac arrest caused by electrocution, anaphylaxis.
17. Recognize how to treat cardiac arrest in pregnant women and after an overdose.
18. Identify four therapies that should not be routinely used to treat cardiac arrest.
19. Identify treatments for VF, pulseless VT, and unstable bradycardia,

20. Identify three aspects of pacemaker function that must be assessed.

## ABBREVIATIONS

**ACS**: Acute coronary syndromes
**AED**: Automatic external defibrillator
**AF**: Atrial fibrillation
**BLS:** Basic life support.
**NSTEMI**: Non-ST-segment myocardial infarction
**PEA**: Pulseless electrical activity
**PCI:** Percutaneous coronary intervention.
**PSVT**: Paroxysmal supraventricular tachycardia
**STEMI**: ST-segment elevation myocardial infarction
**SVT:** Supraventricular tachycardia
**TDP**: Torsades de pointes
**VF**: Ventricular fibrillation
**VT**: Ventricular tachycardia

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## STARTING OUT

The first step in ACLS is **ABCD.** Assess ***a***irway, ***b***reathing, and ***c***irculation, and determine whether ***d***efibrillation is needed. If the victim is unconscious, apneic and pulseless, activate the EMS system/call for help and prepare to defibrillate. Remember: The American Heart Association has revised BLS protocol. It is now recommended that cardiac compressions be done first. It is felt that even in someone who has suffered a cardiac arrest, there is enough oxygen saturation in the blood so that circulation of the oxygenated blood is a more important concern immediately after cardiac arrest.

**Learning Break:** If the victim has/suspected to have a cervical spine injury and is lying face down, the current ACLS guidelines consider it acceptable to roll the victim to the supine position. Restrict motion of the spine and keep the head, neck, and spine in alignment.

**Learning Break:** Regardless of the where you are or the situation, it is vital that you call for help or activate the EMS system. BLS techniques can provide temporizing care, but the medical emergencies covered in ACLS require sophisticated and complex care. BLS may prevent someone from dying immediately, but if the patient has suffered an MI or VF, etc., BLS will not keep them alive.

## AIRWAY MANAGEMENT

Airway control in medical emergencies is critical. If the victim is apneic, has no gag reflex, or is not ventilating adequately, an **advanced airway** may be needed to a) protect airway patency, and b) deliver oxygen efficiently. There are complicated methods that can be usedd to assess whether or nor someone needs an advanced airway. However, in many of the medical emergencies discussed in ACLS, the patient is apneic and pulseless and the need for an advanced airway is obvious.

Common advanced airways are the endotracheal tube (ET), esophageal-tracheal double lumen airway, and the laryngeal mask airway (LMA). When inserting an advanced airway, each attempt should last no longer than 30 seconds; oxygenate with 100% $O_2$ between attempts. Confirm correct airway position (an end-tidal $CO_2$ works best), and then secure it.

- **Endotracheal tube:** The ETT delivers oxygen very efficiently and protects well against aspiration.

- **Esophageal-tracheal double lumen airway**: This has a tracheal lumen, an esophageal lumen and a cuff that seals the airway. It can be inserted by personnel not authorized for endotracheal intubation. The patient can be ventilated and protected against aspiration even if the tracheal lumen is inserted into the esophagus.

- **Laryngeal mask airway:** The LMA has an airway attached to a face mask, but does not have an inflatable cuff to protect against aspiration. It is quick to use and can be inserted by personnel that are not authorized for endotracheal intubation.

**Learning Break:** *The LMA and the esophageal-tracheal double lumen airway* are *not intended for long-term use.*

If the victim does not need an advanced airway, but needs supplemental oxygen, a **nasal cannula**, **Venturi mask**, **simple face mask**, or **nonrebreather mask** are possibilities. The amount and precision of oxygen delivery differs with each device.

## PHARMACOLOGY

The following drugs are the one most commonly used in ACLS. Some could fit into more than one category.

### Antidotes

- **Naloxone:** Naloxone is a competitive antagonist at opioid receptors used to reverse respiratory depression caused by opioid overdose. Dose: 0.4-2.0 mg, IV; repeat PRN every few minutes, up to 10 mg. It can be given IM, SC, and via an ETT. Adverse effects: Agitation and tachycardia,

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caused by return to conscious and/or precipitation of withdrawal. Contraindications: Use cautiously if the patient has cardiovascular disease or cardiovascular instability.

- **Flumazenil:** Flumazenil is a competitive antagonist at benzodiazepine receptor sites. It is used to reverse sedation and respiratory depression caused by benzodiazepines. Dose: 0.2 mg IV over 15 seconds. If no response, give 0.3 mg IV over 30 seconds; no response, give 0.5 mg over 30 seconds; no response, 0.5 mg at 1 minute intervals to a total of 3 mg. Adverse effects: Agitation, hypertension, seizures. Contraindications: Seizure disorder, overdose with a tricyclic antidepressant, benzodiazepine dependency, head injuries.

- **Glucagon:** Glucagon acts a positive inotrope, dromotrope, and chronotrope by increasing intracellular concentrations of cAMP. Glucagon is used to reverse the effects of beta blocker and calcium channel blockers. Dose: 3-10 mg IV over 1 minute: continuous infusion of 1-5 mg/H as needed. Adverse effects: Vomiting, hypotension. Contraindications: Sensitivity to glucagon, pheochromocytoma. Glucagon is not compatible with normal saline solution.

- **Digoxin-specific antibody fragments:** Aka **Fab fragments.** These bind to digoxin and decrease serum digoxin level. They are used to treat digoxin toxicity. Dose: Based on the serum digoxin level or the amount of digoxin ingested. Give the dose IV over 30 minutes using a 22 micron filter. Adverse effects: Precipitation of heart failure, hypokalemia, rapid ventricular rate in patients with atrial fibrillation. No contraindications.

- **Glucose and insulin**: This combination has been used to treat beta-blocker and calcium channel blocker overdose. Empirical dosing.

- **Calcium:** Used to treat calcium channel blocker and beta blocker overdose. Dosing is empirical: start with 1 gm calcium chloride, IV bolus, repeat PRN.

- **Cyanide antidote:** Sodium thiosulfate. Dose: 12.5 g IV, rapid IV or over 10-30 minutes as needed. Nitrites induce methemoglobinemia and prevent cyanide binding to hemoglobin, but must be used cautiously. Cyanocobalamine also binds cyanide and is safer.

- **Sodium bicarbonate:** Used to treat overdose with tricyclic antidepressants. Dosing is empirical: use boluses and continuous IV infusion to maintain blood pH between 7.50-7.55.

**Vasopressors**

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- **Dopamine:** Dopamine stimulates alpha and beta-1 receptors. It is used to treat hypotension and unstable bradycardia. Low doses have a positive inotropic and chronotropic action; high doses cause peripheral vasoconstriction. Dose: IV infusion, titrate PRN, doses > 50 mcg/kg/min cause arrhythmias and vasoconstriction. Correct hypovolemia before using dopamine, and *do not mix dopamine with alkaline solutions/medications*. Adverse effects: Hypertension, arrhythmias. Contraindications: Tachyarrhythmias, ventricular arrhythmias, hypovolemia.

- **Norepinephrine:** Norepinephrine stimulates alpha and beta receptors, and is used to treat hypotension. It has positive inotropic and chronotropic action, and causes peripheral vasoconstriction. Dosing is empirical: 2 – 8 mcg/minute IV infusion is a common starting point. Do not infuse with alkaline solutions. Adverse effects: Hypertension, peripheral vasoconstriction, bradycardia. Contraindications: Chloral hydrate intoxication, concurrent/recent use of some anesthetics and MAO inhibitors, hypovolemia.

- **Epinephrine:** Epinephrine stimulates alpha and beta receptors. It acts as a positive inotrope and chronotrope, causes peripheral vasoconstriction. It is used to treat hypotension, unstable bradycardia, cardiac arrest, PEA, VF, pulseless VT, and anaphylaxis. Dose: IV bolus, 1 mg every 3-5 minutes; continuous infusion, start at 1 mcg/min and titrate. Adverse effects: Hypertension, arrhythmias, and cerebral hemorrhage. Contraindications: Ventricular arrhythmias.

**Learning Break:** Dobutamine, dopamine, epinephrine, and norepinepherine can cause serious tissue damage if the drug infiltrates the tissue. Regitine should be used to treat extravasation of these drugs.

## Beta Blockers

Beta blockers are competitive antagonists of catecholamines at beta receptors and: some stabilize the cardiac membrane. They lower blood pressure, decrease heart rate, decrease myocardial oxygen consumption, and decrease cardiac conduction. They can be given PO or IV.

Beta blockers reduce the risk of reinfarction and arrhythmias in STEMI, NSTEMI, and unstable angina and slow down or convert tachyarrhythmias. Adverse effects: Bradycardia, hypotension, heart blocks. Contraindications: Reactive airway disease, heart blocks, hypotension, severe bradycardia.

## Calcium Channel Blockers

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Calcium channel blockers inhibit calcium influx into the cell. They decrease heart rate, decrease cardiac oxygen consumption, slow conduction, decrease blood pressure, and decrease cardiac contractility.

Calcium channel blockers are used emergently to convert/control some tachyarrhythmias. They can be given PO or IV. Adverse effects: Bradycardia, hypotension, heart blocks. Contraindications: Wide complex tachycardias of unknown origin, heart blocks, severe, left ventricular dysfunction.

**Learning Break:** Current (2010) ACLS guidelines do not recommend the use of calcium channel blockers in STEMI

## Anticoagulants

Heparin and low molecular weight heparins (LMWH) decrease the activity of specific clotting factors. They prevent clot formation and extension of existing clots. They do not dissolve existing clots. They are used for patients with STEMI , NSTEMI, DVT, PE, and AF. They are given as a weight-based IV bolus, then a continuous IV infusion (heparin) or SC injections (LMWH). Adverse effects: Bleeding, bleeding complications. Contraindications: Heparin-induced thrombocytopenia, platelet count < 100,000/$mm^3$

## Sympathomimetics

- **Isoproterenol:** Isoproterenol stimulates beta 1 and beta-2 receptors. It acts as a positive inotrope and chronotrope and causes peripheral vasodilation. It is used to treat severe bradycardia, TDP, and calcium channel blocker and beta blocker overdose. Dose: 2-10 mcg/min continuous IV infusion. Adverse effects: Arrhythmias, hypertension, tachycardia. Contraindications: VT or VF.

- **Dobutamine:** Dobutamine stimulates beta 1 receptors and acts as a positive inotrope; it also (less so) increases heart rate. It is used as short-term therapy to correct hypotension. Dose: 2-20 mcg/kg/min, continuous IV infusion. Adverse effects: Tachycardia, hypertension, hypotension. Contraindications: Drug-induced shock, concomitant use of beta-blockers, idiopathic hypertrophic subaortic stenosis, mixing with alkaline solutions.

## Morphine

Morphine interacts with opiate receptors to produce analgesia, and it is used to treat ACS. Dose: 2-4 mg IV over 1-5 minutes, repeat every 5-30 minutes PRN. Adverse effects: Respiratory depression, sedation, hypotension. Contraindications: Respiratory depression, hypotension.

## Ace Inhibitors

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Angiotensin-converting enzyme (ACE) inhibitors are PO medications that lower blood pressure by preventing the conversion of angiotensin I to angiotensin II. Use in ACSs to reduce the progression of heart failure and reduce the occurrence of sudden death and new MI. Adverse effects: Hypotension, tachycardia, angioedema. Contraindications: Sensitivity to the drugs.

## Aspirin

Aspirin is a non-steroidal anti-inflammatory and analgesic used to treat ACSs and stroke. Dose: 160-325 mg, PO: the aspirin should be chewed. Adverse effects: GI distress, GI bleeding, tinnitus. Contraindications: Active bleeding, bleeding disorders, hypersensitivity to NSAIDs, asthma, vitamin K deficiency, hypoprothombinemia.

## Vasodilators

- **Nitroprusside:** Nitroprusside relaxes vascular smooth muscle, and it is used to treat severe hypertension. Dose: 0.5-10 mcg/kg/min, continuous IV infusion. *Start with low doses as the decrease in blood pressure is immediate and dramatic.* Adverse effects: Hypotension, cyanide toxicity (some nitroprusside is metabolized to cyanide). Contraindications: Coarctation of the aorta, compensatory hypertension related to increased intracranial pressure. *The solution must be protected from light: cover the bottle with foil.*

- **Nitroglycerin:** Nitroglycerin dilates coronary arteries and relaxes smooth muscle of veins and arteries. It lowers afterload and preload and decreases blood pressure. It is used for chest pain associated with ACSs. Dose: Subingual: 0.3 or 0.4 mg, every 3 minutes up to 3 doses. Aerosol spray: 0.5 – 1.0 second spray, 5 minute intervals, 3 sprays within 15 minutes. IV: 10-20 mcg/min, titrate in increments of 5-10 mcg/min. Adverse effects: Hypotension, headache. Contraindications: Hypotension, inferior wall MI, shock, head trauma, closed angle glaucoma, cardiac tamponade, restrictive cardiomyopathy or pericarditis. *Use a glass bottle and non-absorbent tubing.*

## Antiarrhythmics

- **Adenosine:** Adenosine slows conduction through the AV node. It is used to treat PSVT and can be used to treat stable, regular, wide-complex tachycardia. Dose: 6 mg, rapid IV push over 1-3 seconds; give 12 mg if the arrhythmia continues after 1-2 minutes; give another 12 mg if needed. Adverse reactions: Flushing, headache, palpitations, bradycardia, sinus arrest. Contraindications: Asthma, third degree heart block, sick sinus syndrome, AF or atrial flutter caused by WPW syndrome.

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- **Atropine:** Atropine increases conduction through the AV node and increases impulse formation in the SA node. It is used to treat symptomatic bradycardia. *Atropine is unlikely to be effective for treating Type II second-degree AV block or third-degree AV block.* Dose: 0.5-1.0 mg, IV push, repeat every 3-5 minutes. Adverse effects: Anticholinergic signs/symptoms, tachycardia. Contraindications: Bradycardia, myocardial ischemia, narrow angle glaucoma.

- **Amiodarone**: Amiodarone prolongs the refractory period and the action potential. It is used for treating VF, pulseless VT, and stable, wide-complex tachycardia. Dose: For VF/VT, 300 mg IV bolus, PRN 150 mg every 5 minutes. Wide-complex tachycardia: 150 mg IV bolus over 10 minutes, PRN every 10 minutes, then 1 mg/minute IV infusion. The 24 hour maximum dose is 2.2 grams. Adverse effects: Bradycardia, hypotension, heart blocks, sinus arrest. Contraindications: Digitalis-induced arrhythmias, second and third-degree AV blocks. Use cautiously if the patient takes beta-blockers or calcium channel blockers.

- **Lidocaine**: Lidocaine suppresses automaticity and increases the refractory period. It second-choice therapy for VF or pulseless VT and not very effective. It can be used for stable, monomorphic VT. Dose: 1.0/1.5 mg/kg, IV. Maintenance infusion of 1-4 mg/min. Adverse effects: Hypotension, bradycardia, conduction delays, confusion. Contraindications: Adams-Stokes Syndrome, Wolff-Parkinson-White (WPW) syndrome, SA or AV node blocks.

- **Sotalol:** Sotalol is a potassium channel blocker and non-selective β-blocker. It is used to treat stable, monomorphic VT. Dose: 1.5 mg/kg IV over 5 minutes. Adverse effects: Bradycardia, hypotension, TDP. Contraindications: Prolonged QT, CHF.

- **Procainamide**: Procainamide slows action potential. It is used to treat stable, monomorphic VT and AF caused by pre-excitation (e.g., WPW syndrome). Dose: 20-50 mg/minute until the arrhythmia is controlled, hypotension occurs or QRS > 50%. The maximum dose is 17 mg/kg. Infuse at 1-4 mg/minute for maintenance. Adverse effects: Hypotension, heart block, confusion, conduction delays. Contraindications: Prolonged QT, CHF, Second and third-degree heart block.

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**Miscellaneous Drugs**

- **Glycoprotein IIb/IIIa inhibitors:** These drugs bind to platelet surface receptors and inhibit platelet aggregation. They are used (if indicated) for patients suffering an acute coronary syndrome. Adverse effects: Bleeding, bradycardia, hypotension, thrombocytopenia. Contraindications: Vary with

each drug, but intracranial hemorrhage, bleeding, platelet count < 100,000/$mm^{3,}$ and hypertension are common to them all.

- **Vasopression**: Vasopresin is a diuretic that causes peripheral and coronary vasoconstriction. It is used as an alternative to epinephrine for treating cardiac arrest, VF, pulseless, and PEA. Dose: 40 units, IV. Adverse effects: Necrosis if the drug extravasates, myocardial ischemia, bronchoconstriction, angina Contraindications: Coronary artery disease.

- **Magnesium:** Magnesium is used to treat torsades de pointes (TDP) and life-threatening arrhythmias caused by digoxin toxicity. Dose: Cardiac arrest, 1-2 g over 5-20 minutes. Adverse effects: Asystole, hypotension, circulatory collapse, heart block if given to digitalized patients, Contraindications:

### Fibrinolytics

- **Alteplase, tenecteplase, etc.:** These drugs interrupt the clotting process. They break down existing clots and prevent new ones from forming. They are used for patients with STEMI and ischemic stroke. Dosing varies with each drug. Adverse effects: Hemorrhage. Contraindications: Absolute contraindications include prior intracranial hemorrhage, cerebral vascular lesion, brain tumor, suspected aortic dissection, active bleeding (exclude menses), significant close head or facial trauma within three months, ischemic stroke within three months *except* acute ischemic stroke within three hours. There is also an extensive list of relative contraindications.

## DEFIBRILLATION, CARDIOVERSION, AND TEMPORARY PACEMAKERS

### Defibrillation

**Defibrillation** using an **AED** or a **defibrillator** is used to treat VF and pulseless VT. It delivers electrical current to the heart, depolarizing the entire myocardium and reestablishing sinus rhythm.

Using an AED: Place the electrode pads on the patient's chest. Press the **on** button. The AED will signal it is ready: press the **analyze** button. The AED will give a prompt if a shock is needed: hit the **shock** button. A **no shock** needed message is displayed if no shock is needed. *It is not possible to misuse an AED and deliver a shock to the operator or a bystander.*

Using a defibrillator: Put conductive gel on the patient's chest, and set the energy level to 360 joules (monophasic) or 120-200 joules (depending on model) for biphasic. Apply the paddles to the chest, press firmly, hit the charge button, and press the charge button on each paddle. (Note: **Monophasic** defibrillators deliver a shock in one direction. **Biphasic** defibrillators deliver a shock that travels back and forth and less current and fewer shocks are needed)

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- Defibrillation effectiveness decreases 5-10% each minute it is delayed.
- Make sure the patient is not lying in water.
- Have bystanders have step away and avoid touching the patient during defibrillation.
- Place AED electrode pads/defibrillator paddles at least 1” from a pacemaker or ICD.
- Remove transdermal medication patches.
- Use special AED pads for children under 8 years.

## Cardioversion

**Cardioversion** (aka countershock) is used to treat unstable tachycardia, unstable atrial flutter/AF of less than 48 hours duration, unstable/monomorphic VT in the awake patient, and supraventricular tachycardia. Cardioversion delivers an electrical current to the heart that is synchronized to the R wave. This interrupts reentry circuits and establishes SA node control.

Apply conductive gel to the chest, set the defibrillator to synchronize mode, and select the energy level: 50 joules for elective cardioversion, up to 200 joules for unstable atrial arrhythmias/unstable ventricular tachycardia. Place the paddles on the chest and press the shock buttons. There is a slight risk that cardioverison will cause arrhythmias.

**Learning Break:** Atrial flutter and AF are arrhythmias that greatly increase the risk of developing peripheral thrombi and thrombi in the heart. The longer these arrhythmias have been present, the more likely it is a thrombi is present and sudden conversion to a normal rhythm can dislodge a thrombus.

## Temporary Pacemakers

Temporary pacemakers include **transcutaneous** and **transvenous** pacemakers. They are used for unstable patients with bradycardia or second or third-degree block, and TDP.

Transcutaneous pacemakers use large electrodes, similar to cardiac monitor electrodes: one posterior, the other anterior. Transvenous pacemakers use an electrode that is threaded into the right atrium. Both are then are attached to a pulse generator that sends an electrical impulse to the heart, initiating a heart beat. The electrical impulse is seen on the ECG as a “spike.” The generator controls rate, sensitivity (sensing intrinsic heart beats), and the amount of pacing energy. The pulse generator delivers enough energy to stimulate cardiac contraction, and it will not do so if the heart beats normally (it will “sense”).

**Learning Break:** Make sure the pacemaker is **firing, capturing** (each pacemaker spike initiates a heart beat) and **sensing.**

## ARRHYTHMIA RECOGNITION AND TREATMENT

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Follow these steps to assess an ECG for an arrhythmia.

- Is the rate fast or slow?
- Is the rhythm regular or irregular?
- Where does the impulse originate?
- Is the impulse conducted normally?

**A normal ECG has a rate of 60-100, the rhythm is regular, and the cardiac impulse originates in the SA node. Normal impulse conduction is: PR interval, QRS complex, ST segment, QT interval, and T wave. All have normal lengths and appearances.**

- **Sinus tachycardia**: The rate is > 100, otherwise the ECG (except perhaps the QT) is normal. Sinus tachycardia is a *response* to stress, e.g, fever, hemorrhage, hypoxia. Patients are usually asymptomatic, but signs/symptoms of low cardiac output are possible. Treat the cause.

- **Sinus bradycardia:** The rate is < 60 but otherwise the ECG is normal. Patients may be asymptomatic, but signs/symptoms of low cardiac output are possible. Causes: Inferior wall MI, digoxin, beta blocker, calcium channel blocker, clonidine, or opioid toxicity, increased intracranial pressure, or increased vagal tone. Treatment: Pacing, atropine, dopamine or epinephrine, antidotes when appropriate.

- **Atrial fibrillation:** The heart rate can be slow, fast, or normal. The rhythm is irregularly irregular. The pacing impulse is initiated from many ectopic atrial sites. P waves are rarely visible. The PR isn't measurable. The QT can't be measured. Causes: Myocardial infarction, hypertension, congestive heart failure, drugs, thyroid disease. Patients may be asymptomatic, but signs/symptoms of low cardiac output are possible. Treatment: Unstable patients, duration < 48, cardioversion. Unstable patients, > 48 hours duration, use digoxin, beta blockers, calcium channel blockers, amiodarone.

- **Atrial flutter:** The heart rate is usually normal, but can be > 100. The rhythm can be regular or irregular. The pacing impulse is from a single ectopic atrial focus. The P waves appear as saw toothed. The PR and the QT can't be measured. The T wave is often obscured by the ectopic focus. Causes: MI, digoxin toxicity, thyroid disease, pericarditis. Patients are usually asymptomatic, but signs/symptoms of low cardiac output are possible. Treatment: Cardioversion for unstable patients.

- **Atrial tachycardia:** The heart rate is > 100. The rhythm is normal. The pacing impulse is generated from a single atrial focus. The P wave is usually normal, and the PR may/may not be measurable. Patients are usually asymptomatic, but signs/symptoms of low cardiac output are

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possible. Causes: Digoxin toxicity, MI, pericarditis, COPD, and stress. Cardioversion and adenosine can be used to treat.

- **Junctional tachycardia:** The heart rate is > 100. The rhythm is normal. The pacing impulse is generated from the AV node. P waves may/may not be visible, and the PR interval often can't be measured. Causes: Digoxin toxicity, MI, cardiomyopathy, sinus node dysfunction, hypoxia. Junctional tachycardia is treated with calcium channel blockers or beta blockers.

- **Premature ventricular contractions:** PVCs) are ectopic impulses originating in the ventricles. The rate is normal. The rhythm is irregular. There is no P wave preceding the PVC, the QRS is prolonged and abnormal. The T wave is deflected in the opposite direction of the QRS. PVCs are dangerous if they are coupled, multiform, occur every other beat (bigeminy) or every third beat (trigeminy), or if they are close to the T wave. Most patients are asymptomatic. Causes: MI, hypoxia, stimulant drugs, digoxin toxicity, and hypokalemia or hypocalcemia. PVCs can precipitate VT. Treatment: Correct the cause, use amiodarone, procainamide, sotalol, or lidocaine if the patient is symptomatic or the PVCs are dangerous.

- **Ventricular tachycardia:** VT. The rate is > 100. The rhythm is regular. The pacing impulse is generated in the ventricles. There are no P waves, the QRS is prolonged and bizarre. The PR and QT intervals can't be measured. The T wave deflection is opposite of the QRS, but the T wave may not be seen. Causes: MI, electrolyte imbalances, PVCs, digoxin toxicity. Most patients are unconscious, pulseless, and do not have a blood pressure. Treatment : Amiodarone, sotalol, or procainamide if the patient is conscious. If the patient is conscious but unstable, synchronized cardioversion is used. If the patient is unconscious, defibrillation and IV epinephrine or vasopressin are used.

- **Torsades de pointes:** The rate is > 100. The rhythm is usually regular. The pacing impulse is generated in the ventricles. There are no P waves or PR, the QT interval can't be measured, and the T waves are absent or are opposite in deflection of the QRS. The QRS is prolonged and bizarre in appearance, randomly changes deflection, and the baseline of the rhythm undulates. Causes: Electrolyte abnormalities, drug-induced or inherited QT prolongation, and myocardial ischemia. The patient is almost always (or soon becomes) unconscious and pulseless. Treatment: Electrolyte supplementation, overdrive pacing, IV magnesium, isoproterenol, or defibrillation and epinephrine if the patient is unconscious and pulseless.

- **Ventricular fibrillation:** The rate can't be measured. The rhythm is very irregular. All intervals, complexes, waves, etc, are absent or

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unmeasurable. The impulses for ventricular fibrillation (VF) originate in the ventricles from many foci, and the ECG appears as an irregular, undulating line with no waves, complexes, etc. Causes: MI, ischemia, coronary artery disease, electrolyte imbalances. The patient is unconscious, apneic, and pulseless. Treatment: CPR, defibrillation,

- **Pulseless electrical activity:** The rate is normal, the rhythm is regular, and the ECG appears normal. However, the ECG represents electrical activity that does not cause cardiac contraction. The patient will be unconscious, apneic, and pulseless. Causes: MI, hypothermia, acidosis, electrolyte abnormalities, and cardiac tamponade. Treatment: CPR, epinephrine or vasopressin. **Look for and treat the underlying cause.**

**Learning Break:** Hypovolemia, hypoxia, hydrogen ion acidosis, hypo/hyperkalemia and hypothermia, toxins, tamponade, trauma, thrombosis, and tension pneumothorax are common causes of PEA: the **five Hs and five Ts.**

- **First degree AV block:** The rate is normal and the rhythm is regular. The ECG is normal except for a PR interval > 0.20 seconds, caused by delayed conduction through the AV node, atria or His-Purkinje system. Causes: Digoxin toxicity, coronary artery disease, inferior wall MI, electrolyte imbalances. First degree AV block is dangerous if it is caused by an inferior wall MI. Almost all patients are asymptomatic. Symptomatic patients may need a permanent pacemaker.

- **Second-degree AV block, Type I:** Aka **Mobitz I** or **Wenckebach.** The rate is normal. The atrial rhythm is regular, but the ventricular rhythm is irregular. The ECG is normal except for the PR interval; this is prolonged and gets progressively longer until a P wave that is not conducted. Causes: Inferior wall MI, coronary artery disease, digoxin toxicity. The patients are usually asymptomatic, but Mobitz I can progress to a more serious block. Treatment: Atropine, pacing. Dopamine, epinephrine, or isoproterenol are second-line therapies.

- **Second-degree AV block, Type II:** Aka **Mobitz II**. The rate is normal. The rhythm can be regular or irregular. The pacing impulse originates in the SA node, but only some P waves are conducted, e.g., every third or fourth. Most patients are asymptomatic, but third degree heart block often develops. Causes: Anterior wall MI, coronary artery disease, myocarditis. Treatment: Atropine (may not work), pacing. Dopamine, epinephrine, or isoproterenol are second-line therapies.

- **Third-degree AV block:** Aka **complete heart block.** The rate is < 100, usually 20-40. No P waves are conducted: the pacing impulse originates in the AV junction (normal QRS) or the ventricles (Wide QRS, opposite T-wave deflection). Patients can be asymptomatic, but signs/symptoms of

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low cardiac output are possible. Causes: Anterior or inferior wall MI, digoxin, beta blocker or calcium channel blocker toxicity, coronary artery disease. Treatment: Atropine (may not work), pacing. Dopamine, epinephrine, or isoproterenol are second-line therapies.

- **Premature atrial contractions:** Premature atrial contractions (PACs) are caused by an ectopic focus atrial focus. The rhythm is irregular, but otherwise the ECG is normal. A P wave often precedes a PAC, but can be hard to see. After a PAC, there is compensatory pause. Patients are asymptomatic. PACs may initiate atrial flutter or fibrillation, otherwise they are not dangerous. Causes: Atherosclerotic heart disease, myocardial ischemia, digoxin toxicity, stress, stimulant drugs, and congestive heart failure. No treatment is needed.

## TREATMENT ALGORITHMS

Algorithms provide criteria for identifying medical emergencies and they outline a treatment plan for each emergency. ACLS algorithms: Cardiac arrest, pulseless arrest, bradycardia, tachycardia, and acute coronary syndromes.

### Cardiac Arrest

Follow the BLS sequence then assess the rhythm for **PEA**, **VF, pulseless VT**, or **asystole**.

- **PEA:** perform five cycles/two minutes of CPR.
- Administer epinephrine or vasopressin.
- Perform five cycles/two minutes of CPR.
- Assess the rhythm, defibrillate if indicated, and continue with the ventricular tachycardia/ventricular fibrillation treatment.
- If there is no shockable rhythm, repeat epinephrine and vasopressin.

- **Asystole:** follow PEA guidelines.

- If **pulseless VT** or **VF,** defibrillate.
- Perform five cycles/two minutes of CPR.
- Assess the rhythm. If not shockable, administer epinephrine or vasopressin, perform five cycles/two minutes of CPR and assess. If the rhythm is shockable, defibrillate, give epinephrine or vasopressin, perform five cycles/two minutes of CPR and assess. Consider using amiodarone.

**Fluid boluses, atropine, sodium bicarbonate, calcium, and pacing should not be routinely used to treat cardiac arrest. The precordial thump should**

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**only be used for a witnessed, unstable ventricular arrhythmia if defibrillation is not immediately possible.**

**Learning Break:** Hypovolemia, hypoxia, hydrogen ion acidosis, hypo/hyperkalemia and hypothermia, toxins, tamponade, trauma, thrombosis, and tension pneumothorax are common causes of failure to respond to ACLS: the **five Hs and five Ts.**

## Bradycardia

Assess for complications e.g., hypotension, SOB, alterations in consciousness. Correct obvious causes of the bradycardia, then apply the bradycardia algorithm.

- If the patient is stable, treat the cause of the bradycardia and prepare to use a pacemaker.
- If the patient is unstable, give atropine, If atropine is unsuccessful, begin pacing. If the pacemaker and atropine are unsuccessful or while waiting for a pacemaker, give epinephrine, isoproterenol, or dopamine.

**Atropine is unlikely to be effective for treating Type II second-degree AV block or third-degree AV block, and doses of atropine < 0.5 mg may *cause* bradycardia.** If bradycardia doesn't respond to the algorithm, look for an overdose of a beta blocker, calcium channel blocker, clonidine, digoxin, or opioid.
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## Tachycardia

Treat tachycardia by assessing a) patient stability, b) QRS duration, and c) regular or irregular rhythm.

- Assess for complications, e.g., hypotension, SOB, alteration in consciousness.
- Unstable/complications? Perform synchronized cardioversion.
- If the patient is stable, measure the QRS.
- If the QRS is > 0.12 msec and the rhythm is *regular*, determine if it is VT or wide complex SVT. If it is ventricular tachycardia, give amiodarone. Perform synchronized cardioversion if amiodarone does not work. If the rhythm is SVT, give adenosine.
- If the QRS is > 0.12 msec and the rhythm is *irregular,* determine if it is atrial or ventricular. If it is atrial (e.g., AF) try rate and rhythm control, e.g., digoxin, beta blockers, calcium channel blockers. If it is ventricular (e.g., TDP), defibrillate, consider using magnesium and overdrive pacing. If it is ventricular and the patient is awake but unstable, cadiovert.
- If the QRS is < 0.12 msec and the rhythm is irregular, it is most likely AF or atrial flutter. If unstable, cardiovert. If stable and duration > 48 hours, do not cardiovert, use rate and rhythm control drugs.

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- If the QRS is < 0.12 msec and the rhythm is regular, it is most likely SVT. Use vagal maneuvers, adenosine, beta-blockers, calcium channel blockers.

## Acute Coronary Syndromes

The acute coronary syndromes (ACS) are **ST-segment elevation MI, non-ST-segment MI, unstable angina**, and **angina**.

**Basic care for ACS:** a) assess the ABCs, including oxygen saturation, b) get IV access, c) give aspirin, oxygen, nitroglycerin, and morphine if nitroglycerin is not successful, d) obtain a 12-lead ECG, serum electrolytes, coagulation studies, and cardiac marker levels.

Then assess for the need for fibrinolytics: a) Chest pain < 15 minutes or > 12 hours? Don't use fibrinolytics; b) Chest pain > 15 minutes, < 12 hours, look for ST-segment elevation or new LBBB. If these are absent, stop. If they are present, prepare for fibrinolytic therapy and check for contraindications to fibrinolytic therapy.

If the patient has ST-segment elevation or a new LBBB, **STEMI** is possible.

- Start adjunctive therapies: Nitroglycerin, clopidogrel, beta-blockers, glycoprotein IIb/IIIa inhibitors (possibly), ace inhibitors, and low-molecular weight heparin.
- If the time of the onset of symptoms is ≤ 12 hours, prepare for PCI or fibrinolytic therapy. The goals are PIC within 90 minutes of arrival or fibrinolytics within 30 minutes of arrival.
- If the onset of symptoms is > 12 hours, start adjunctive therapy.
- Some patients with STEMI who arrive > 12 hours after symptom onset may benefit from PCI or invasive strategies, but *not* fibrinolytics.

If the patient has ST-segment depression or T-wave inversion, it likely represents **NSTEMI** or **unstable angina.** NSTEMI is distinguished from unstable angina by elevated cardiac biomarkers.

- Start adjunctive therapy.
- Some patients with NSTEMI or unstable angina may benefit from PCI or other invasive strategies, but not from fibrinolytics.

**Angina** is characterized by chest pain, a normal or non-diagnostic ECG, and no elevation of cardiac biomarkers.

- If the cardiac biomarker is elevated, the ECG indicates ischemia, or the patient has shock, pulmonary edema, a heart rate ≥ 100/min AND systolic BP ≤ 100 mm Hg, consider invasive strategies and start adjunctive therapies.
- If none of those are present, treat conservatively.

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## SPECIAL SITUATIONS

### Cardiac Arrest and Anaphylaxis

Cardiac arrest caused by anaphylaxis is due to airway edema/obstruction and poor perfusion caused intense vasodilation and capillary leaking. Patients with a severe anaphylactic reaction are tachycardic, dyspneic, wheezing, cyanotic, and hypotensive.

- **Airway protection is critically important.**
- Give 0.2-0.5 mg epinephrine, 1:1000, IM.
- IV fluids and continuous epinephrine infusion to treat hypotension.
- Vasopressin, antihistamines, inhaled β-adrenergics, and corticosteroids can be used.

### Cardiac Arrest and Pregnancy

The best way to save the fetus is to save the mother. Standard care consists of:

- Patient supine, use manual left uterine displacement (decreases uterine compression on major vessels). If compressions are not inadequate, tilt the patient 30° left.
- Standard chest compressions and rescue breaths; compressions high on the sternum.
- IV access *above* the diaphragm.
- Airway control is critical because intrapulmonary shunting is high.
- Follow ACLS protocols.
- **If resuscitation is not successful within five minutes, perform emergency caesarian.**

**Learning Break:** Causes of cardiac arrest in the pregnant patient include **b**leeding/DIC, **e**mbolism, **a**nesthetic complications, **u**terine atony, **C**V disease, **h**ypertension, **o**ther, **pl**acentia abruptio or previa, **s**epsis: **BEAUCHOPS**.

### Cardiac Arrest and Hypothermia

Hypothermia is a body temperature < 30°C or 86°F. Hypothermia disrupts ion flow and nerve conduction in critical organs.

- **Treat patients aggressively.** Patients with cardiac arrest due to hypothermia have survived after long down times.
- Warm the patient: cardiopulmonary bypass, warm water thoracic lavage, warm IV fluids.
- Standard ACLS protocols.

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**Learning Break:** Drugs, defibrillation, etc. are less effective and may cause harm in hypothermia, but current ACLS protocols do *not* recommend withholding treatment until the patient is warmed.

### Cardiac Arrest and Drowning

- Hypoxia kills drowning victims so start CPR with ABC, not CAB.
- Routine cervical spine stabilization is not indicated unless clearly needed.
- Aspirated water is minimal and rapidly absorbed; don't delay BLS or ACLS trying to remove it.
- Follow ACLS protocols.

### Cardiac Arrest and Electrocution

- *Electrocution and lightning victims can survive long down times if treated aggressively.*
- Due to common exposure circumstances in these cases, musculoskeletal and spine injuries are common: evaluate for their presence.
- Follow ACLS protocols.

**Learning Break:** *Tissue damage and fluid loss can be extensive but internal.* If resuscitation is not successful, make sure the patient isn't volume-depleted.

### Cardiac Arrest and Drug Overdose

Use BLS and ACLS protocols when treating a patient with a cardiac arrest due to drug overdose. Specific issues for this situation:

- For questions about gastric decontamination, call a Poison Control Center. Never use ipecac. Lavage and whole bowel irrigation are rarely needed. Use activated charcoal if the patient is awake, has a gag reflex, and presents within an hour of ingestion.
- There are few antidotes; symptomatic/supportive care is the key to treatment.
- Unknown ingestion? Look for a toxidrome, a cluster of signs/symptoms caused by a particular drug. Example: Opiod toxidrome of sedation, respiratory depression, hypotension, and miosis.

### Stroke

- ABCs, stabilize the patient, ECG, laboratory studies.
- Perform a basic stroke assessment for facial droop, arm drift, abnormal speech.
- Activate stroke team.

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- Emergency CT scan or MRI within 25 minutes of arrival.
- Ischemic stroke? Consider fibrinolytics. If not a candidate, give aspirin
- Hemorrhagic stroke? Consult neurology or neurosurgery.
- Closely serum glucose and temperature: elevations increase morbidity and mortality.

## REFERENCES

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