BLS & ACLS


Chain of survival
Early recognition and call for help
- to prevent cardiac arrest
Early CPR
- to buy time
Early Defibrillation
- to restart the heart
Post resuscitation care
- to restore quality of life

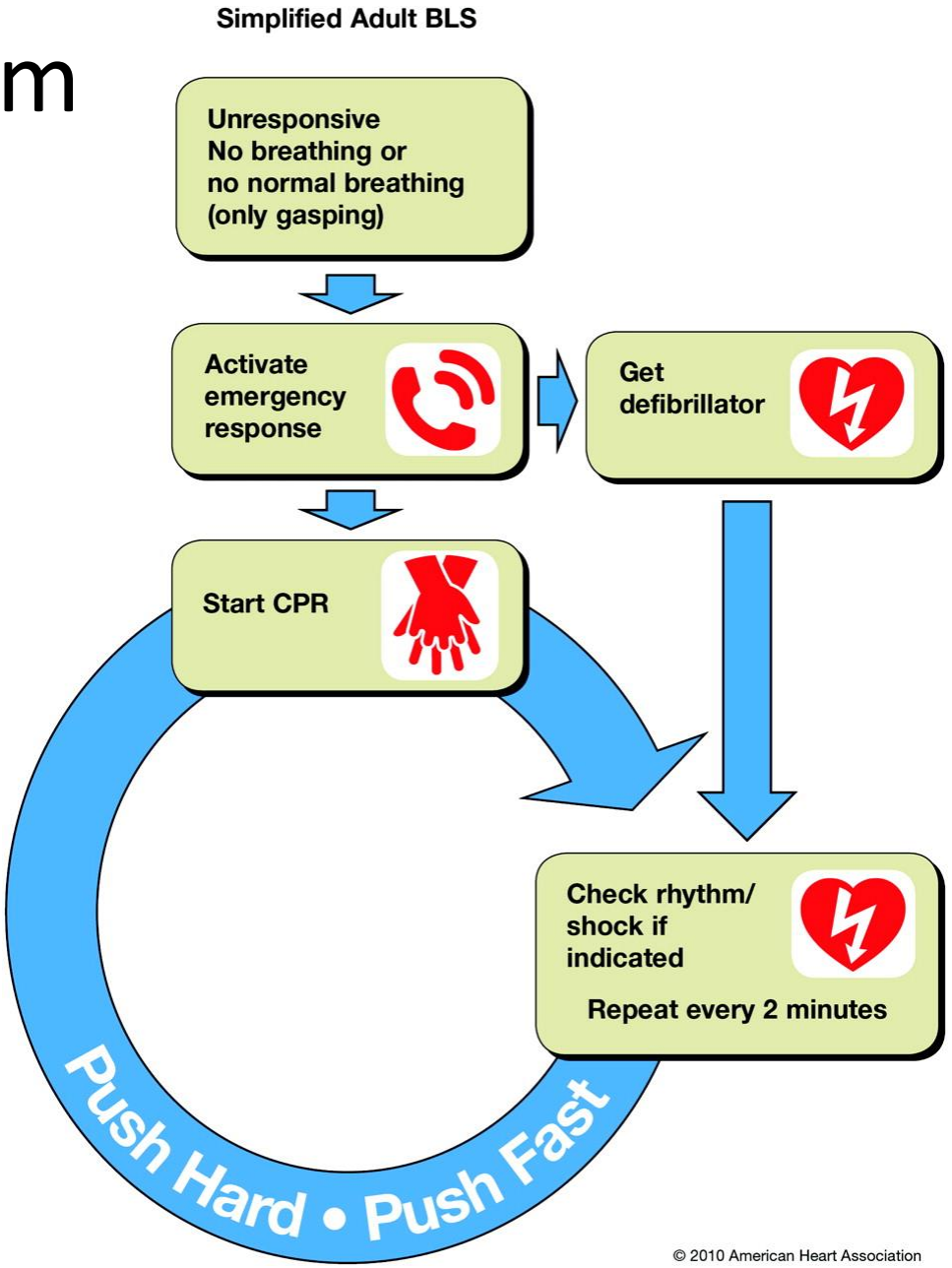
What is the difference?

- Basic life support (BLS) – the initial stage of resuscitation provided by trained medical or non-medical personnel to a victim without any proper equipment.

- Advanced cardiac life support (ACLS) – hospital stage of the first aid, provided by specially trained medical team with appropriate medical equipment and drugs.

BASIC LIFE SUPPORT

Major algorithm of Adult BLS



Assess and minimize your own risks

- Take measures considering your safety:
  - Check traffic
  - Check wires around
  - Check possible gas leak
  - Check water
- Dispose and minimize the risks
- Displace the victim to a safe place carefully

Possible risks for a rescuer: poisoning

(according to the RC UK)

- Gas poisoning – gas mask set
- Chemicals – rubber gloves, protective uniform

Possible risks for a rescuer: Infections

- There are 15 known cases of contamination during CPR: most of them Neisseria meningitidis, tuberculosis.
- There are no known cases of Hepatitis B or C contamination
- 3 cases of HIV contamination due to damaged skin contact

Possible risks for a rescuer: Infections

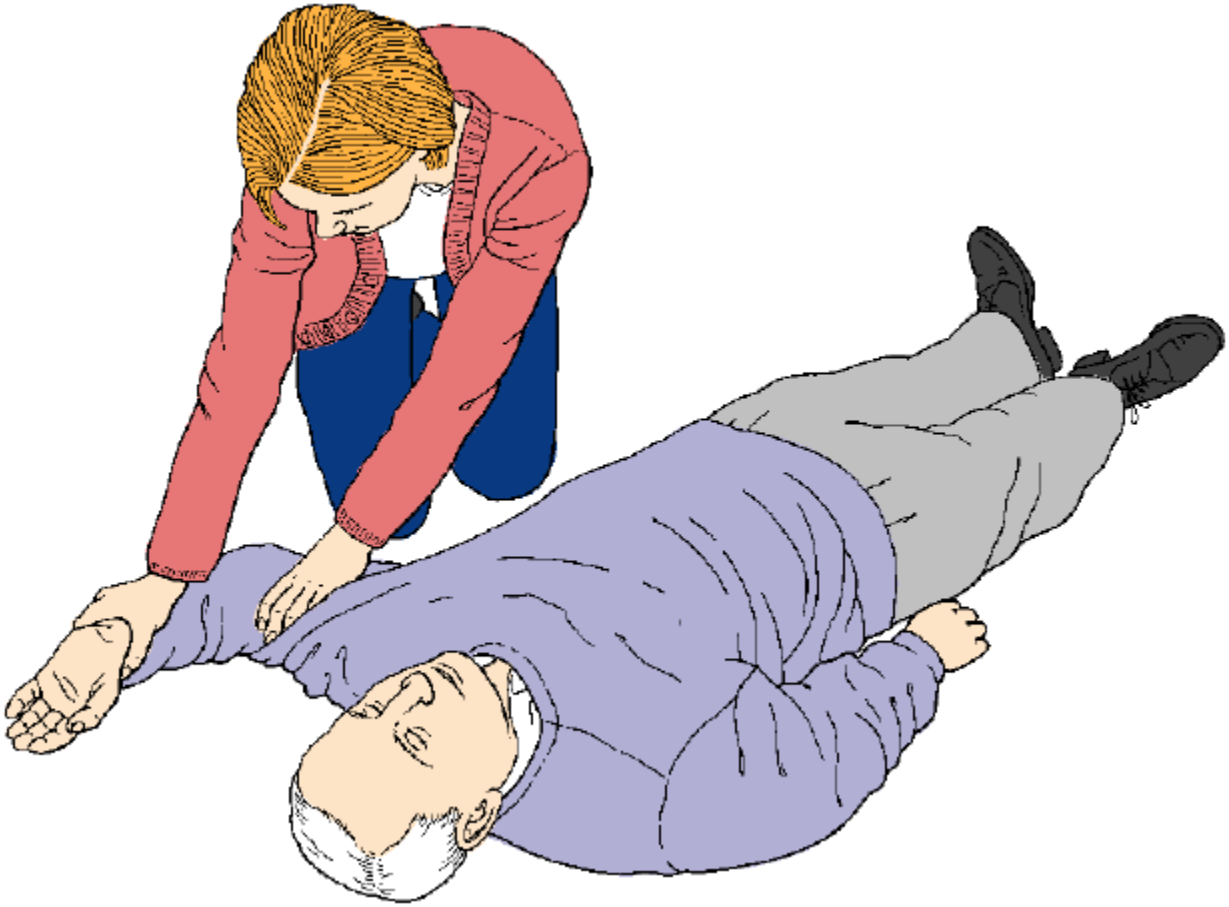
- You can protect yourself using:
  - Rubber gloves
  - Facial masks
  - Plastic glasses
  - Rescue facial masks for “mouth to mouth” ventilation
  - Plastic containers for wastes

CHECK FOR RESPONSE

If conscious, semi-conscious

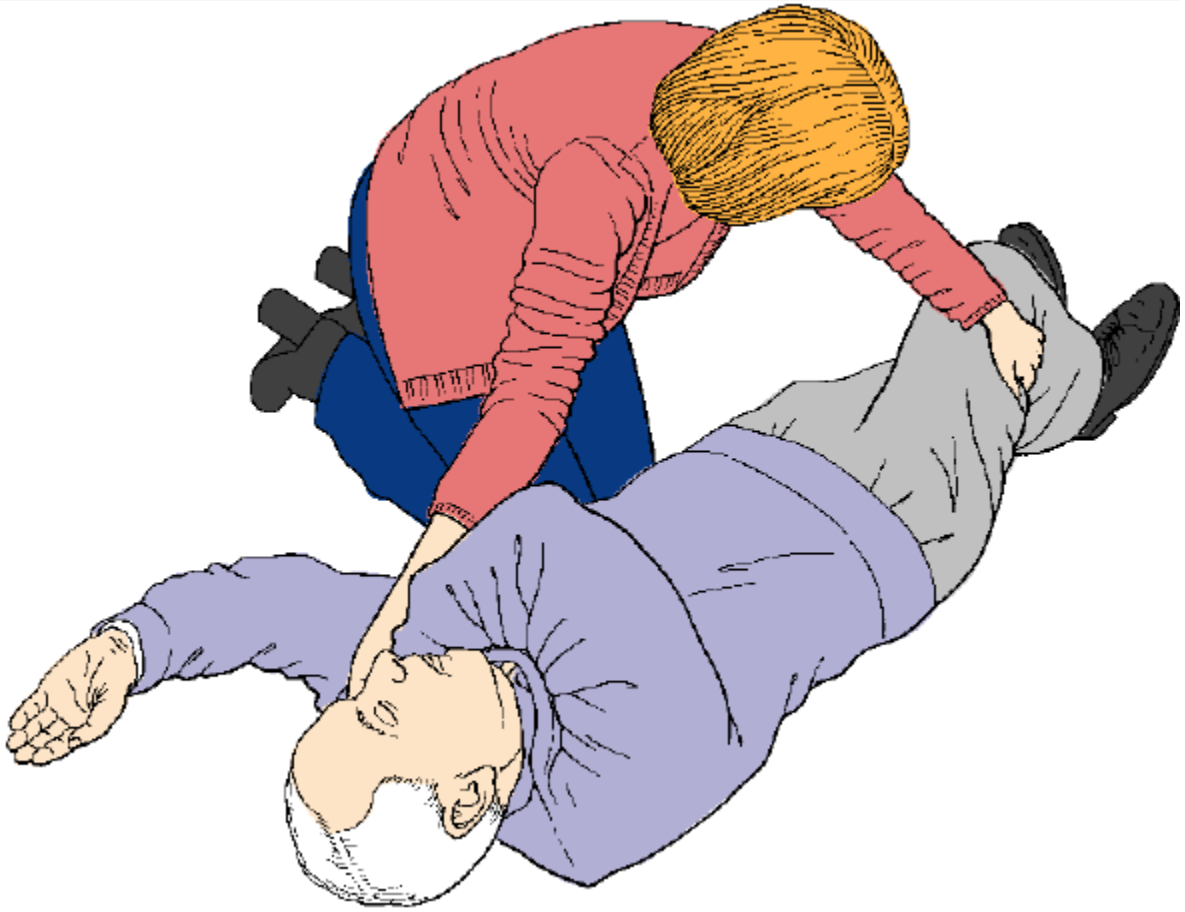
- Call for help
- Assess airway patency and circulation (look, listen, feel) – 10 seconds
- Check for present injuries and bleeding
- Put a person in a “recovery position”

Recovery position
1st stage

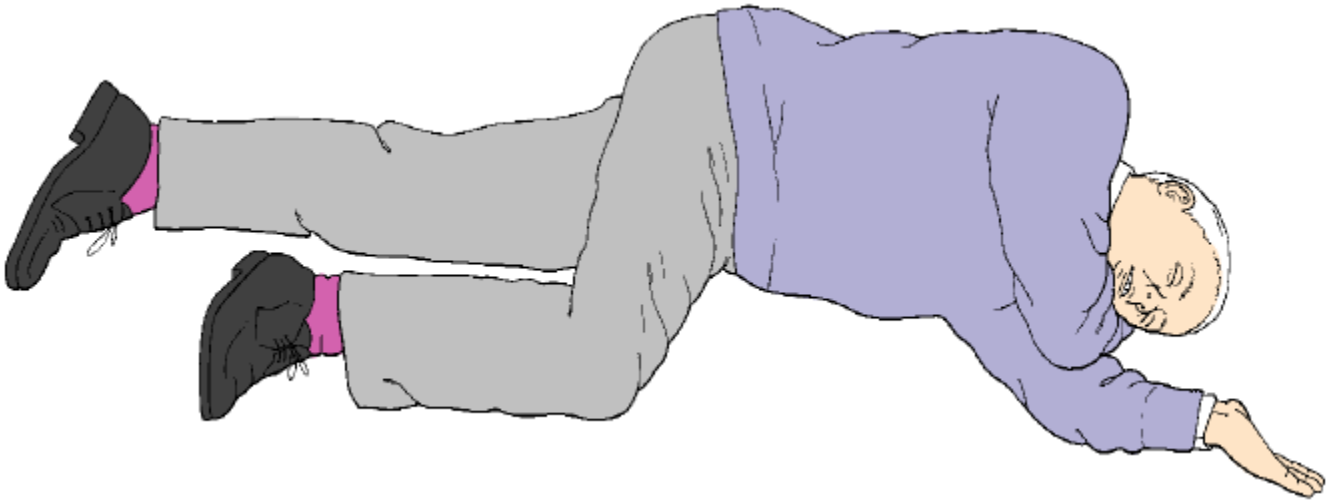


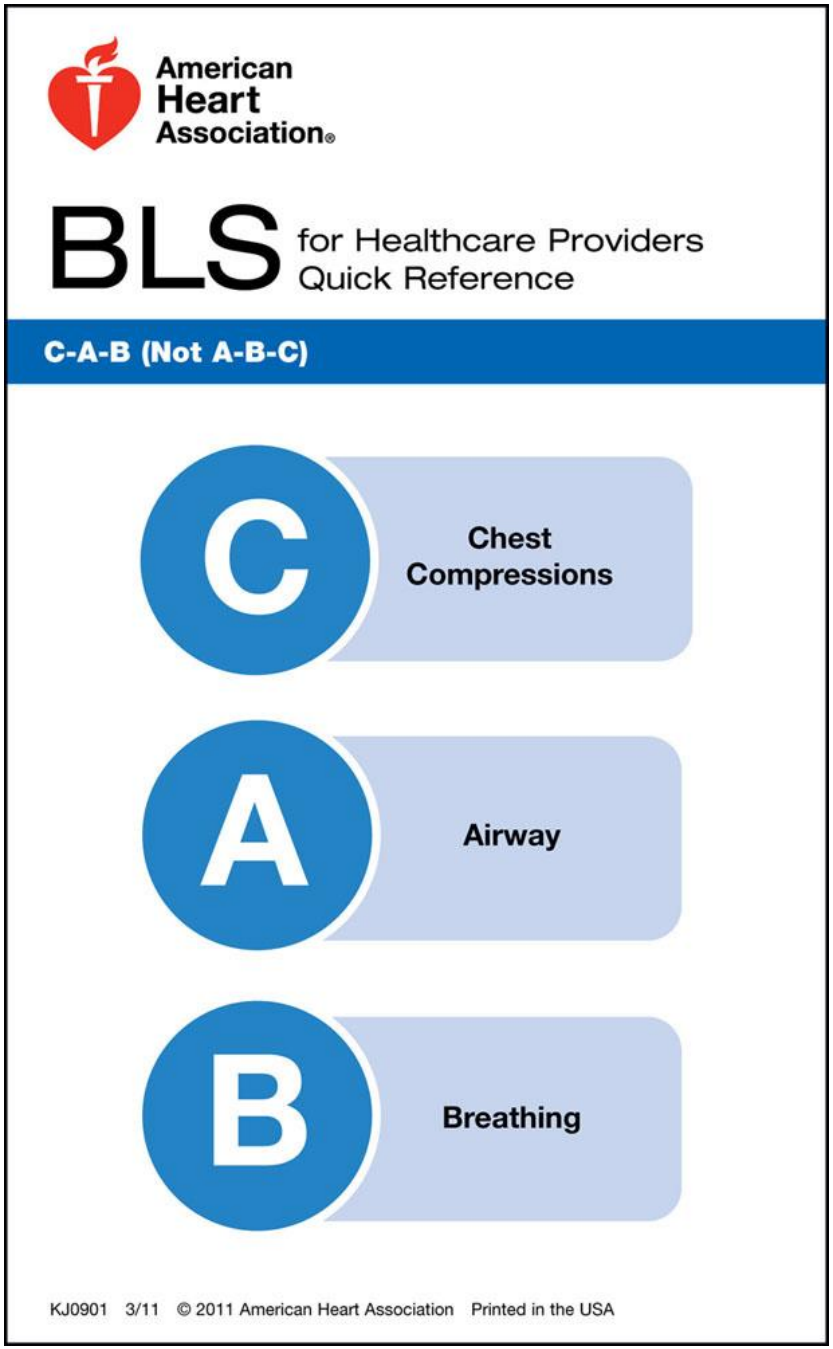
Recovery position
2nd stage

Recovery position
3rd stage



Recovery position





If the person is <u>unconscious</u>:

1. Call for help
2. Assess blood circulation, airway patency, breathing during *10 seconds*:

one hand on a chest of a victim, 2 fingers of another hand in carotic triangle, ear listens to the breathing, eyes watch chest movements

Not breathing normally?

- Open airways – assess the state of the victim
- Still not breathing normally:
  - send someone for help and AED,
  - start CPR with chest compressions!

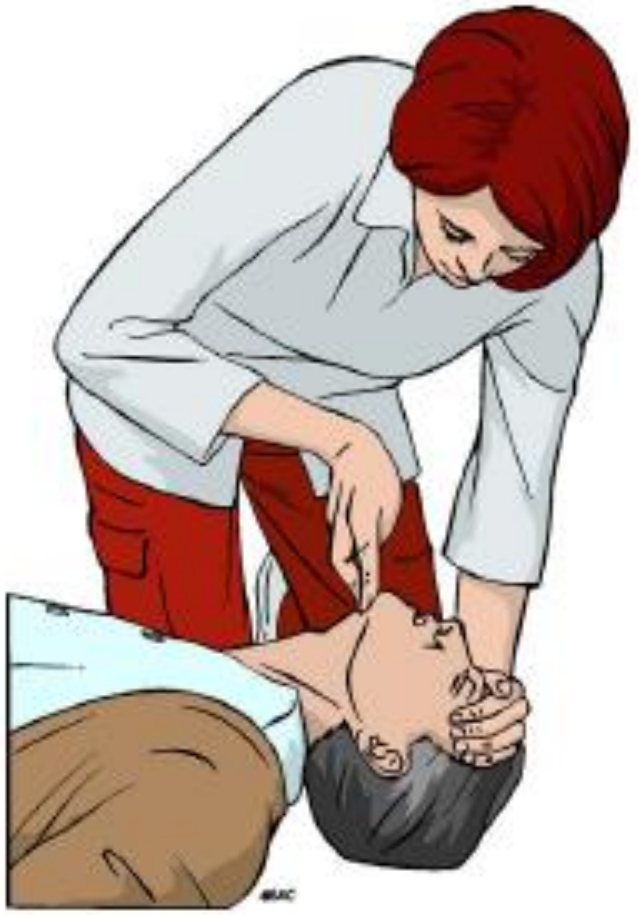


Fig. 2.5. Head tilt and chin lift.

Fig. 2.7. Place the heel of one hand in the centre of the victim's chest.

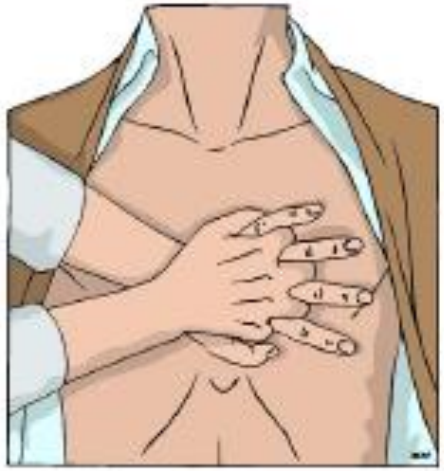


Fig. 2.8. Place the heel of your other hand on top of the first hand.

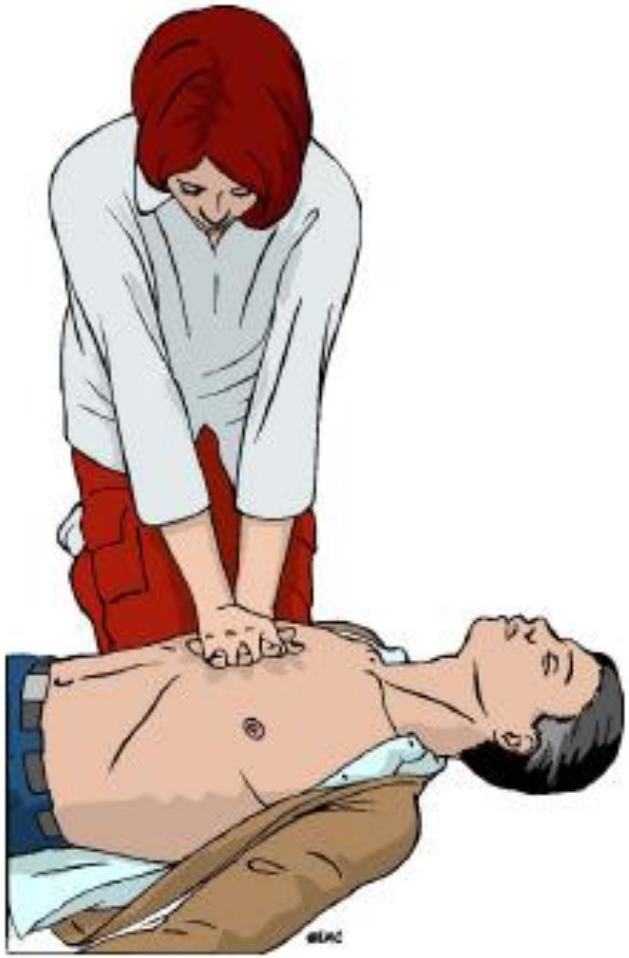


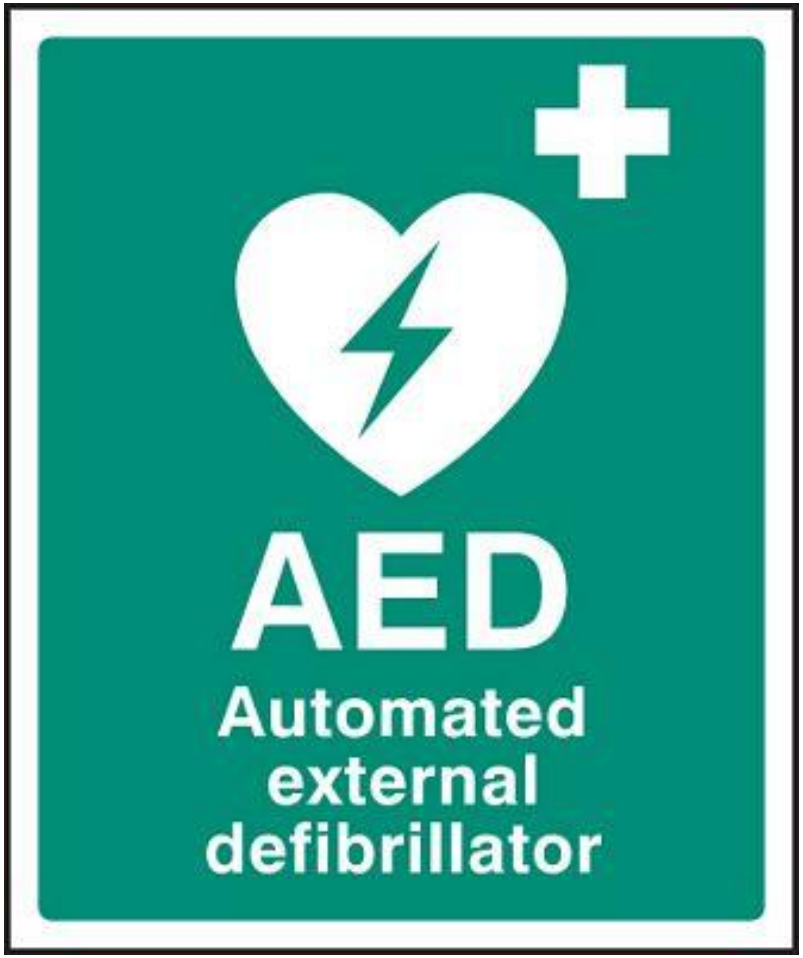
Fig. 2.9. Interlock the fingers of your hands. Keep your arms straight.

Fig. 2.10. Press down on the sternum at least 5 cm.

Numbers

- 30 compressions : 2 breaths
- 100-120 compressions per minute
- 5-6 cm depth of compressions
- 2 breaths during 5 seconds
- Assess of breathing and circulation every 2 minutes

Automated external defibrillator



AED

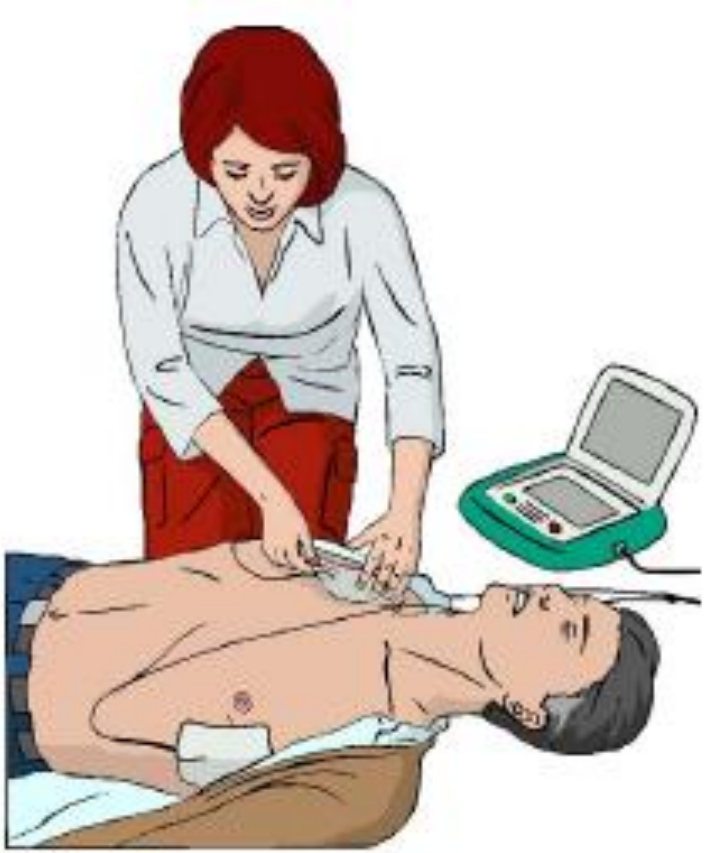


Fig. 2.19. Attaching the electrode pads. Place the first electrode pad in the mid-axillary line just below the armpit. Place the second electrode just below the right collarbone (clavicle). © 2010 ERC.

1. Switch on AED
2. Attach the electrodes to the chest of the victim
3. Follow the spoken/visual directions
4. Be sure that nobody touches the patient while the AED analyzes

AED

- 5. Ensure that nobody touches the victim
- 6. Push a shock button as directed
- 7. Immediately restart CPR
- 8. Follow the further instructions

Fig. 2.21. When the shock button is pressed, make sure that nobody touches the victim. © 2010 ERC.

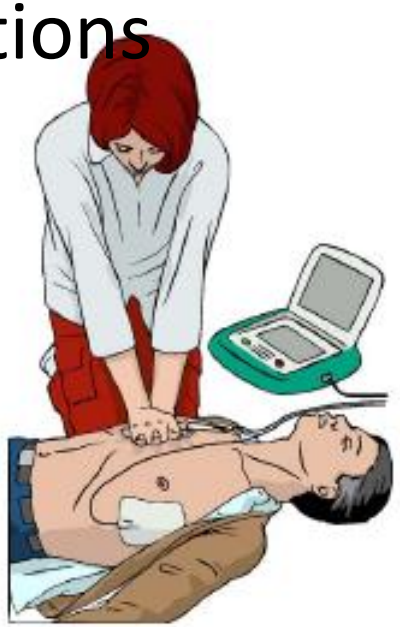
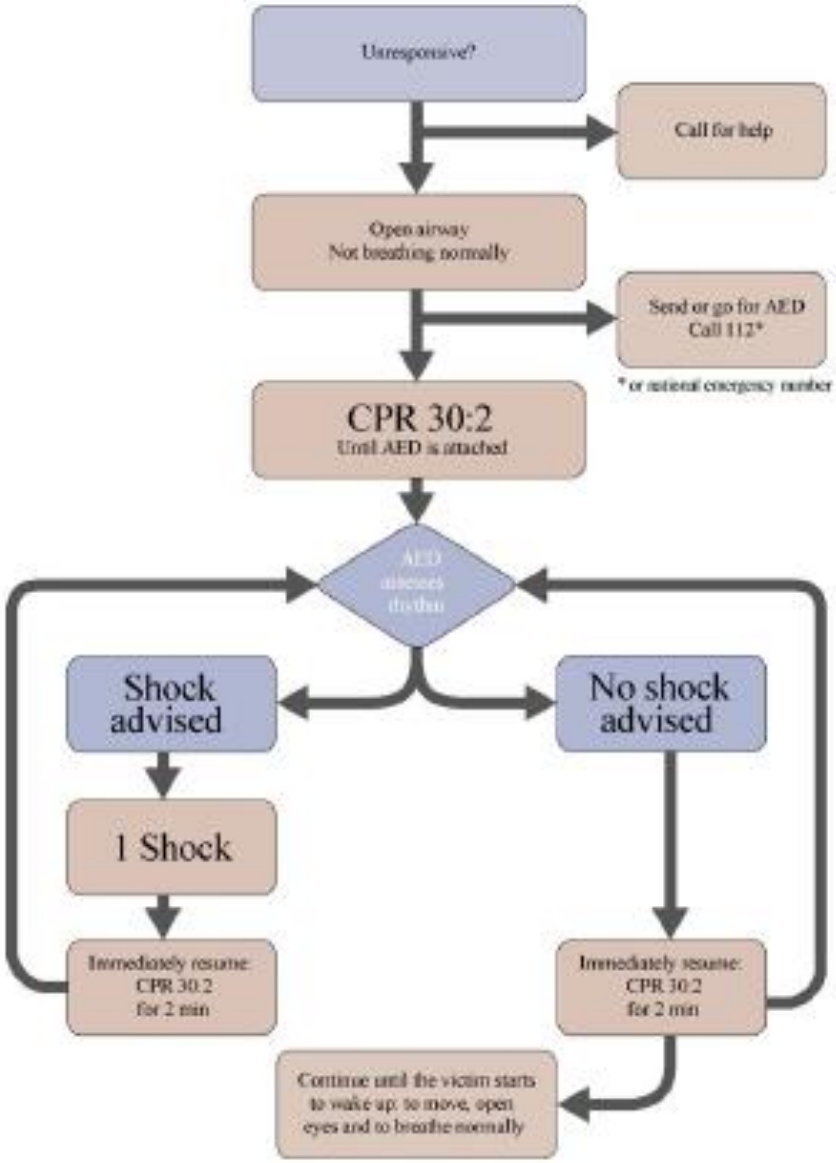


Fig. 2.22. After the shock the AED will prompt you to start CPR. Do not wait—start CPR immediately and alternate 30 chest compressions with 2 rescue breaths. © 2010 ERC.

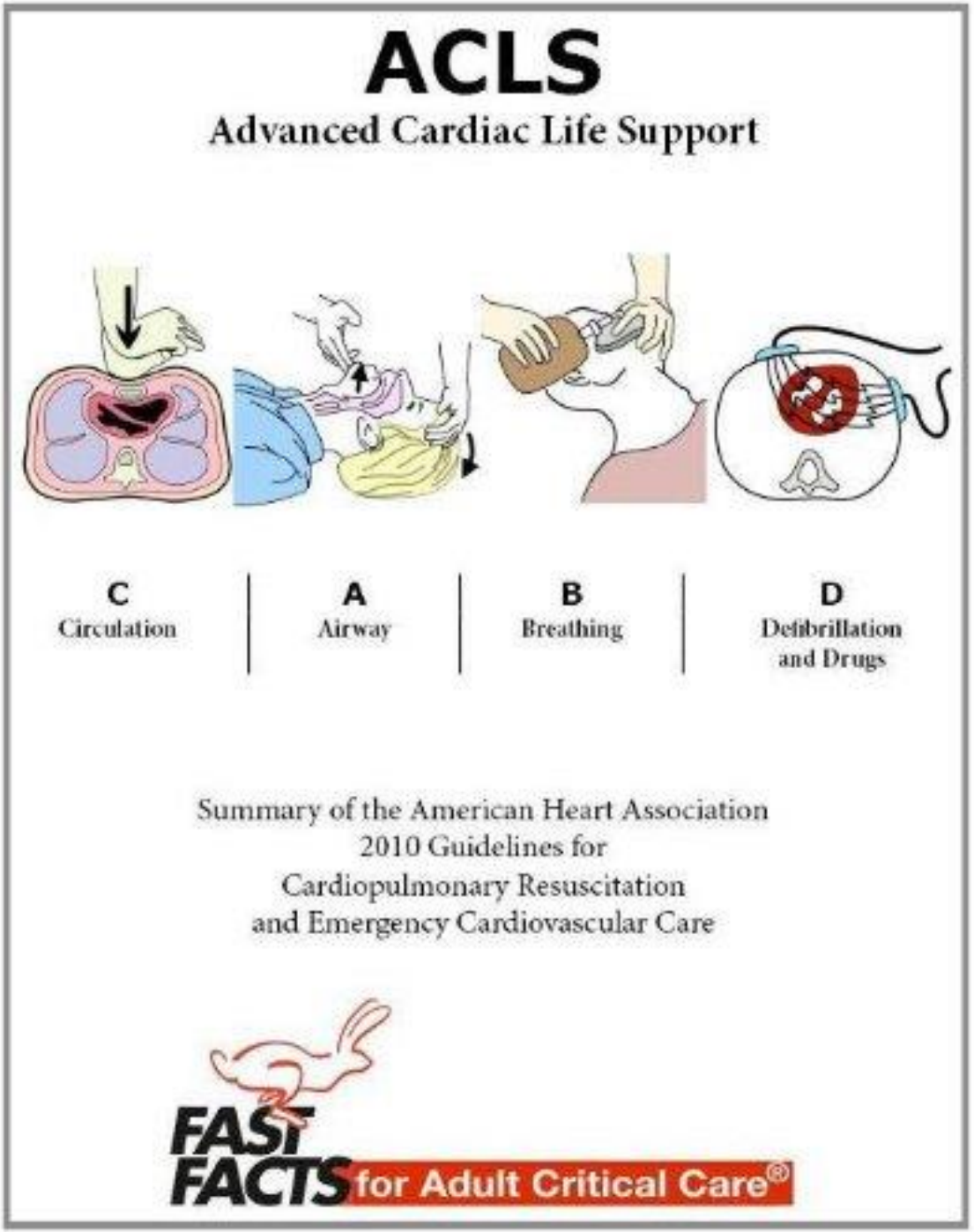
Automated External Defibrillation Algorithm



- Shock is delivered 3 times
- The maximum is 4 J per kg
- First shock is 360 J

Do not interrupt resuscitation until:

- Professional help arrives and takes over
- Victim wakes up: opens eyes, starts to breath, coughs, etc.
- You become exhausted



ACLS

- In-hospital resuscitation's advantages:
  - Immediate cardiac arrest recognition
  - CPR within 3 minutes
  - Help is called using standard # and codes
  - Fast arrival of the resuscitation team.

Algorithm

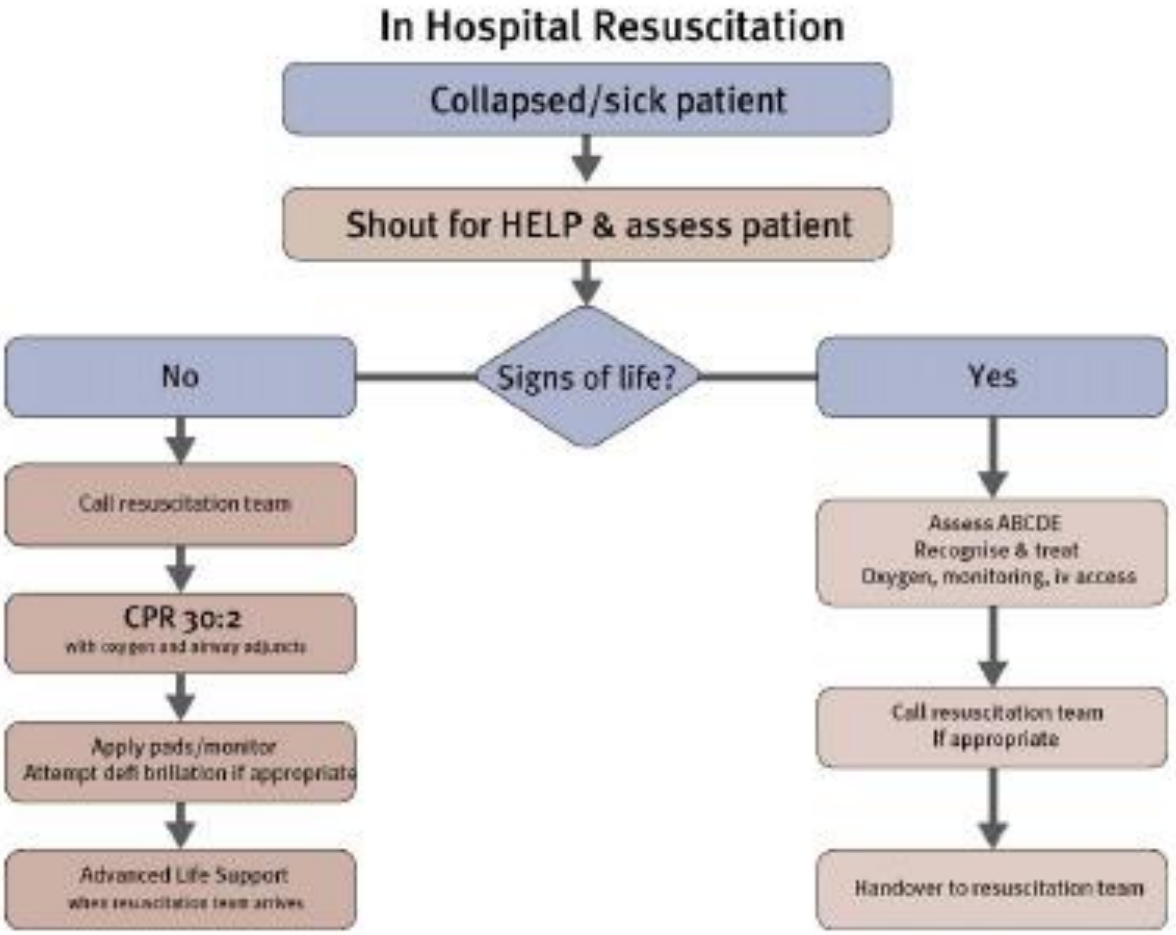


Fig. 4.1. Algorithm for the treatment of in-hospital cardiac arrest. © 2010 ERC.

When the team arrives!

- All necessary equipment arrives too
- Installation of cardiomonitor electrodes
- Availability to assess “shock able” - “non-shock able” rhythms
- Availability to provide airway patency, ventilation with advanced devices
- Tracheal intubation if necessary (10 pumps per minute without synchronizing with the compressions)
- Intravenous access provided

They follow **CABD** model of the ACLS survey

So when ambulance brigade arrives what happens? what do they do?

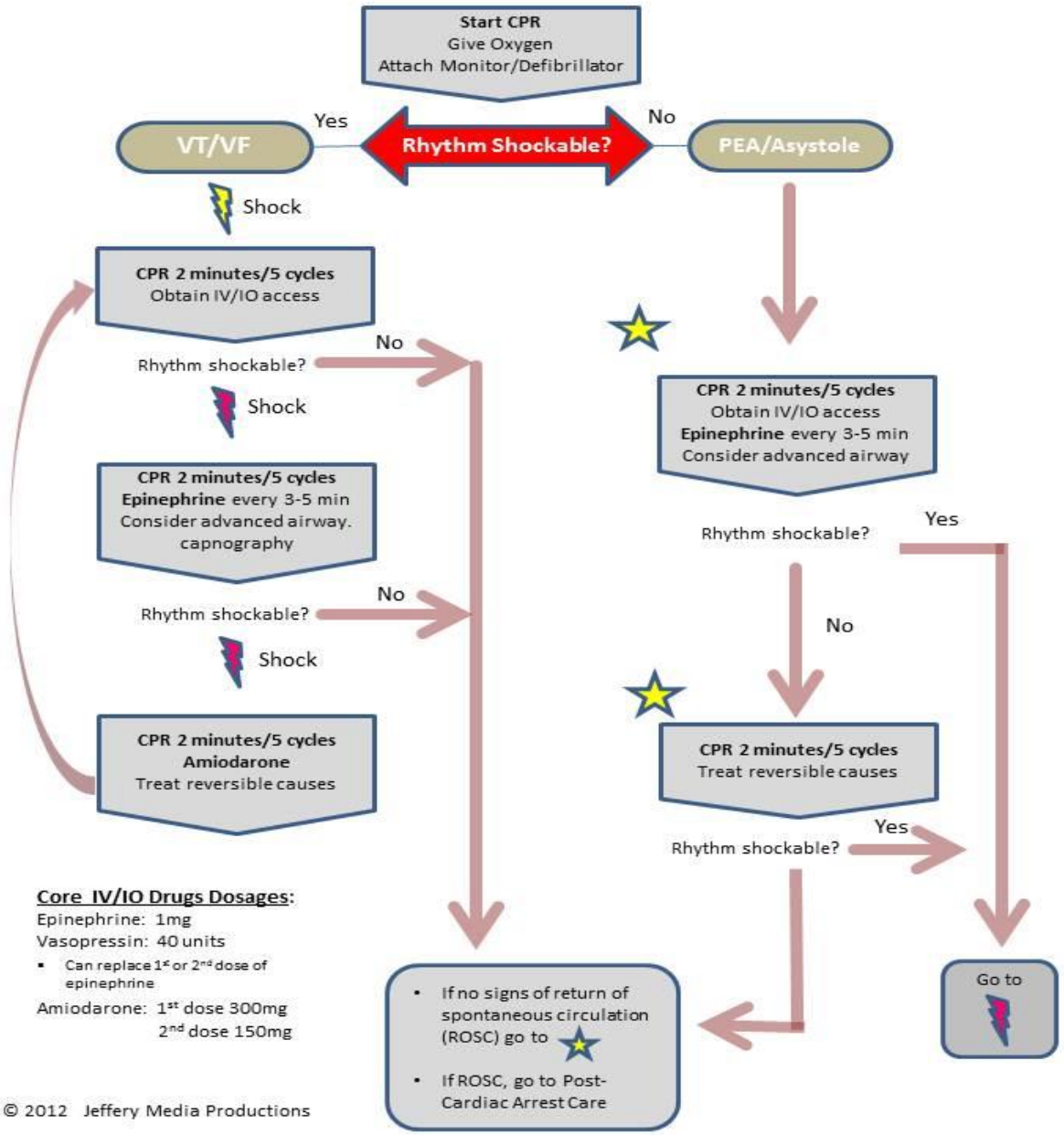
- Take over chest compressions (C)
- Intubate the patient and use quantitative waveform capnography if available (A)
- Connect the oxygen (B)
- Fix pulseoxymeter (B)
- Provide IV or IO access (C)

So when ambulance brigade arrives what happens? what do they do?

- Take blood tests (Total blood account, biochemistry BT) if needed
- Give fluids if necessary
- If AED has been used during BLS, brigade continues to use it. If not they attach ECG patches, identify and monitor arrhythmias and go on according to the following algorithm:

AHA ACLS Adult Cardiac Arrest Algorithm

Shout for Help/Activate Emergency Response



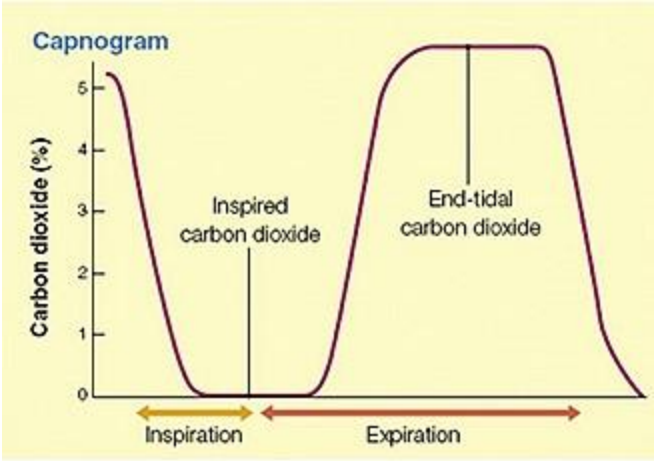
So when ambulance brigade arrives what happens? what do they do?

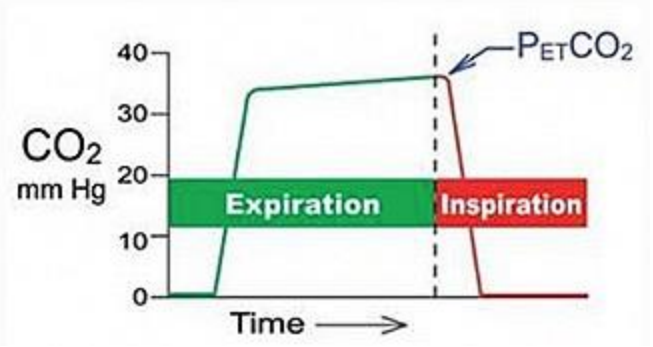
- Differential diagnosis (D) – look for reversible causes and contributing factors for emergency (H's and T's).

- NB! Also, "D" stands for "DRUGS"

What is the Quantitative waveform capnography?

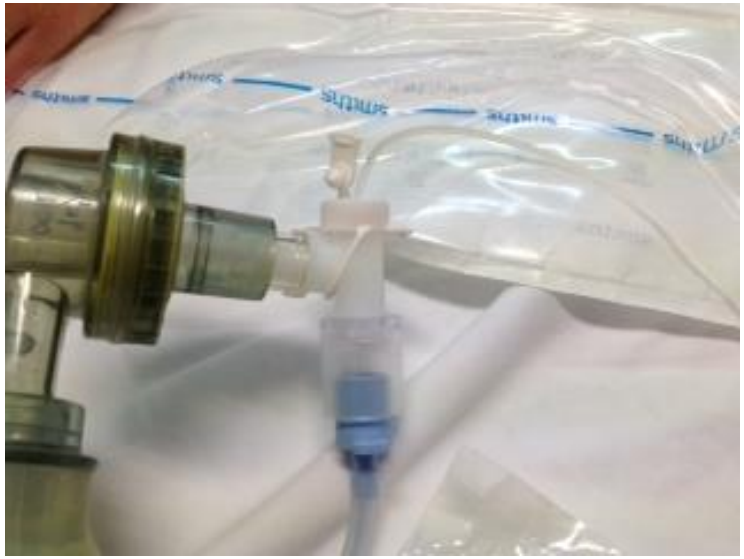
- is the continuous, noninvasive measurement and graphical display of end-tidal carbon dioxide/ETCO2 (also called PetCO2). It uses a sensor placed for optimum evaluation of expired CO2. The inhaled and exhaled carbon dioxide is graphically displayed as a waveform on the monitor along with its corresponding numerical measurement.

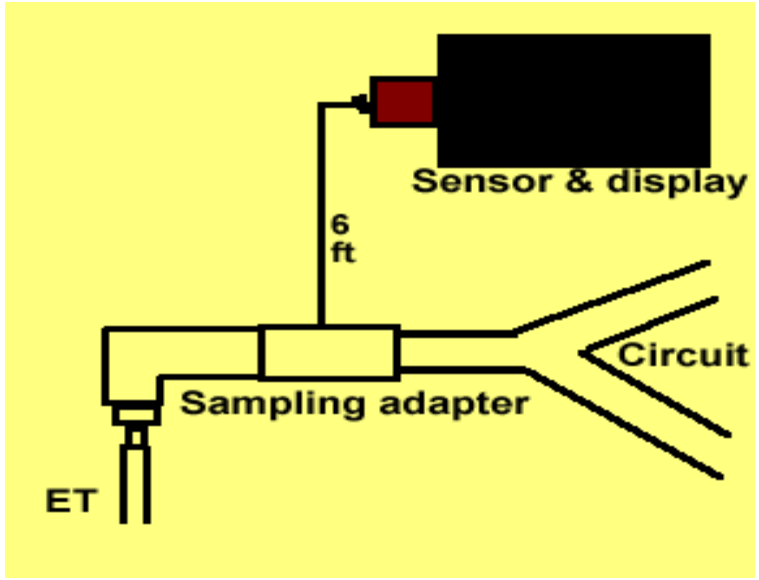




Quantitative waveform capnography

- It is used in intubated patients during CPR.
- allows providers to monitor CPR quality
- optimizes chest compressions,
- and detects ROSC (return of spontaneous circulation) during chest compressions.





VIDEO

Numbers

- Normal ETCO2 in the adult patient should be 35-45 mmHg
- High quality chest compressions are achieved when the ETCO2 value is at least 10-20 mmHg
- If there will be a significant increase in the ETCO2 to 35-45 mmHg it indicates return of spontaneous circulation!

Differential diagnosis

Table 1. Treatable Causes of Cardiac Arrest: The H's and T's

H's	T's
Hypoxia	Toxins
Hypovolemia	Tamponade (cardiac)
Hydrogen ion (acidosis)	Tension pneumothorax
Hypo-/hyperkalemia	Thrombosis, pulmonary
Hypothermia	Thrombosis, coronary

For further explanation of the H's and T's, see Part 12: "Special Resuscitation Situations."

Hypoxia

or deprivation of adequate oxygen supply

What to do?

- Ensure that the patient's airway is open, and that the patient has chest rise and fall and bilateral breath sounds with ventilation.
- Also ensure that your oxygen source is connected properly.
- Provide airway management if necessary

Hypovolemia

or the loss of fluid volume in the circulatory system

What to do?

- Look for obvious blood loss in the patient with cardiac arrest is the first step
- After CPR, the most import intervention is obtaining intravenous access/IO access.
- A fluid bolus will also help determine if the arrest is related to hypovolemia.

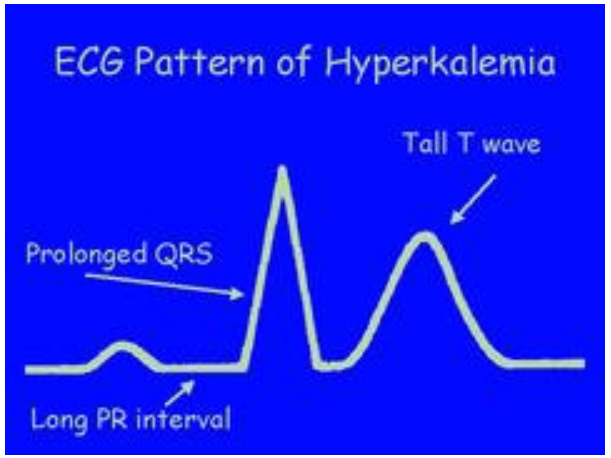
Hydrogen ion (acidosis)

What to do?

- To evaluate an arterial blood gas to identify acidosis
- Provide adequate ventilation to prevent respiratory acidosis
- Give the patient *sodium bicarbonate* to prevent metabolic acidosis

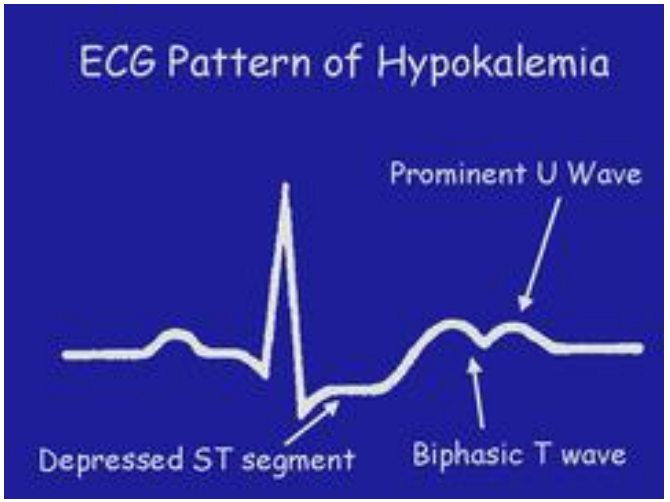
Hyper-/hypokalemia

- The major sign of hyperkalemia is taller and peaked T-waves. Also, a widening of the QRS-wave may be seen.
- Can be treated with sodium bicarbonate (IV) or glucose+insulin, or calcium chloride (IV), or Kayexalate, or dialysis, and possibly salbutamolum.
- All of these will help reduce serum potassium levels.



Hyper-/hypokalemia

- The major signs of hypokalemia are flattened T-waves, prominent U-waves, and possibly a widened QRS complex.
- Treatment of hypokalemia involves rapid but controlled infusion of potassium.
- NB! Giving IV potassium has risks. Never give undiluted intravenous potassium.

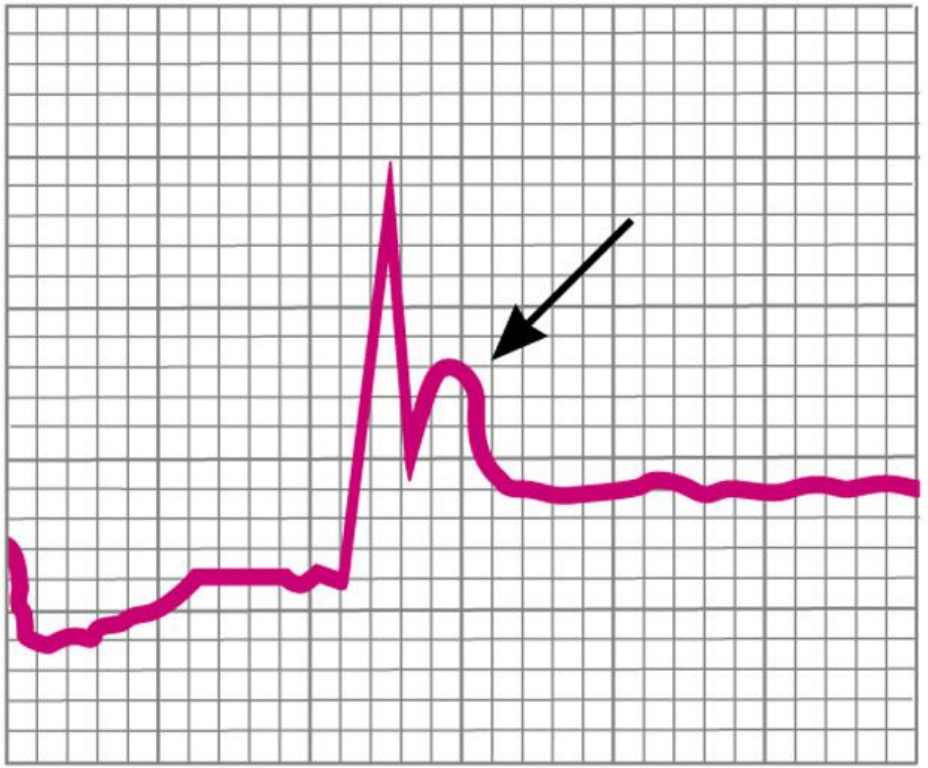


Hypothermia

- The hypothermic patient may be unresponsive to drug therapy and electrical therapy (defibrillation or pacing).
- If a patient has been exposed to the cold, warming measures should be taken.
- Core temperature should be raised above 86 F (30 C) as soon as possible.

ECG Changes Due to Electrolyte Abnormalities and Hypothermia

- Hyperkalemia
  - Tall Ts
    - Suspect in patients with a history of renal failure.
- Hypokalemia
  - Prominent U waves
- Hypothermia
  - Osborn wave (“J” wave)
  - T wave inversion, sinus bradycardia, atrial fibrillation or flutter, AV blocks, PVCs, VF, asystole



Toxins

- Accidental overdose of medications (tricyclics, digoxin, betablockers, and calcium channel blockers), street drugs (cocaine) and other chemicals can cause cardiac arrest.
- ECG signs are: prolongation of the QT interval.
- Physical signs include bradycardia, pupil symptoms, and other neurological changes.

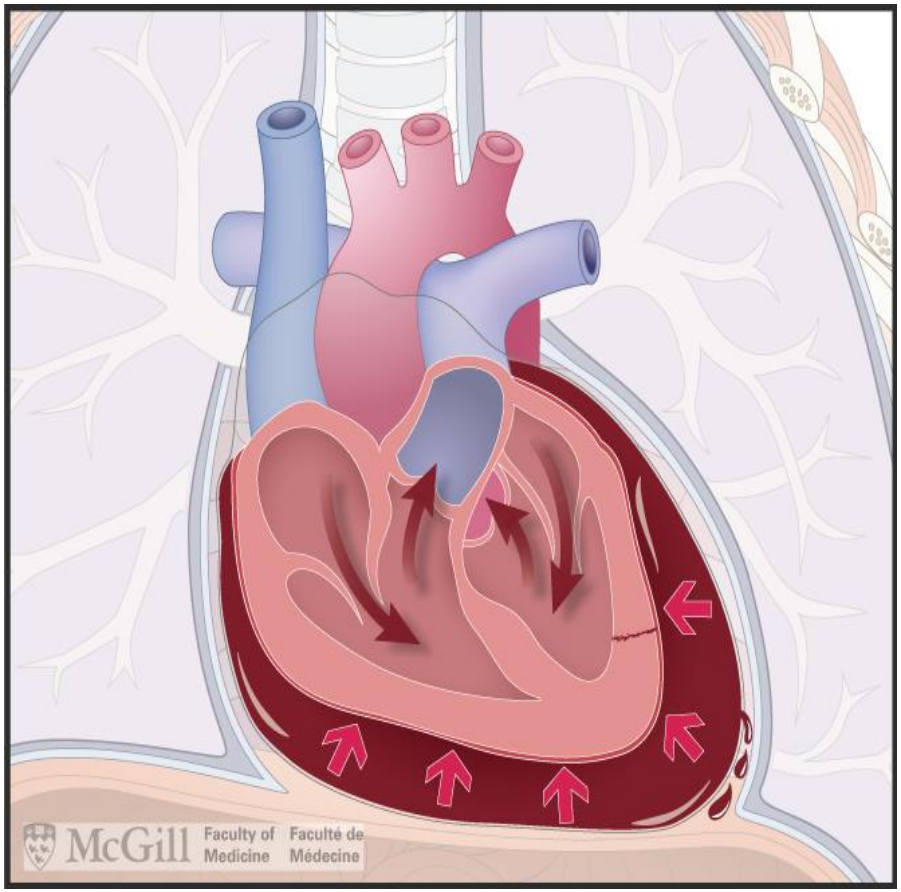
Toxins

What to do?

- Support of circulation while an antidote or reversing agent is obtained is of primary importance.
- Poison control can be utilized to obtain information about toxins and reversing agents.

Tamponade

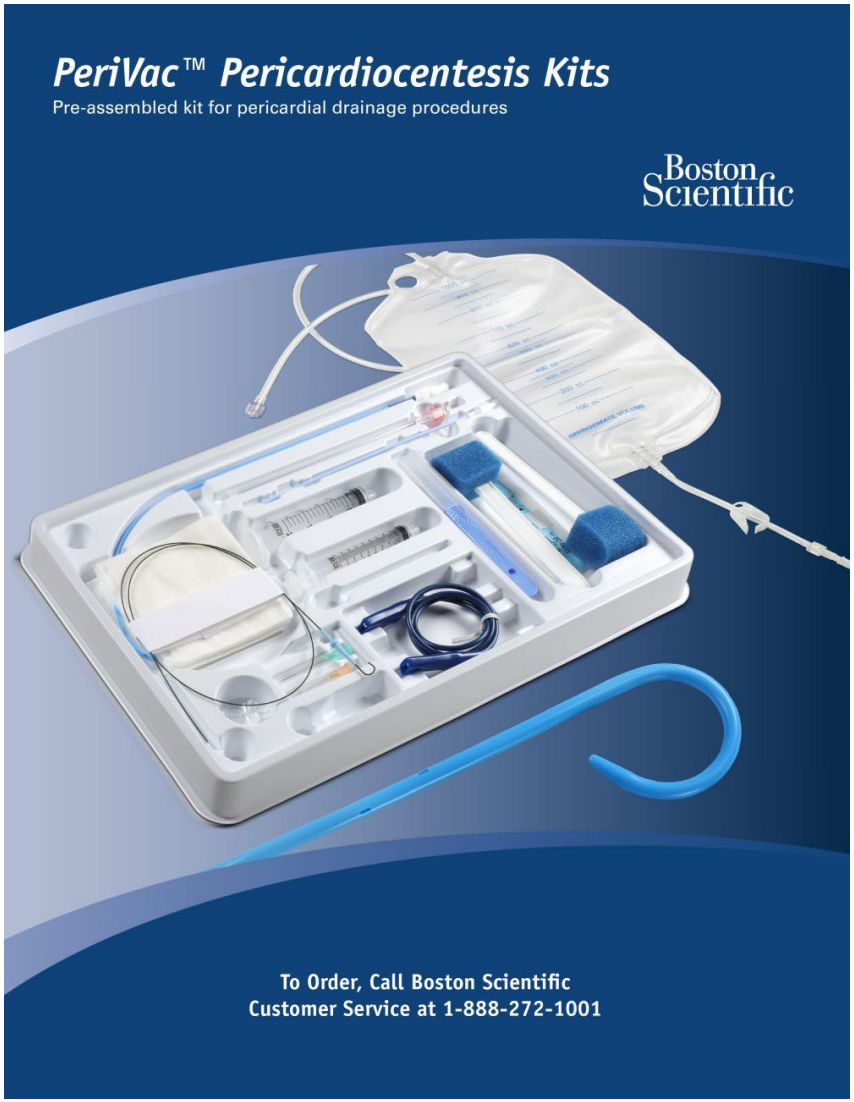
fluid accumulates in the pericardium



- The buildup of fluid results in ineffective pumping of the heart
- ECG symptoms - low QRS complex and rapid heart rate.
- *Beck's triad*- jugular vein distention, low blood pressure, and muffled heart sounds due to fluid inside the pericardium.

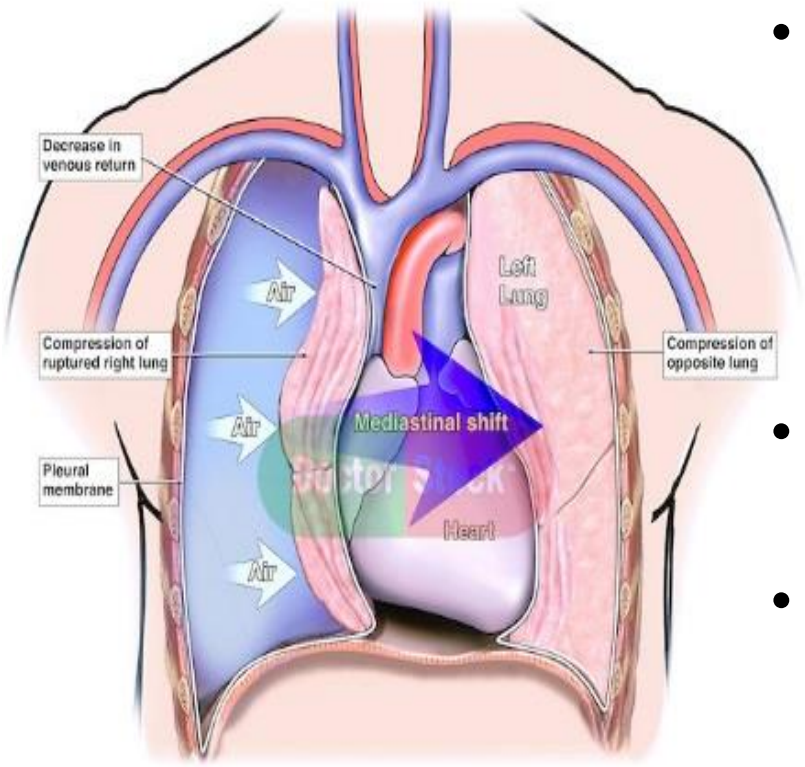
Tamponade

- The recommended treatment for cardiac tamponade is pericardiocentesis.
- *VIDEO "PCC", "PCC ATLS"*



Tension Pneumothorax

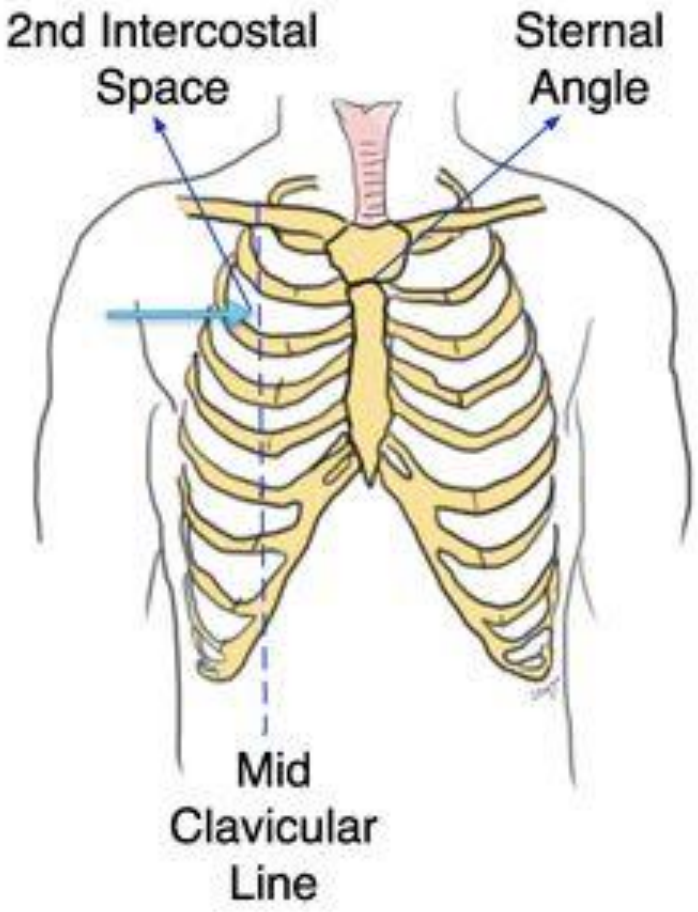
occurs when air enters the plural space and is not able to escape naturally.



In a tension pneumothorax, air from a ruptured lung enters the pleural cavity without a means of escape. As air pressure builds up, the affected lung is compressed and all of the mediastinal tissues are displaced to the opposite side of the chest.

- This leads to a build up of tension that causes shifts in the intrathroacic structure that can rapidly lead to cardiovascular collapse and death.
- Can easily be found clinically or with the help of chest X-ray
- *Physical signs* are respiratory distress, chest pain, JVD, tracheal deviation, unequal breath sounds, difficulty with ventilation, and no pulse felt with CPR.

Tension Pneumothorax



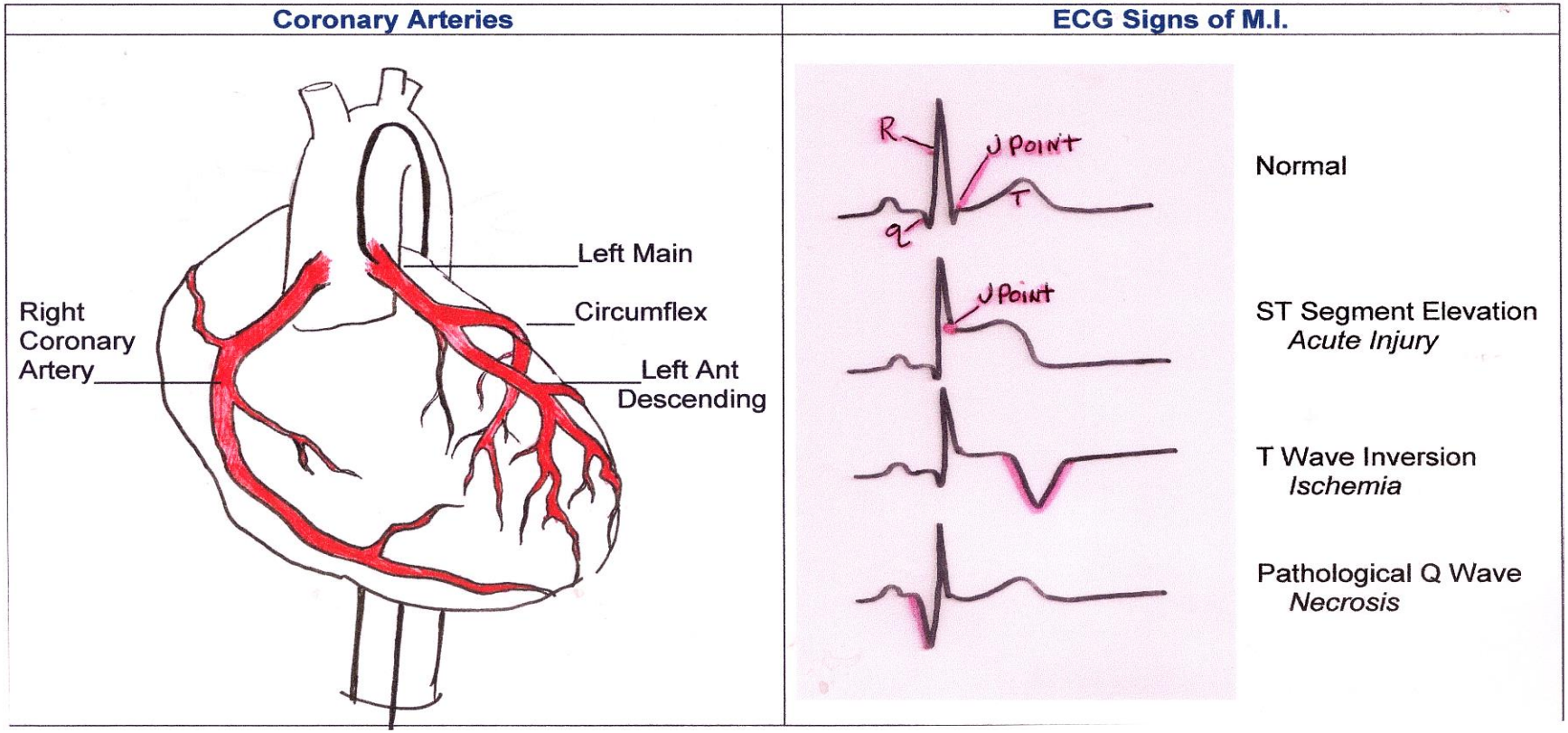
- Treatment of tension pneumothorax is needle decompression.
- *VIDEO "Decompressing A Tension Pneumothorax"*

Thrombosis (heart: acute, massive MI)

- **Coronary thrombosis** is an occlusion or blockage of blood flow within a coronary artery caused by clot within the vessel.
- The clotted blood causes an ***acute myocardial infarction*** (acute ischemia) which destroys heart muscle and can lead to sudden death depending on the location of the blockage.

Thrombosis (heart: acute, massive MI)

- ECG signs indicating coronary thrombosis include ST-segment changes, T-wave inversions, and/or Q waves.



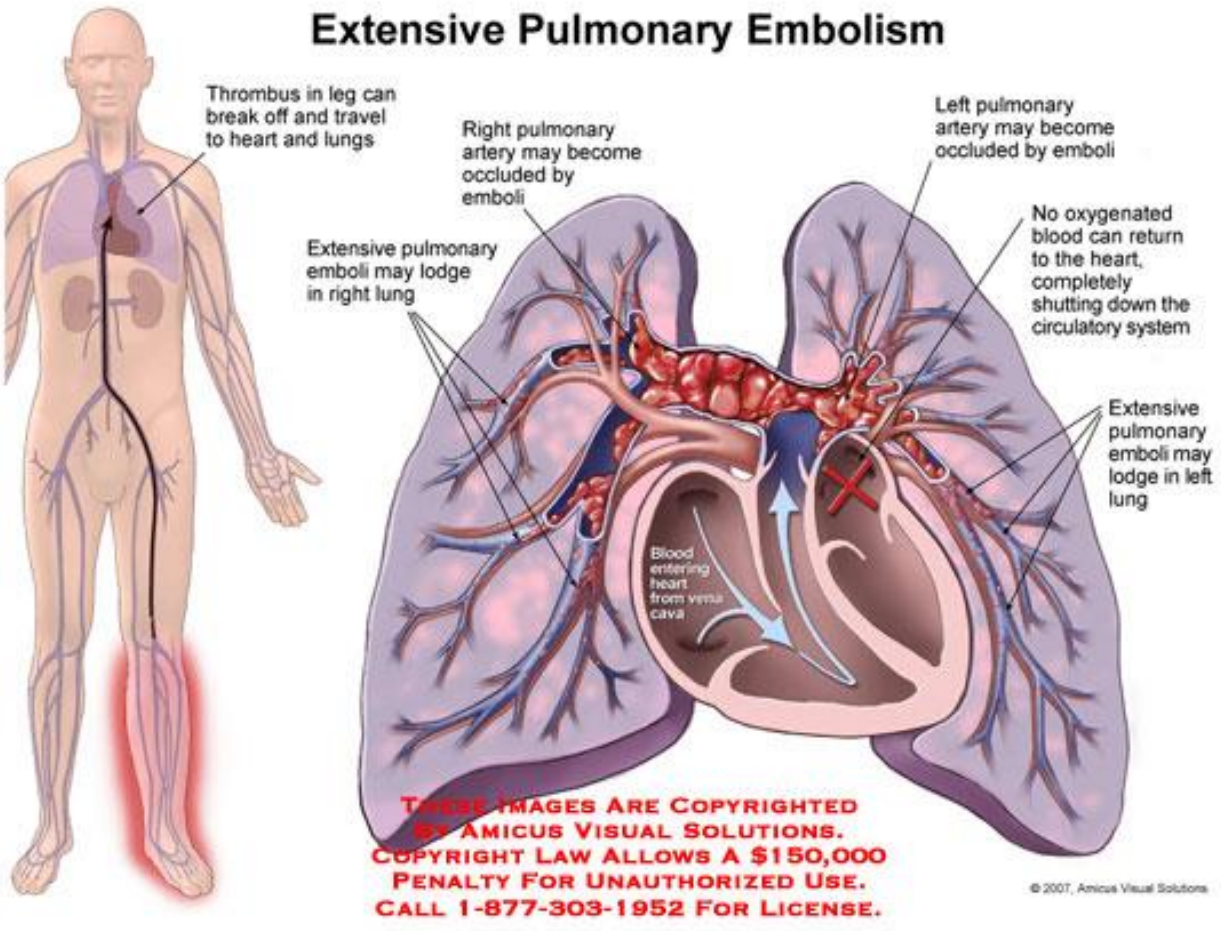
Thrombosis (heart: acute, massive MI)

- *Physical signs*: chest pain over 5 minutes, which irradiates to the left hand, jaw; resistant to nitrates, ect.
- *Lab findings*: elevated cardiac markers (troponins) on lab test.
- For patients with cardiac arrest and without known pulmonary embolism (PE), *routine fibrinolytic treatment given during CPR has shown no benefit and is not recommended*.

Thrombosis (heart: acute, massive MI)

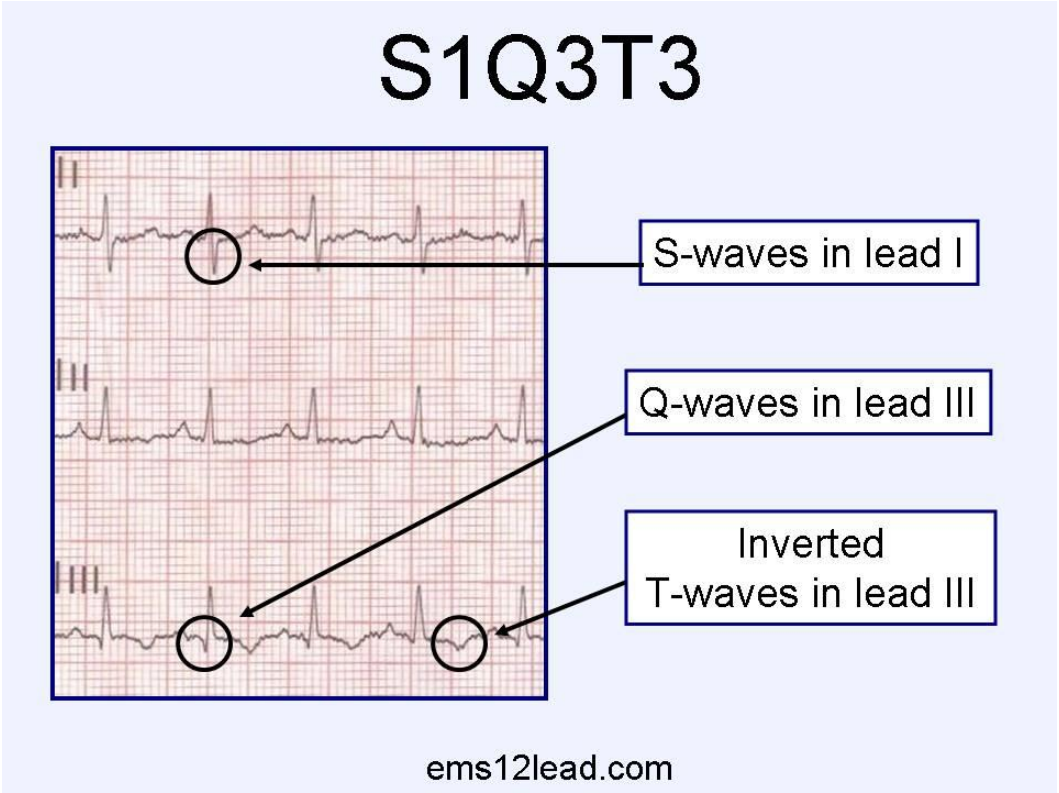
- Treatments for coronary thrombosis **before** cardiac arrest include use of fibrinolytic therapy, or percutaneous coronary intervention.
- The most common percutaneous coronary intervention procedure is **coronary angioplasty** with or without stent placement.

Thrombosis (lungs: massive pulmonary embolism)



- Pulmonary thrombosis or **<u>pulmonary embolism</u>** (PE) is a blockage of the main artery of the lung which can rapidly lead to respiratory collapse and sudden death.

Thrombosis (lungs: massive pulmonary embolism)



- ECG signs of PE include the classic signs are
  - a large S wave in lead I,
  - a large Q wave in lead III
  - and an inverted T-wave in lead III **(S1Q3T3)**

Thrombosis (lungs: massive pulmonary embolism)

- *Physical signs* – shortness of breath, chest pain worsened by breathing, coughing with blood, growing cyanosis, collapse, sudden death.
- *Lab findings*: positive D-dimer test, prior positive test for DVT or PE.
- *Treatment* includes surgical intervention (pulmonary thrombectomy) and fibrinolytic therapy.

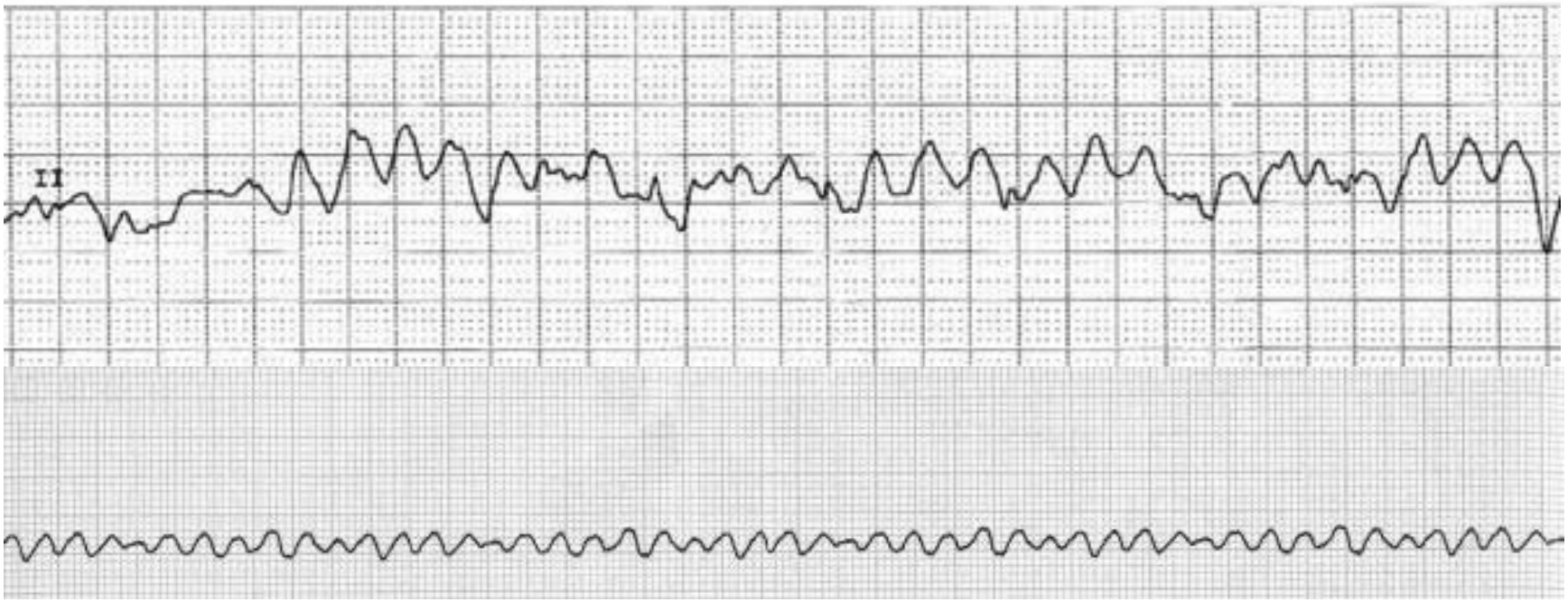
What rhythms give us absences of pulse?

- **Pulseless Ventricular Tachycardia**
- **Ventricular Fibrillation**
- **Pulseless Electrical Activity**
- **Asystole**

Pulseless rhythms

- 1. **Shockable**
  - Ventricular fibrillation
  - Pulseless ventricular tachycardia
- 2. **Non-shockable**
  - Pulseless electrical activity (PEA)
  - Asystole

Ventricular Fibrillation

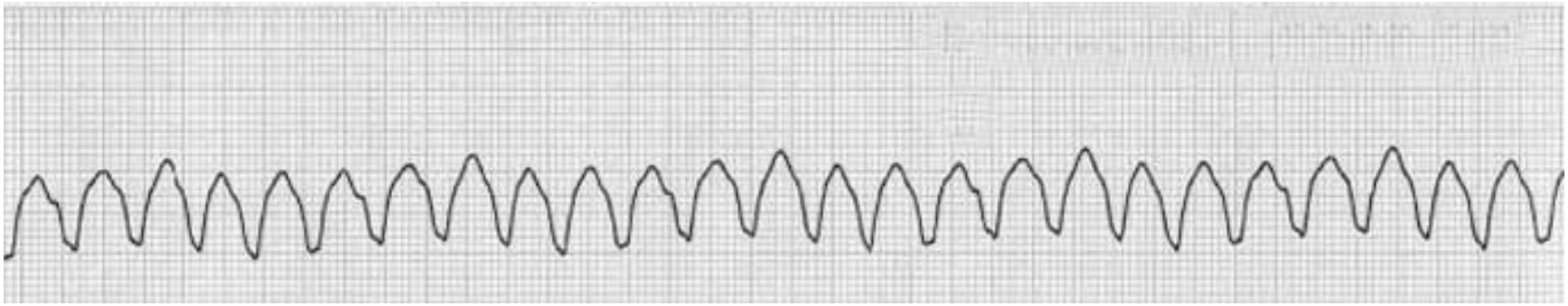


- Occurs when there are uncoordinated contractions within the ventricles of the heart.
- As a result, multiple muscles cells within the ventricles simultaneously fire as pacemakers causing a fibrillation that is ineffective for adequate cardiac output.

Ventricular Fibrillation

- **Total unorganized multifocal rhythm**
- **Ventricular quiver**
- **NO CARDIAC OUTPUT**

Pulseless Ventricular Tachycardia



- Pulselessness with a tachyarrhythmia occurs because the ventricles are not effectively moving blood out of the heart and there is therefore no cardiac output

Pulseless Ventricular Tachycardia

- the rate is greater than 180 beats per minute
- 3 or more Premature ventricular contraction's
- QRS is wide and bizarre, looks like sinusoid
- the patient will be pulseless
- the rhythm originates in the ventricles

SCREAM for VF and Pulseless VT

1. **S**hock360J* monophasic, 1st and subsequent shocks.(Shock every 2 minutes if indicated)
2. **CPR** After shock, immediately begin chest compressions followed by respirations (30:2 ratio) for 2 minutes.
3. **R**hythm check after 2 minutes of CPR (and after every 2 minutes of CPR thereafter) and shock again if indicated**.** Check pulse only if an organized or non-shockable rhythm is present.

SCREAM

Epinephrine1 mg IV/IO q3-5 min. Or vasopressin 40 U IV/IO, once, in place of the 1st or 2nd dose of epi.

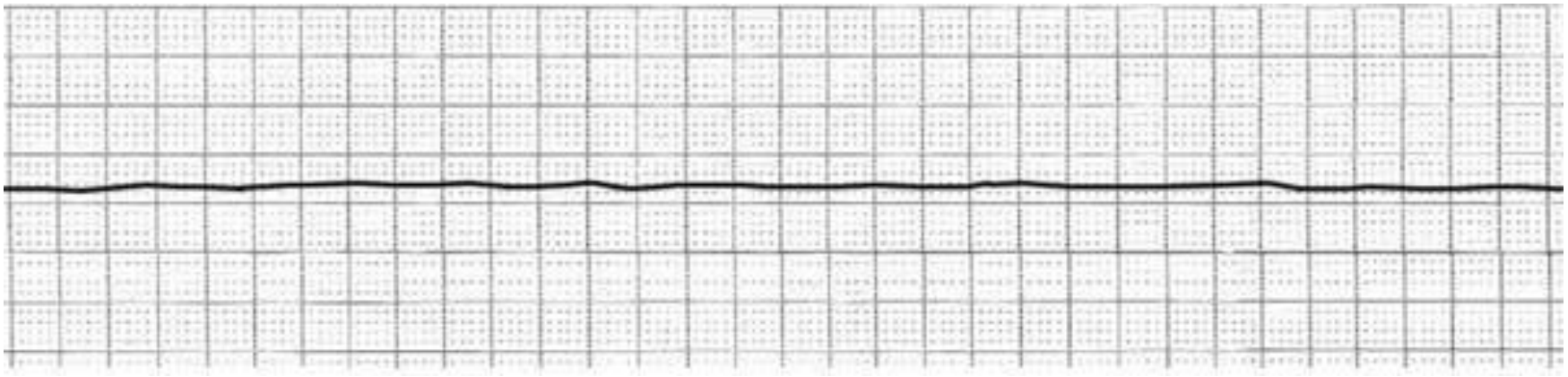
ANTIARRHYTHMIC MEDICATIONS:

Amiodarone 300mg IV/IO, may repeat once at 150mg in 3-5 min. if VF/PVT persists or

Lidocaine (if amiodarone unavailable) 1.0-1.5 mg/kg IV/IO, may repeat X 2, q5-10 min. at 0.5-0.75 mg/kg, (3mg/kg max. loading dose) if VF/PVT persists,or

Magnesium Sulfate 1-2 g IV/IO diluted in 10mL D5W (5-20 min. push) for suspected/ known hypomagnesemia

Asystole or “flatline”



- is a state of no cardiac electrical activity

Asystole

- No blood flow to the brain and other vital organs. This results in very poor outcomes if resuscitation is successful.
- ensure that all leads are connected properly. If all leads are OK, you should rapidly assess for any underlying causes for the asystole, as with pulseless electrical activity (PEA).

Pulseless Electrical Activity (PEA) Rhythm

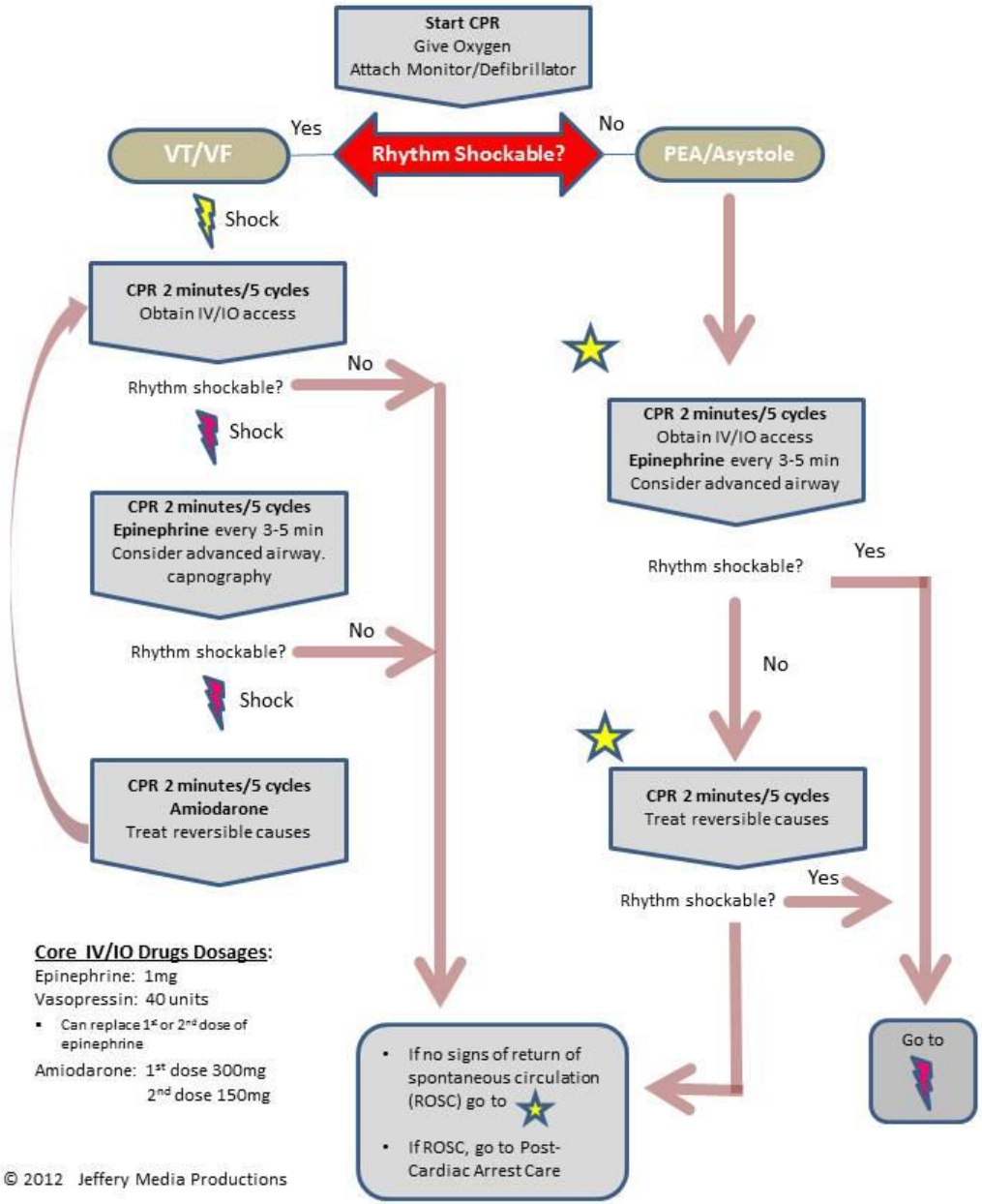
- PEA rhythm occurs when any heart rhythm that is observed on the electrocardiogram (ECG) does not produce a pulse.
- PEA can come in many different forms:
  - Sinus Rhythm,
  - tachycardia,
  - bradycardia can all be seen with PEA.

Pulseless Electrical Activity (PEA) Rhythm

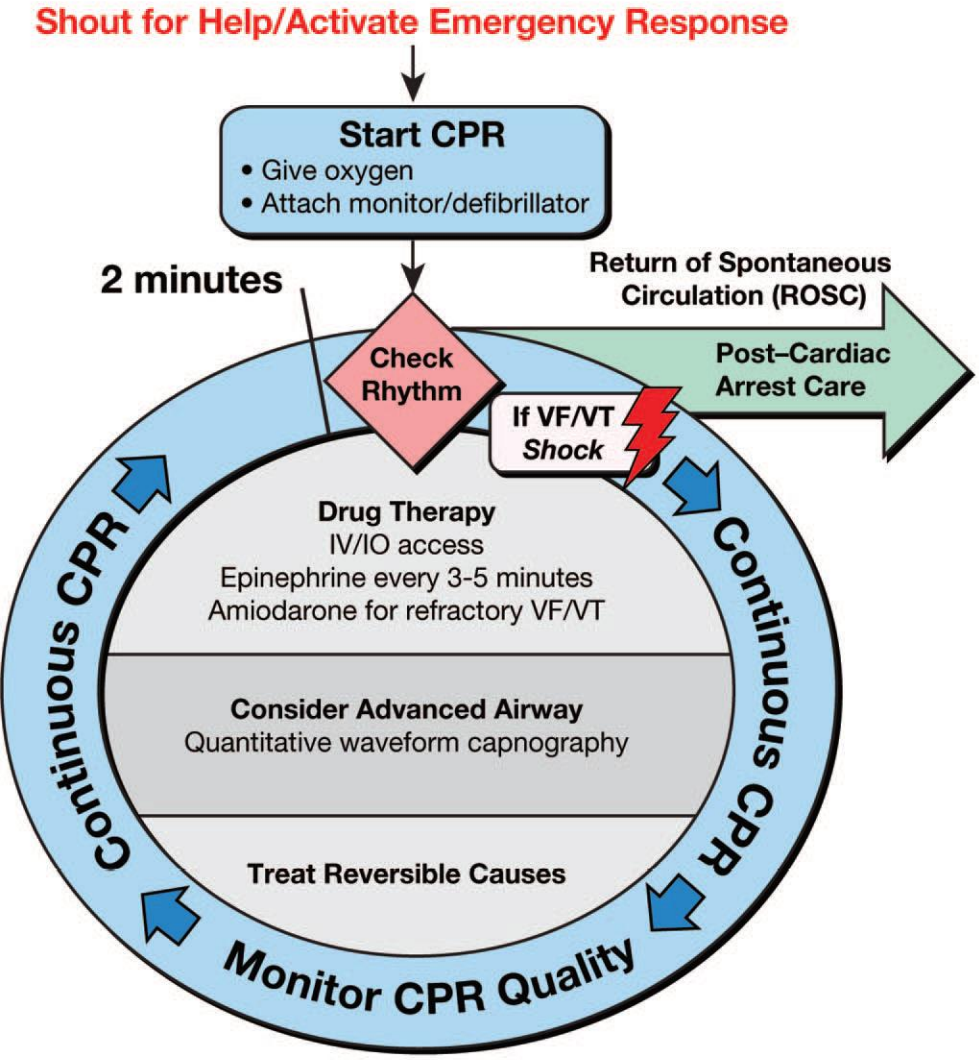
- Check pulse after a rhythm/monitor check
- PEA has an underlying treatable cause.
- The most common cause is **hypovolemia**.
- PEA is treated by assessing and correcting the underlying cause. They are summed up in the H's and T's of ACLS.
- When the cause for PEA cannot be determined, it should be treated in the same way as asystole

AHA ACLS Adult Cardiac Arrest Algorithm

Shout for Help/Activate Emergency Response



Adult Cardiac Arrest



CPR Quality
- Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
  - If P_{ETCO_2} <10 mm Hg, attempt to improve CPR quality
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality

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

Shock Energy
- **Biphasic:** Manufacturer recommendation (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
- **Vasopressin IV/IO Dose:** 40 units can replace first or second dose of epinephrine
- **Amiodarone IV/IO Dose:** First dose: 300 mg bolus. Second dose: 150 mg.

Advanced Airway
- Supraglottic advanced airway or endotracheal intubation
- Waveform capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

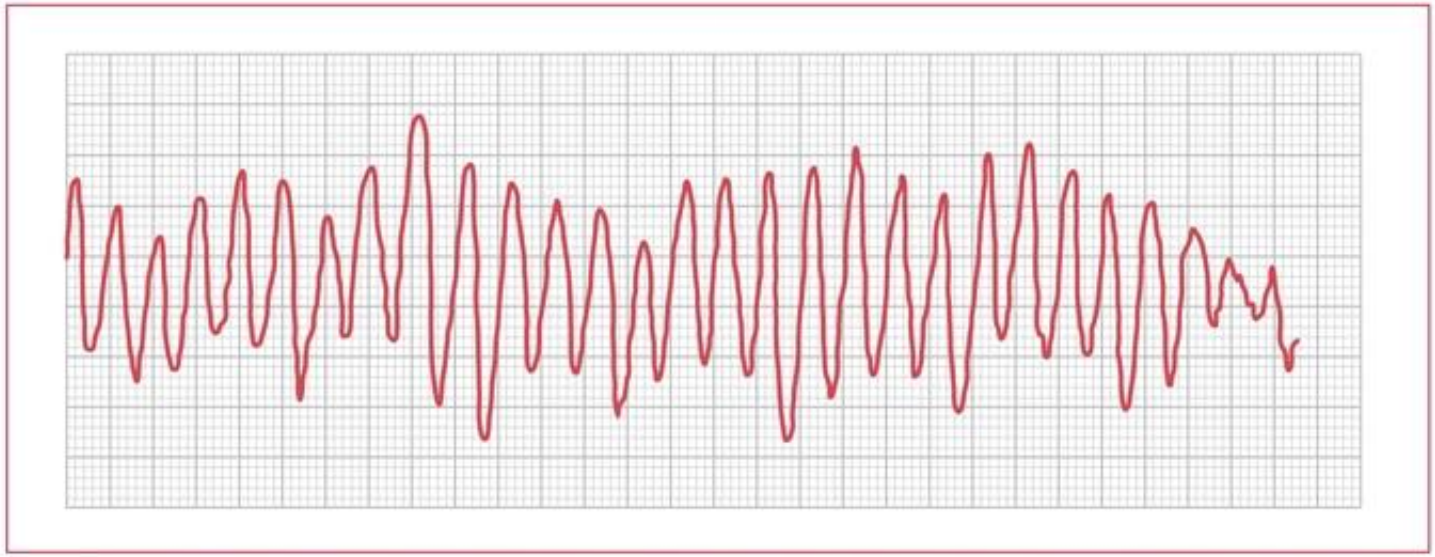
Reversible Causes
- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

SPECIAL CASES

- *Torsade de Pointes*
- *VT with pulse*

Polymorphic (Irregular) VT
torsades de pointes

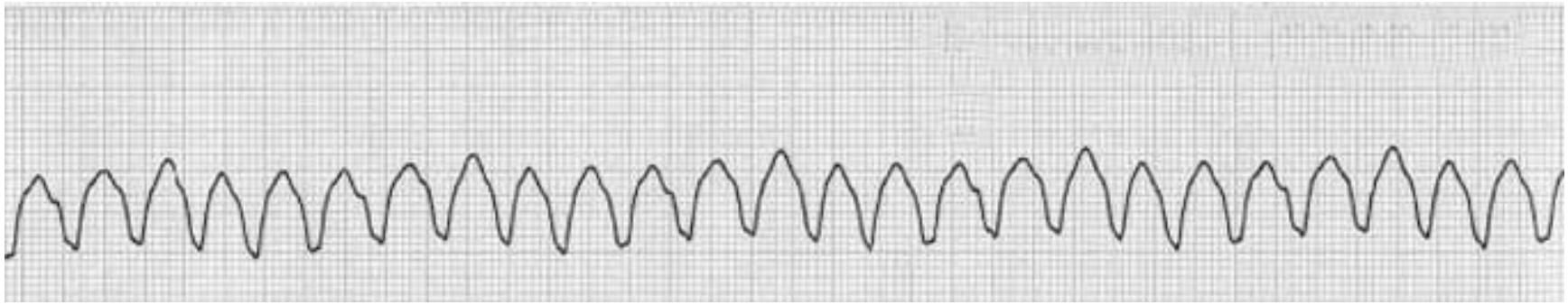
- *Torsade de Pointes*
  - Polymorphic VT.
  - Caused by the use of certain antidysarhythmic drugs.
  - Exacerbated by coadministration of antihistamines, azole antifungal agents and macrolide antibiotics, erythromycin, azithromycin, and clarithramycin.



Polymorphic (Irregular) VT
torsades de pointes

- requires immediate defibrillation with the same strategy used for VF
  - stop medications known to prolong the QT interval
  - Correct electrolyte imbalance
- magnesium is commonly used
  - Polymorphic VT associated with familial long QT syndrome
- isoproterenol or ventricular pacing
  - Polymorphic VT with bradycardia and drug-induced QT prolongation
- IV amiodarone and β–blockers
  - myocardial ischemia induced Polymorphic VT

VT with pulse



- **CARDIOVERSION**
- **MONITOR MORE CLOSELY**

- **PREPARE FOR CARDIOVERSION:**
  - **O_2,**
  - **LIDOCAINE,**
  - **TREAT CAUSE**

Synchronized vs. Unsynchronized Cardioversion

- What is the difference between synchronized and unsynchronized cardioversion?

Synchronized cardioversion

- is a LOW ENERGY SHOCK that uses a sensor to deliver electricity that is synchronized with the peak of the QRS complex (the highest point of the R-wave).
- When the “sync” option is engaged on a defibrillator and the shock button pushed, there will be a delay in the shock. During this delay, the machine reads and synchronizes with the patients ECG rhythm.

Synchronized cardioversion

- The most common indications for synchronized cardioversion are unstable atrial fibrillation, atrial flutter, atrial tachycardia, and supraventricular tachycardias.
- If medications fail in the stable patient with the before mentioned arrhythmias, synchronized cardioversion will most likely be indicated.

Unsynchronized cardioversion (defibrillation)

- is a HIGH ENERGY shock which is delivered as soon as the shock button is pushed on a defibrillator. The shock may fall randomly anywhere within the cardiac cycle (QRS complex).
- Unsynchronized cardioversion (defibrillation) is used when there is no coordinated electrical activity in the heart (pulseless VT/VF) or the defibrillator fails to synchronize in an unstable patient.

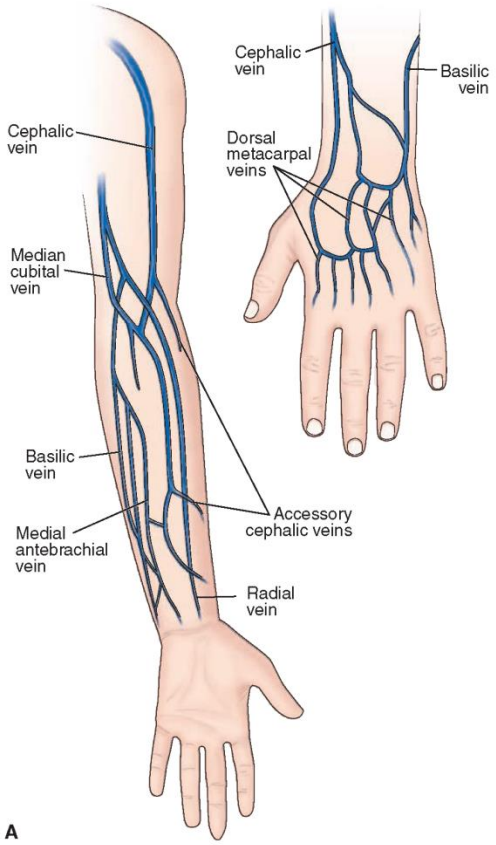
Access for Parenteral Medications During Cardiac Arrest

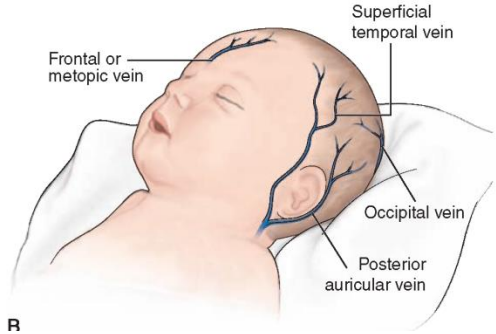
- worsened survival for every minute that antiarrhythmic drug delivery was delayed
- Time to drug administration was also a predictor of ROSC
- insufficient evidence to specify exact time parameters or the precise sequence with which drugs

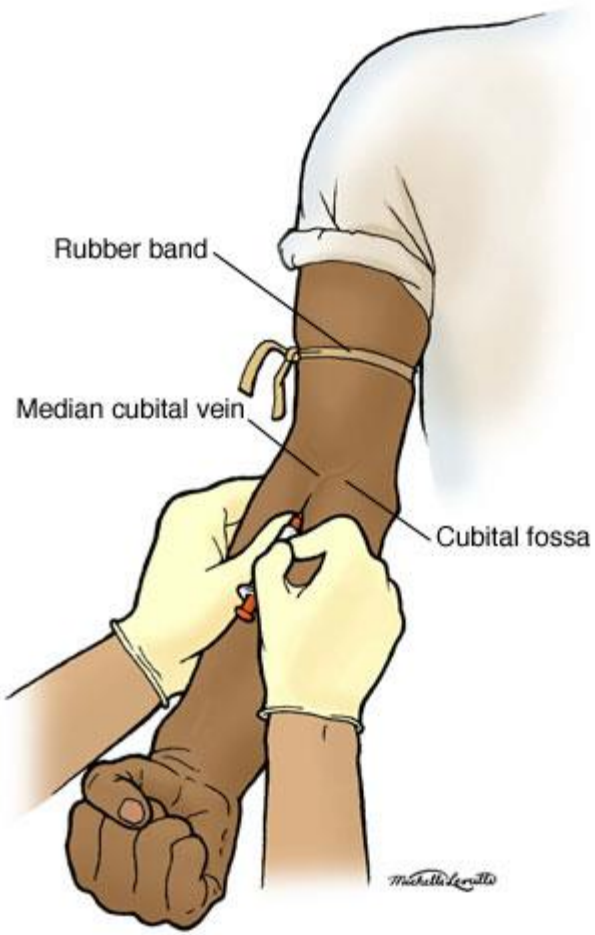
Routes of drugs access

- 1. Peripheral intravenous access
- 2. Central intravenous access
- 3. Intraosseous route (humeral, tibiae way)
- 4. Endotracheal route (unreliable one)

Peripheral IV Drug Delivery





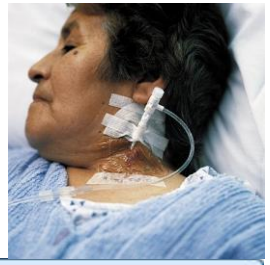


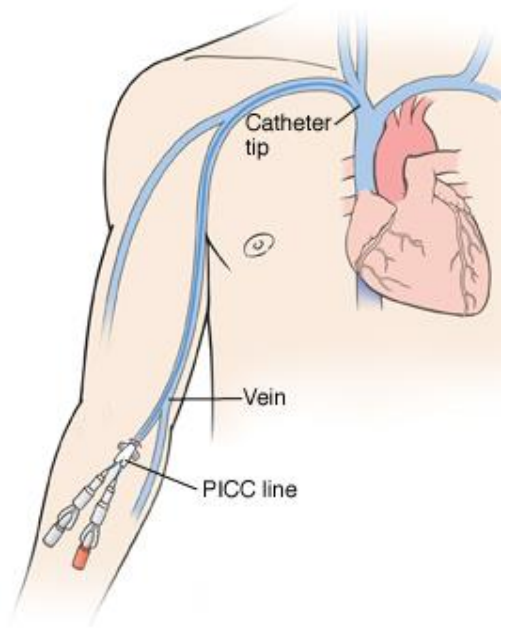
- bolus injection and followed with a 20-mL bolus of IV fluid

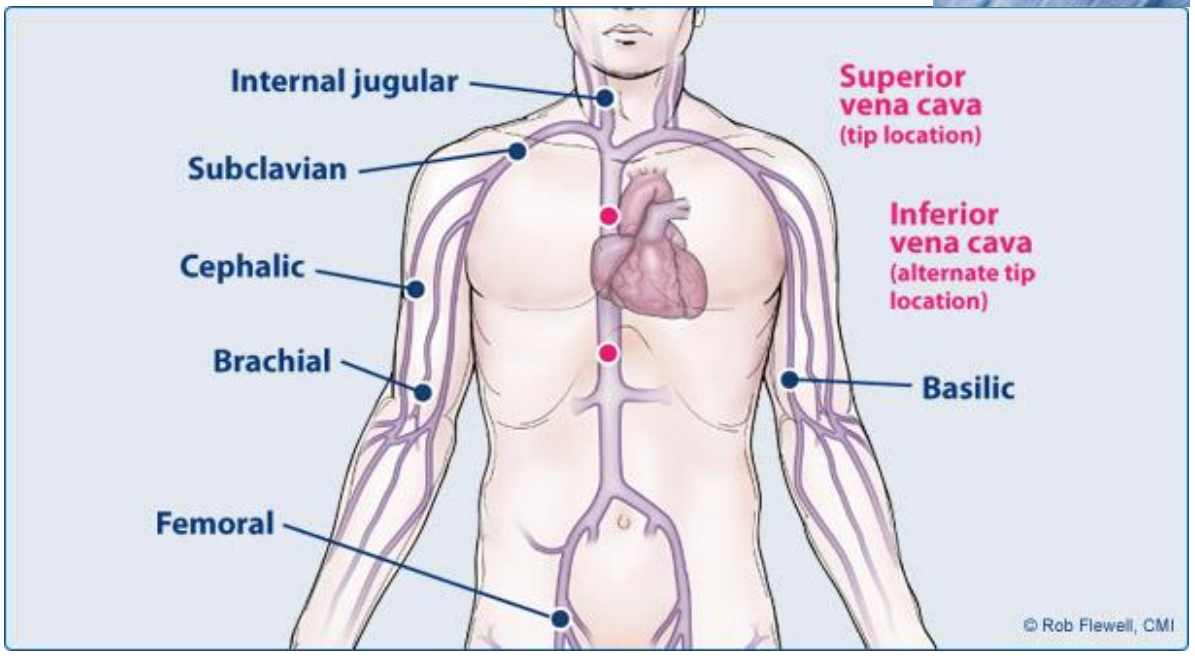
Peripheral IV Drug Delivery

Size(G)	Color Code	Catheter(mm)		Catheter Effective Length (mm)	Flow Rate (ml/min)
		Nominal O.D	O.D (mm)		
14	Orange	1.9 2.0 2.1 2.2	1.85---2.249	45(51)±2	300
16	Grey	1.6 1.7 1.8	1.55---1.849	45(51)±2	195
17	White	1.4 1.5	1.35---1.549	45(51)±2	133
18	Dark Green	1.2 1.3	1.15---1.349	45(32)±2	90
20	Pink	1.0 1.1	0.95---1.149	32(25)±2	50
22	Blue	0.8 0.9	0.75---0.949	25(19)±2	25
24	Yellow	0.7	0.65---0.749		
26	Purple	0.6	0.55---0.649		

Central IV Drug Delivery



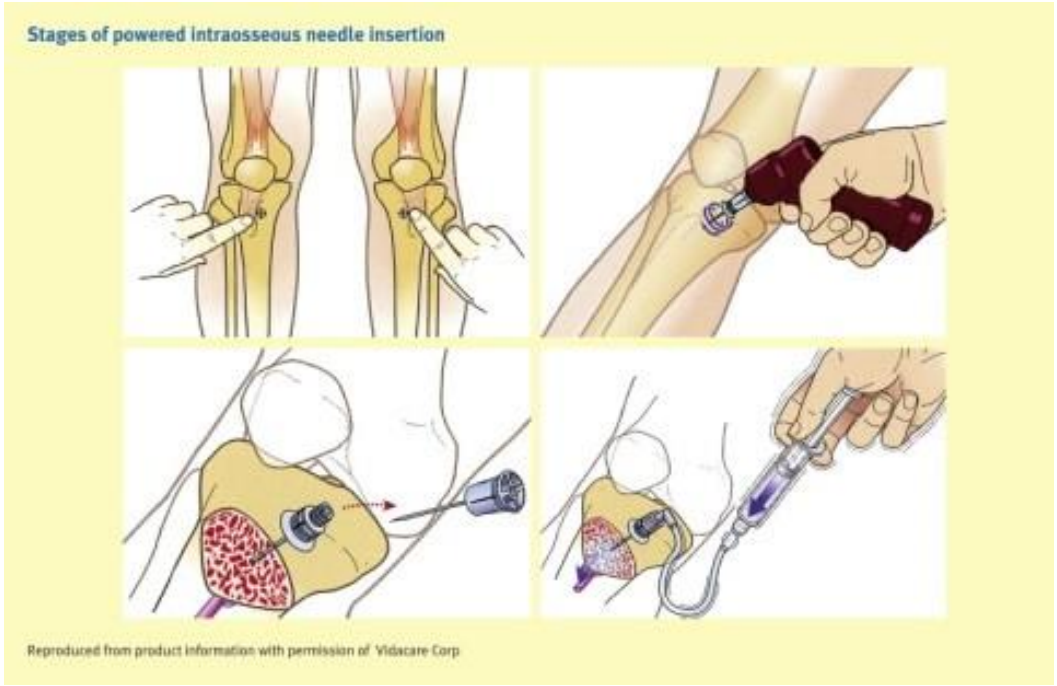




- circulation times shorter
- interrupt CPR
- Central venous catheterization is a relative (but not absolute) contraindication for fibrinolytic therapy in patients with acute coronary syndromes.

IO Drug Delivery

- all age groups



IO Drug Delivery

Pediatric Bone Injection Gun

Adult Bone Injection Gun

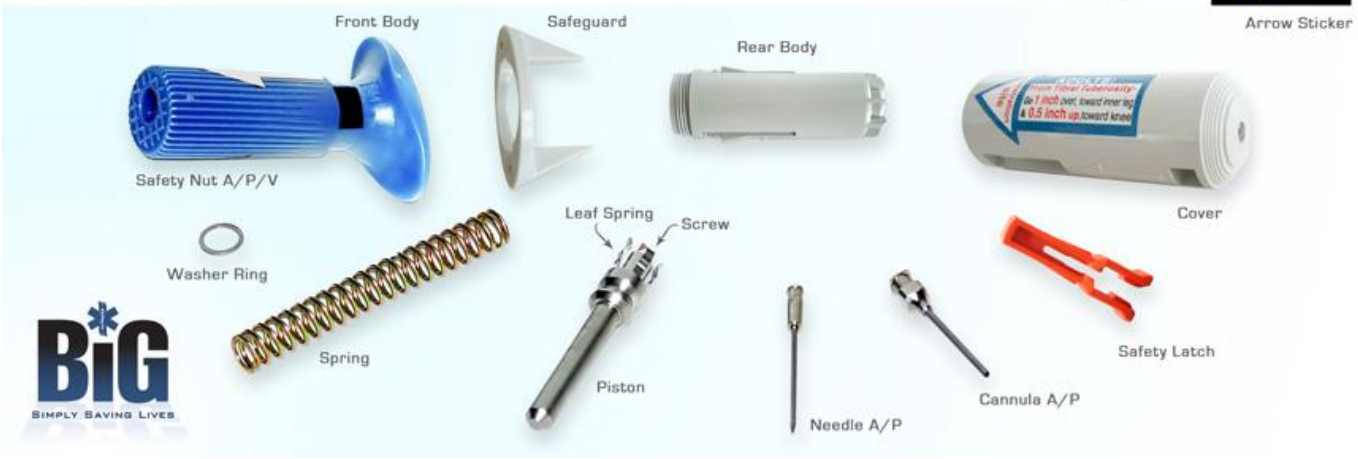
Scale Sticker

Location Sticker

Tayvack

Location Sticker

Arrow Sticker



VIDEO

Endotracheal Drug Delivery

- lidocaine, epinephrine, atropine, naloxone, and vasopressin
- 2-21⁄2 times the recommended IV dose
- dilute the recommended dose in 5-10 mL of sterile water or normal saline and inject the drug directly into the endotracheal tube

Endotracheal Drug Delivery

- Studies with epinephrine and lidocaine showed that dilution with sterile water instead of 0.9% saline may achieve better drug absorption.

Drugs, used in ACLS

- 1. Adrenaline = epinephrine
- 2. Vasopressin
- 3. Amiodarone (resistant VF/VT, other resistant tachyarhythmias)
- 4. Lidocaine (VF/VT)
- 5. Magnesium sulphate (VT, ST, Digoxine toxity)
- 6. Other drugs

Epinephrine

- 1 mg dose of IV/IO epinephrine every 3-5 minutes during adult cardiac arrest .
- If IV/IO access is delayed or cannot be established, epinephrine may be given endotracheally at a dose of 2-2.5 mg.

Vasopressin

- nonadrenergic peripheral vasoconstrictor
- 1 dose of vasopressin 40 units IV/IO may replace either the first or second dose of epinephrine in the treatment of cardiac arrest

Amiodarone

- An initial dose of 300 mg IV/IO can be followed by 1 dose of 150 mg IV/IO.

Lidocaine

- considered if amiodarone is not available
- The initial dose is 1-1.5 mg/kg IV.
- If VF/pulseless VT persists, additional doses of 0.5-0.75 mg/kg IV push may be administered at 5- to 10-minute intervals to a maximum dose of 3 mg/kg.

Magnesium Sulfate

- torsades de pointes
- IV/IO bolus of magnesium sulfate at a dose of 1-2 g diluted in 10 mL D5W

Other drugs

- Atropine
- Sodium Bicarbonate
- Calcium
- Fibrinolysis
- IV Fluids

Summary

- No matter what kind of doctor You are going to be, sooner or later You will have to deal with an emergency situation.
- It is always good to know that You are able to save someone’s life. At least You will know that You did your best.

THANK YOU FOR YOUR ATTENTION!