
بسم الله الرحمن الرحيم

Adult Resuscitation

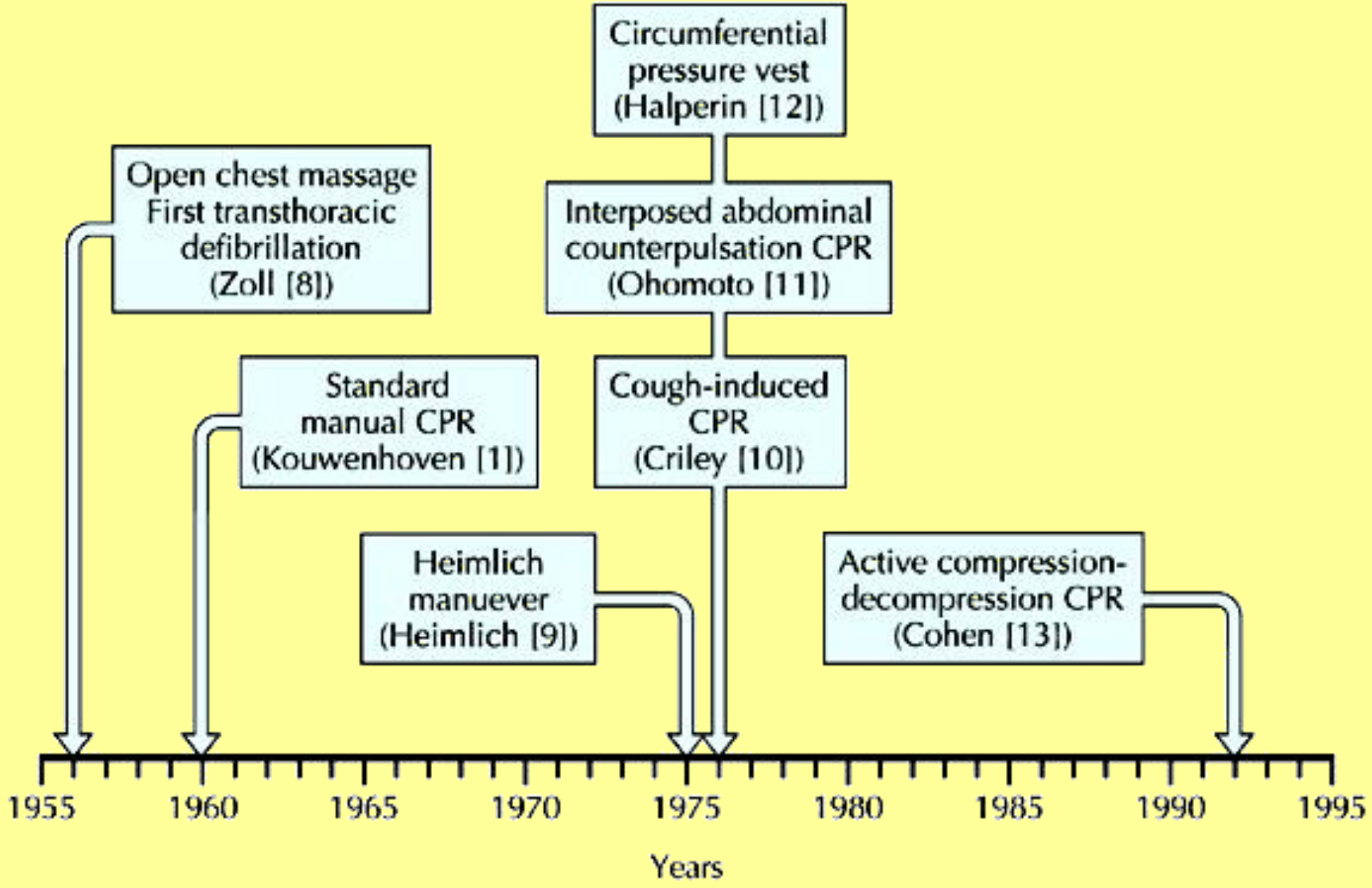
- *Cardiopulmonary arrest* is defined by the triad of ***unconsciousness***, ***apnea***, and ***pulselessness***

Important concepts and practices for the AHA Guidelines for BLS include:

- **Immediate recognition of sudden cardiac arrest (SCA) by noting *unresponsiveness* or absent/gasping breathing**

PERSPECTIVE

- The majority of effective techniques used in resuscitation today were described more than 100 years ago
- . The modern era of cardiopulmonary resuscitation (CPR), however, began in the late 1950s with the rediscovery of closed-chest cardiac massage and mouth-to-mouth ventilation coupled with technical advances in external defibrillation.
- The first successful ***electrical reversal of ventricular fibrillation*** by externally applied paddles was reported by Zoll in 1956
- Safar and Elam described effective techniques of airway management and ***mouth-to-mouth ventilation*** in 1958.
- ***Closed-chest cardiac massage*** was rediscovered by Kowenhoven, Jude, and Knickerbocker in 1960.
- The synthesis of these three noninvasive techniques greatly increased the number of people who could be trained to administer CPR and the locations where it could be performed.
- The realization that most sudden deaths occur outside the hospital led to the extension of CPR and emergency cardiac care to the out-of-hospital setting. Despite intensive research efforts over the past 30 years, **no new clinically feasible intervention has been shown to improve survival from cardiac arrest other than early CPR and early defibrillation**


Circumferential pressure vest (Halperin [12])
Open chest massage First transthoracic defibrillation (Zoll [8])
Interposed abdominal counterpulsation CPR (Ohomoto [11])
Standard manual CPR (Kouwenhoven [1])
Cough-induced CPR (Criley [10])
Heimlich manuever (Heimlich [9])
Active compression-decompression CPR (Cohen [13])
1955
1960
1965
1970
1975
1980
1985
1990
1995
Years

Sudden unexpected death

- Defined as death within 24 hours of symptom onset in a previously functional individual, accounts for up to one third of all nontraumatic deaths, with most occurring outside the hospital.

PATHOPHYSIOLOGY

- Cardiac arrest results in global cessation of blood flow
- vulnerability to ischemic injury varies among different organs and by region within the same organ.
- The brain is the most susceptible organ to ischemic injury, with neurons in the **cerebral cortex**, **hippocampus**, and **cerebellum** being selectively vulnerable.
- The heart is the second most susceptible organ to ischemic injury, with the **endocardium** being more sensitive than the epicardium.
- The **renal**, **gastrointestinal**, **musculoskeletal**, and integumentary systems are much more resistant to ischemia

- Prearrest **A period of hypoxia or hypotension immediately preceding cardiac arrest, as occurs in respiratory arrest, depletes cell energy stores and causes tissue acidosis, both of which worsen the insult severity.**
- Arrest **Once energy depletion occurs, cell membranes depolarize and a cascade of metabolic events is initiated, including intracellular Ca++ overload, generation of free radicals, mitochondrial dysfunction, activation of catabolic enzymes (phospholipases, endonucleases, proteases), and inflammation**
- Resuscitation **Cardiac output generated by standard chest compressions is at best less than 30% of baseline .1- release of catecholamines 2- vasoactive peptides 3-and administration of exogenous vasoconstrictors both the cardiac compression and the thoracic pump mechanisms are working during standard chest compressions, but the impact of the thoracic pump is dominan**
- Postresuscitation ***postresuscitation syndrome***

ETIOLOGY

- ***Cardiac*** :*ventricular fibrillation* ***(VF)*** *or less often as pulseless ventricular tachycardia* ***(VT).***

1. **Coronary artery disease**
2. **Cardiomyopathies**
3. **Structural abnormalities**
4. **Valve dysfunction**

ETIOLOGY

- *Respiratory*
  - **Hypoventilation**
    - CNS dysfunction
    - Neuromuscular disease
    - Toxic and metabolic encephalopathies
  - **Upper airway obstruction**
    - CNS dysfunction
    - Foreignbody
    - Infection
    - Trauma
    - Neoplasm
  - **Pulmonary dysfunction**
    - Asthma, COPD
    - Pulmonary edema
    - Pulmonary embolus
    - Pneumonia

ETIOLOGY

- *Circulatory*

1. ***Mechanical obstruction***

Tension pneumothorax
Pericardial tamponade

Pulmonary embolus

2. ***Hypovolemia***

Hemorrhage

3. ***Vascular tone***

Sepsis

Neurogenic

ETIOLOGY

- *Metabolic*

Electrolyte abnormalities

Hypokalemia
Hyperkalemia
Hypermagnesemia
Hypomagnesemia
Hypocalcemia

ETIOLOGY

- *Toxic*
- **Prescription medications**

  Antidysrhythmics
  Digitalis
  β-blockers
  Calcium channel blockers
  Tricyclic antidepressants

- **Drugs of abuse**

  Cocaine
  Heroin

- **Toxins**

  Carbon monoxide
  Cyanide

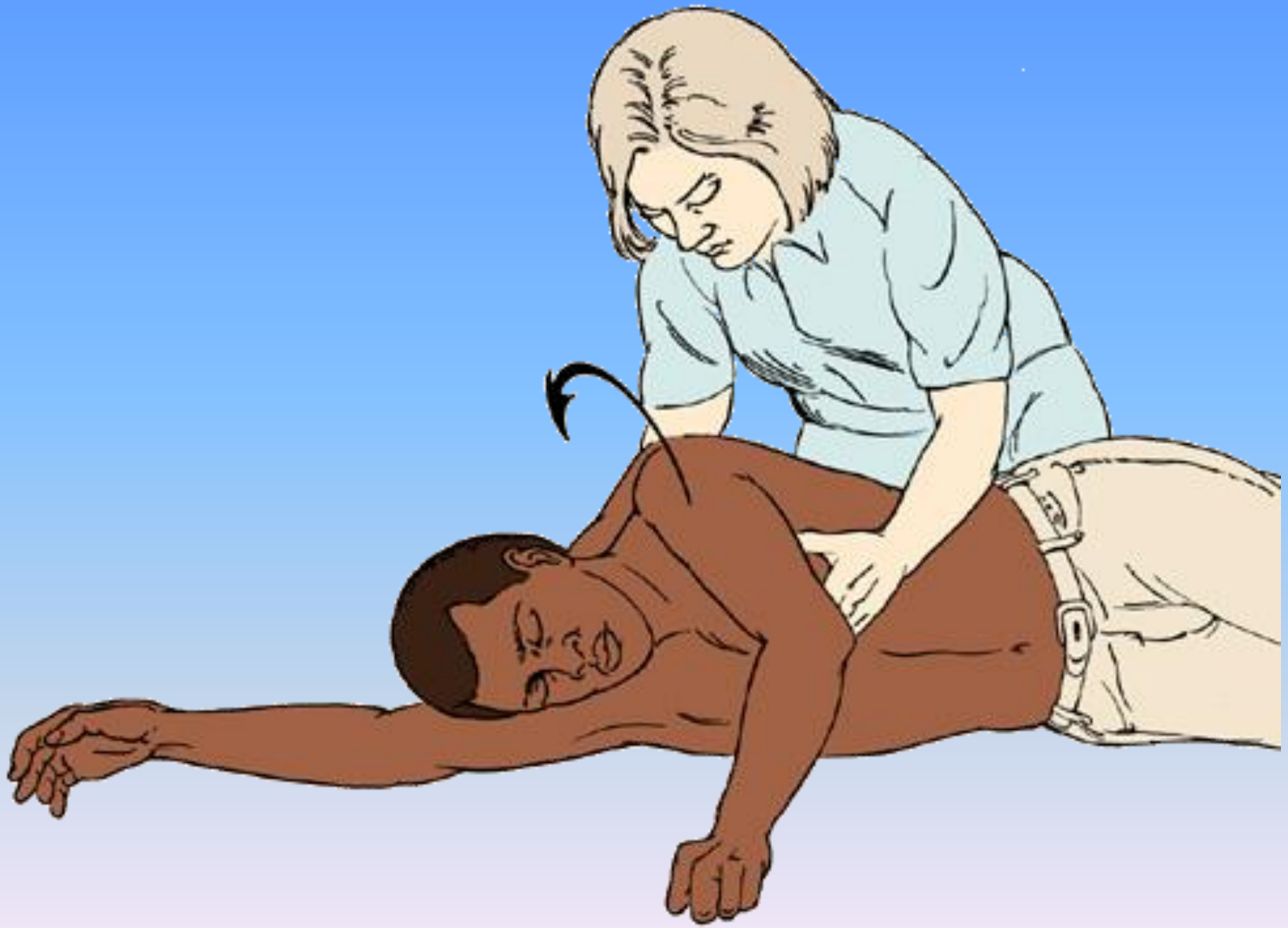
ETIOLOGY

- *Environmental*

Lightning
Electrocution
Hypothermia
Hyperthermia
Drowning/near-drowning


MANAGEMENT


Are you okay?



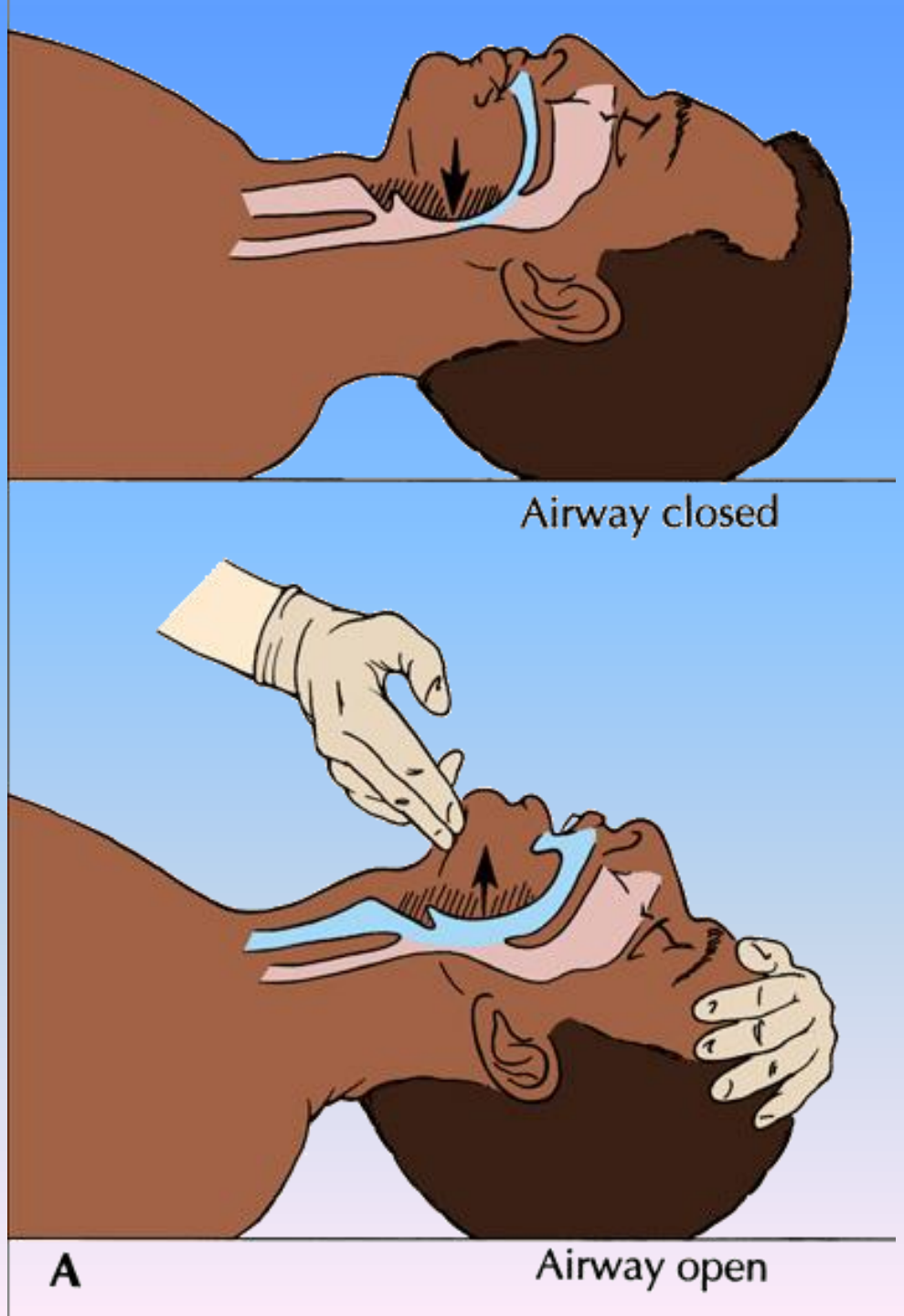
History and Physical Examination

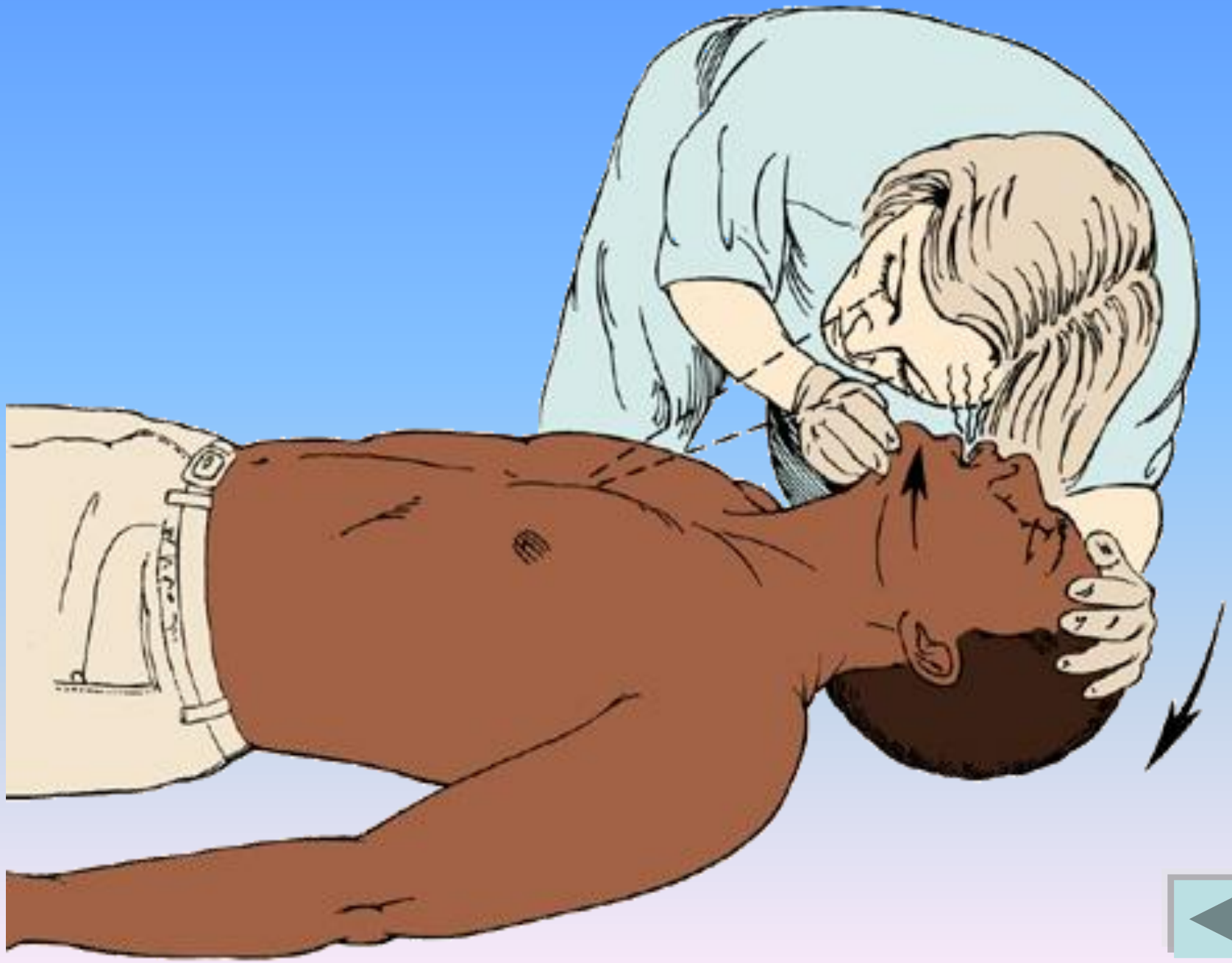
(1) ensure adequacy of airway maintenance and ventilation

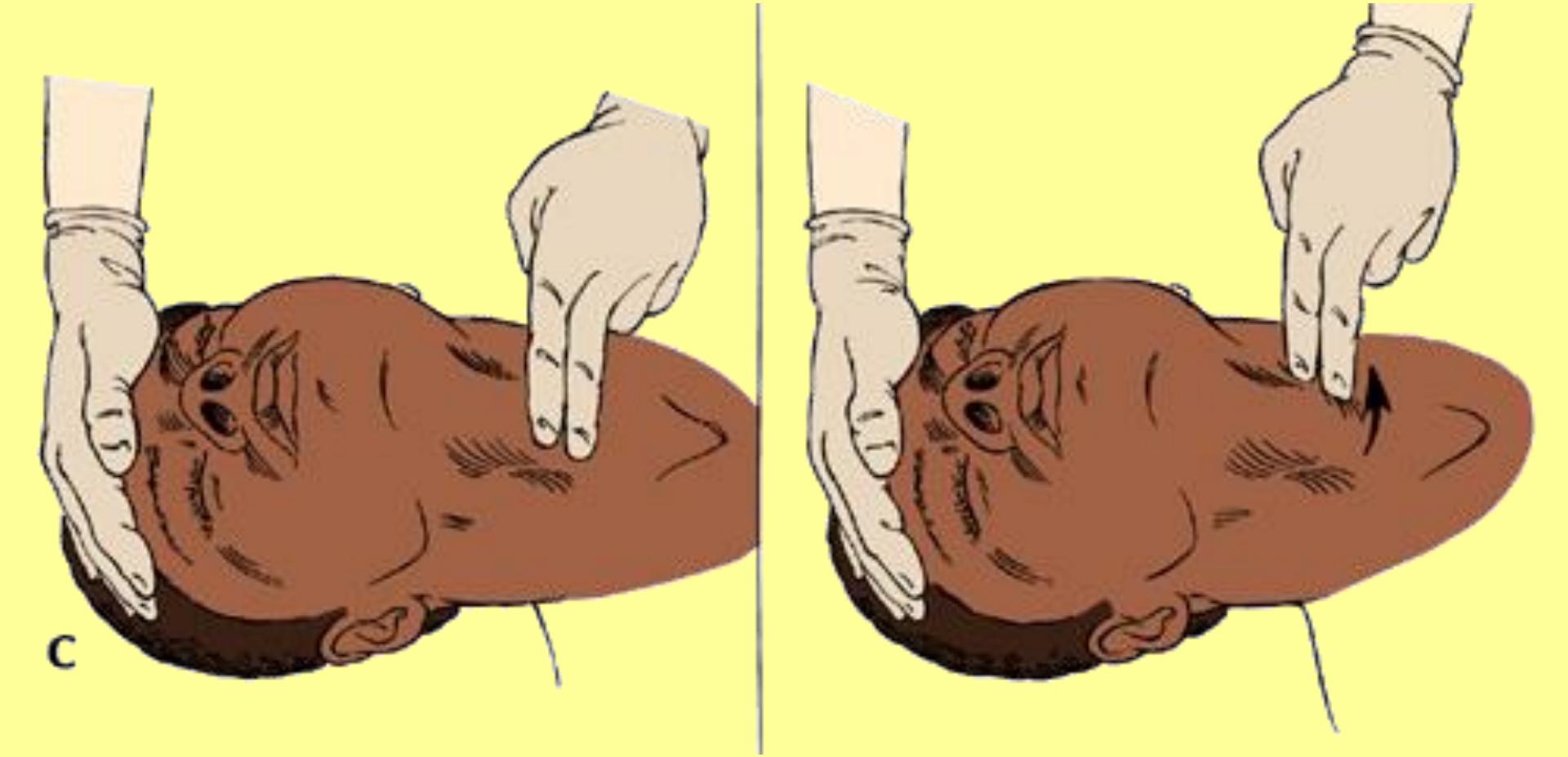
(2) confirm the diagnosis of cardiac arrest

(3) find evidence of etiology

(4) monitor for complications of therapeutic interventions


Airway closed
A
Airway open

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- **With sudden onset of circulatory arrest, as in VF,**

1. *loss of consciousness occurs within 15 seconds*
2. *gasping respirations may persist for up to 60 seconds*
3. *A brief seizure may result from cessation of cerebral blood flow*

- **Primary respiratory arrest**

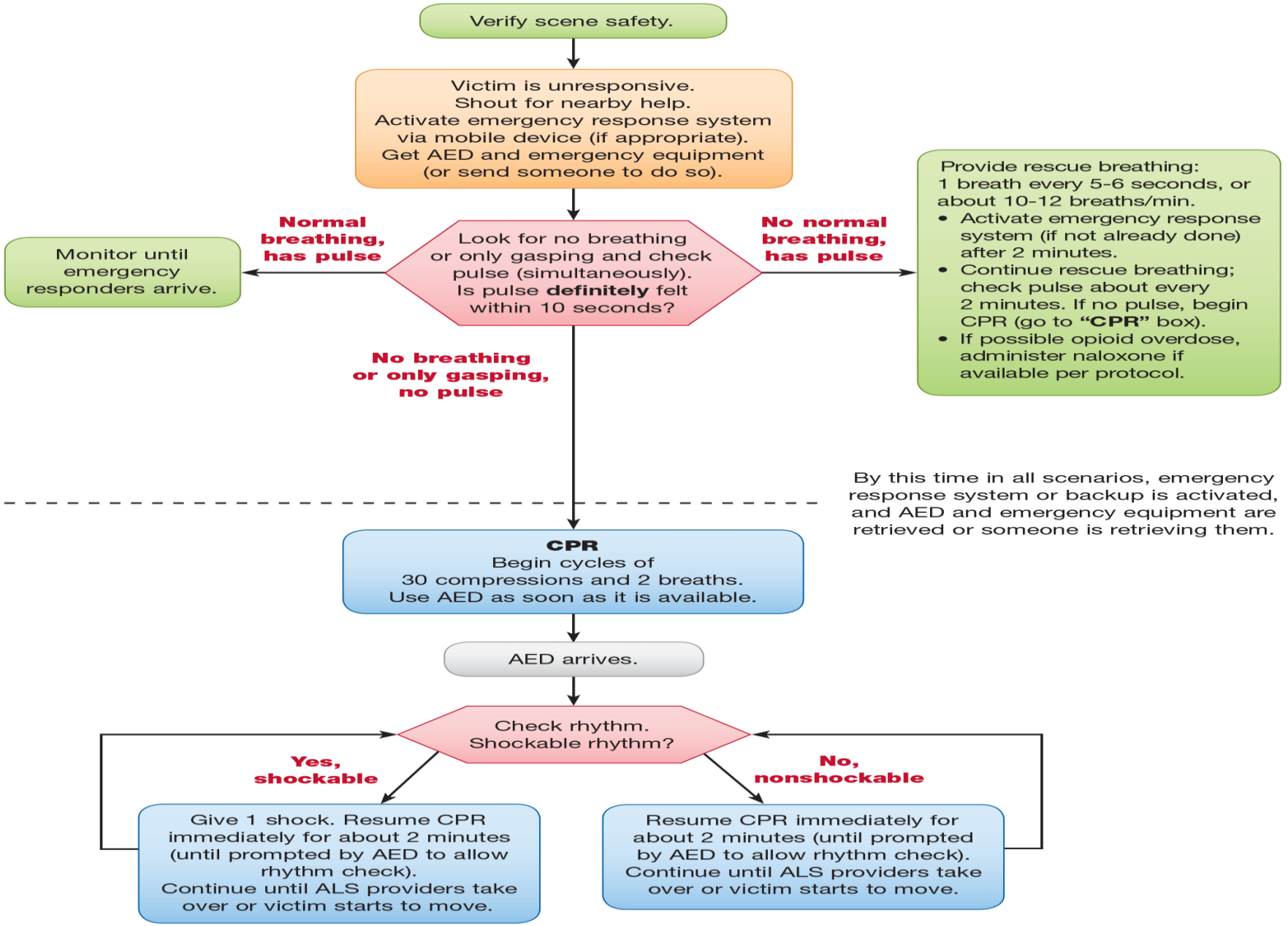
1. *Transient tachycardia and hypertension*
2. *Loss of consciousness*
3. *Bradycardia*
4. *Pulselessness, usually within 5 minutes.*

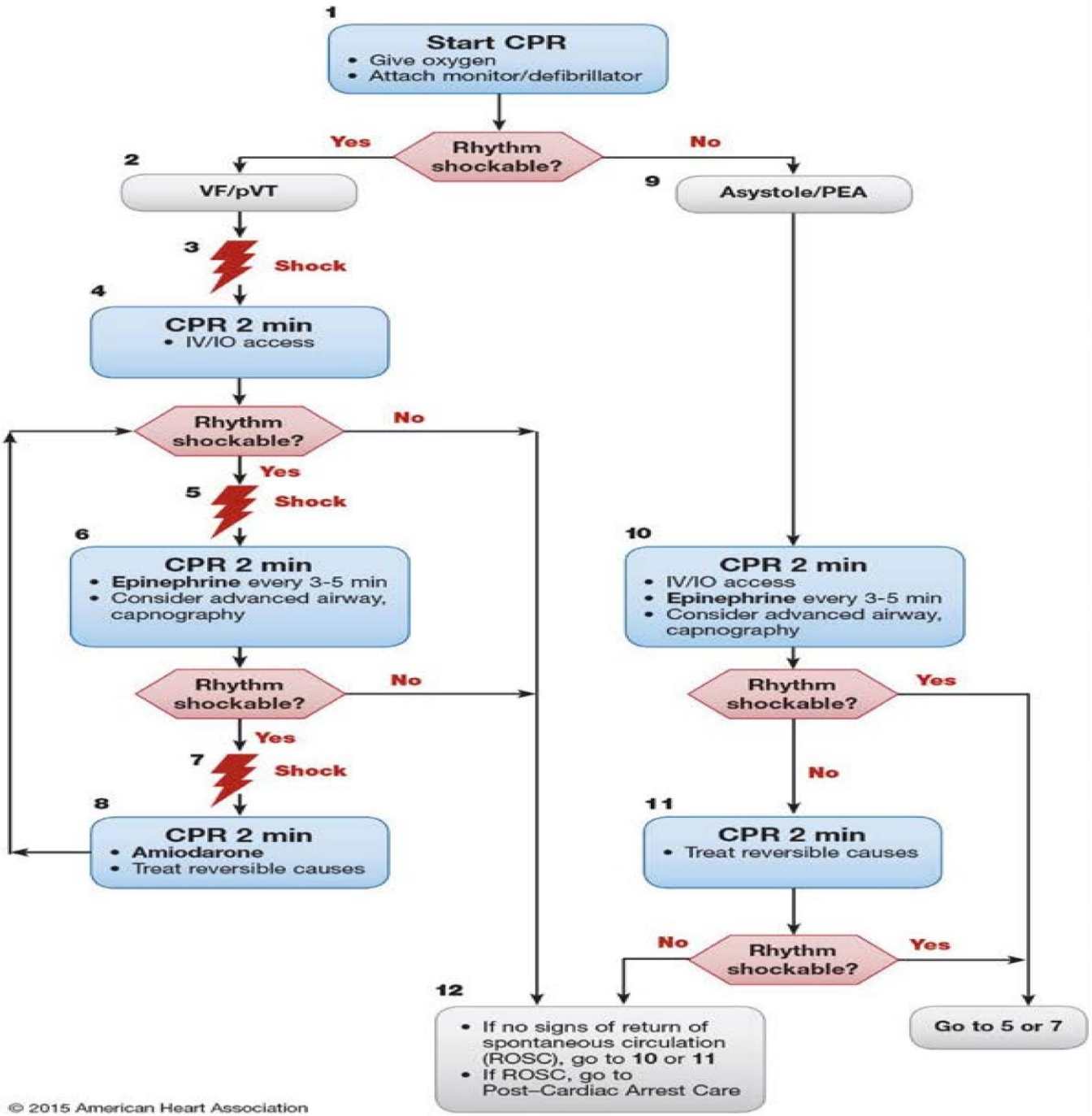
Physical examination	Abnormalities	Potential causes
• **General**	*Pallor*	Hemorrhage
	Cold	Hypothermia
• **Airway**	*Secretions, vomitus, or blood*	Aspiration Airway obstruction
	Resistance to positive-pressure ventilation	Tension pneumothorax Airway obstruction Bronchospasm
• **Neck**	*Jugular venous distention*	Tension pneumothorax Cardiac tamponade Pulmonary embolus
	Tracheal deviation	Tension pneumothorax

Physical examination	Abnormalities	Potential causes
• **Chest**	*Median sternotomy scar*	Underlying cardiac
• **Lungs**	*Unilateral breath sounds*	Tension pneumothorax Right mainstem intubation Aspiration
	Distant or no breath sounds no chest expansion	Esophageal intubation Airway obstruction Severe bronchospasm
	Wheezing	Aspiration Bronchospasm Pulmonary edema
	Rales	Aspiration Pulmonary edema Pneumonia

PHYSICAL EXAMINATION	ABNORMALITIES	POTENTIAL CAUSES
• **Heart**	***NO* Audible heart tones**	Hypovolemia Cardiac tamponade Tension pneumothorax Pulmonary embolus
• **Abdomen**	***Distended and dull***	Ruptured abdominal Aortic aneurysm Ruptured ectopic pregnancy
	Distended, tympanitic	Esophageal intubation Gastric insufflation
• **Extremities**	***Asymmetric pulses***	Aortic dissection
	Arteriovenous shunt fistula	Hyperkalemia
• **Skin**	***Needle tracts or abscess***	Intravenous drug abuse
	Burns	Smoke inhalation Electrocution

BLS Healthcare Provider
Adult Cardiac Arrest Algorithm—2015 Update





CPR Quality

- Push hard (at least 2 inches [5 cm]) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Rotate compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- Quantitative waveform capnography
  - If P_{ETCO_2} <10 mm Hg, attempt to improve CPR quality.
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality.

Shock Energy for Defibrillation

- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy

- **Epinephrine IV/IO dose:** 1 mg every 3-5 minutes
- **Amiodarone IV/IO dose:** First dose: 300 mg bolus. Second dose: 150 mg.

Advanced Airway

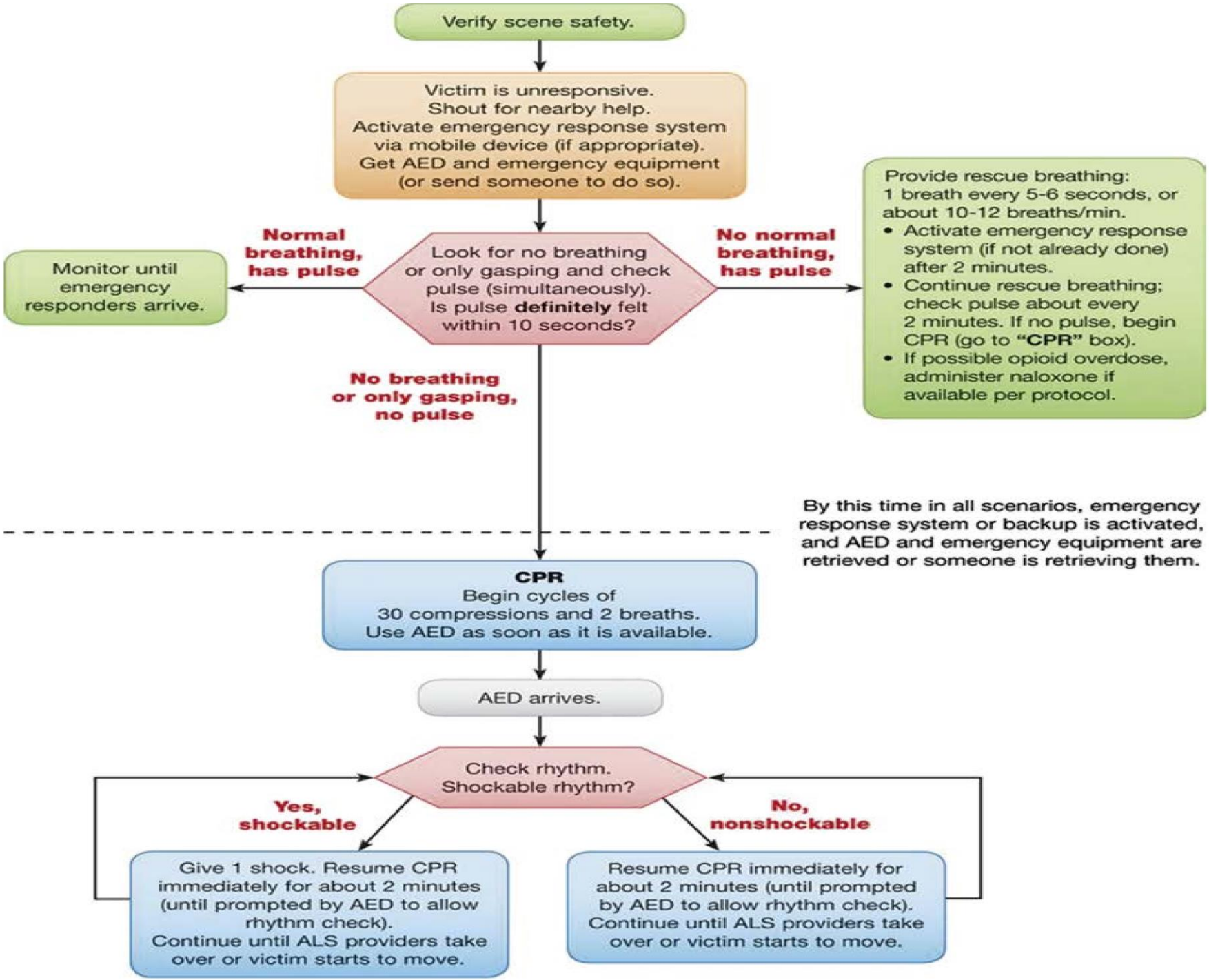
- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

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

Reversible Causes

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


Verify scene safety.
Victim is unresponsive.
Shout for nearby help.
Activate emergency response system via mobile device (if appropriate).
Get AED and emergency equipment (or send someone to do so).
Look for no breathing or only gasping and check pulse (simultaneously). Is pulse definitely felt within 10 seconds?
Normal breathing, has pulse
Monitor until emergency responders arrive.
No normal breathing, has pulse
Provide rescue breathing:
1 breath every 5-6 seconds, or about 10-12 breaths/min.
• Activate emergency response system (if not already done) after 2 minutes.
• Continue rescue breathing; check pulse about every 2 minutes. If no pulse, begin CPR (go to "CPR" box).
• If possible opioid overdose, administer naloxone if available per protocol.
No breathing or only gasping, no pulse
By this time in all scenarios, emergency response system or backup is activated, and AED and emergency equipment are retrieved or someone is retrieving them.
CPR
Begin cycles of 30 compressions and 2 breaths.
Use AED as soon as it is available.
AED arrives.
Check rhythm. Shockable rhythm?
Yes, shockable
Give 1 shock. Resume CPR immediately for about 2 minutes (until prompted by AED to allow rhythm check).
Continue until ALS providers take over or victim starts to move.
No, nonshockable
Resume CPR immediately for about 2 minutes (until prompted by AED to allow rhythm check).
Continue until ALS providers take over or victim starts to move.

Therapeutic Modalities

Circulation

Defibrillation

Airway

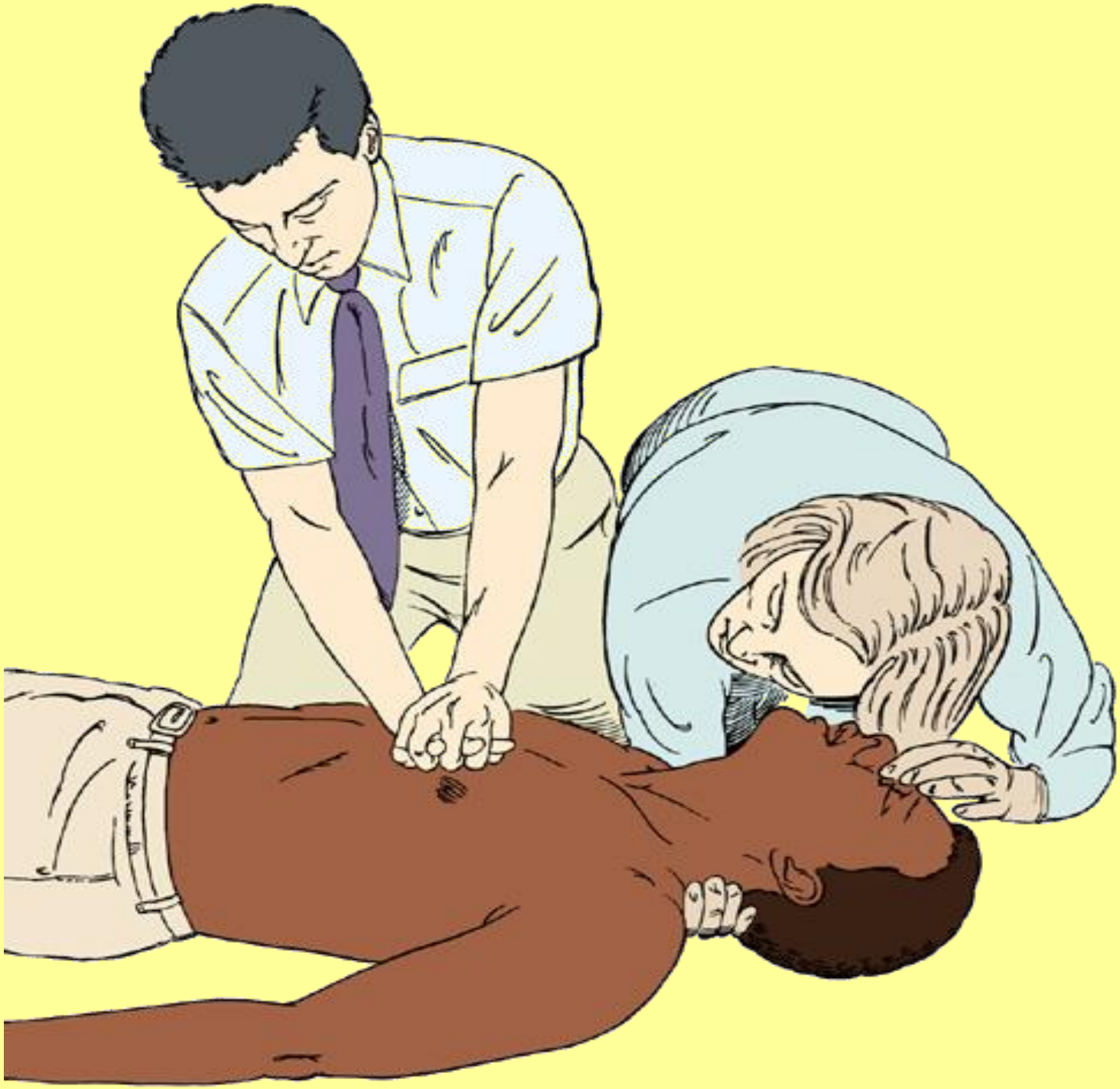
Breathing

Pharmacologic Therapy

Airway and Breathing

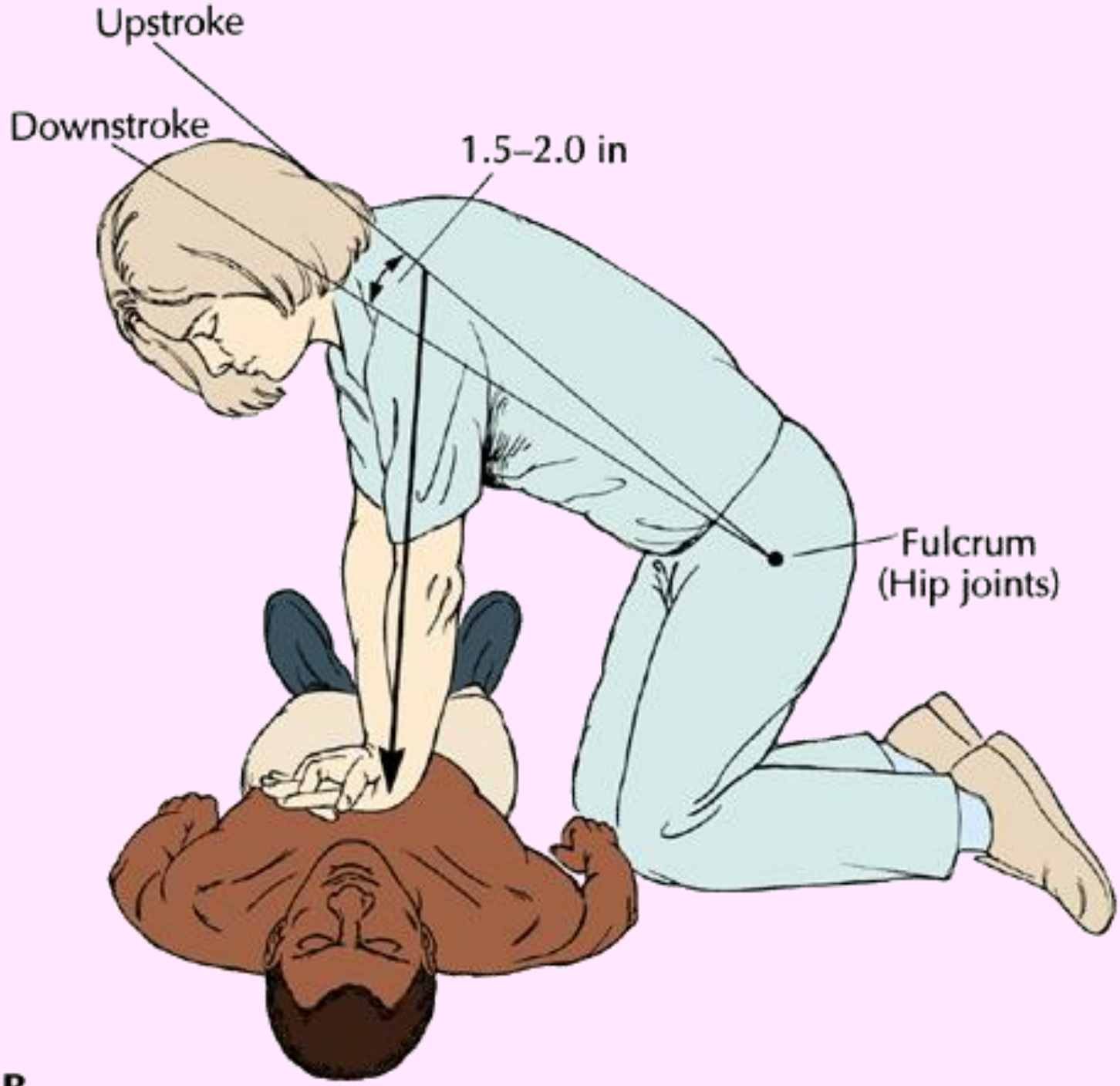
- **Bag/valve/mask ventilation with 100% O2**
- **Endotracheal (ET) intubation**
  adequate ventilation
  oxygenation
  airway protection
  route of drug administration
- ***The recommended rate of ventilation is***
  two breaths for every 15 compressions in one-rescuer CPR
  one breath for each five compressions in two-rescuer CPR


1, and 2,
and 3, and 4, ...



Circulation

- ***Standard chest compression***
  simplicity
  noninvasiveness
  lack of sufficient evidence for improved outcome with other methods (*interposed abdominal compressions, active compression-decompression, and vest CPR)*
- ***Invasive methods :***
  Open-chest cardiac massage (OCCM)
  Cardiopulmonary bypass(CPB)
  logistic and technical limitations
  no clinical trials have demonstrated improved survival or neurologic outcome



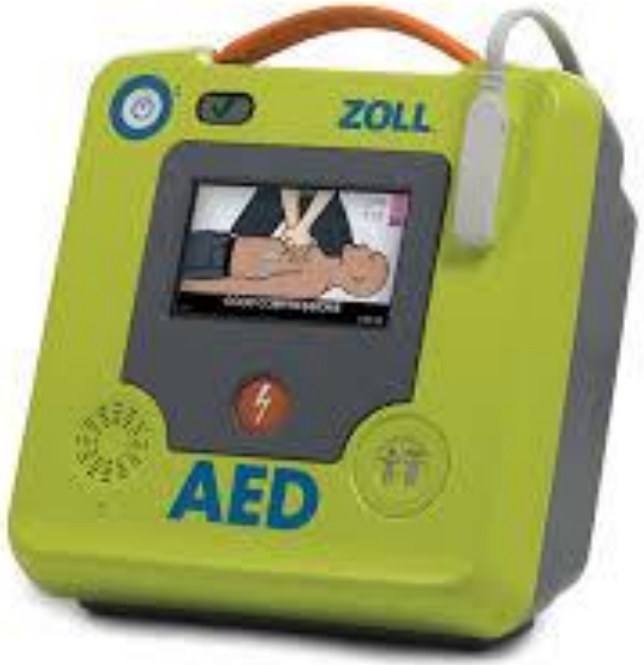
B

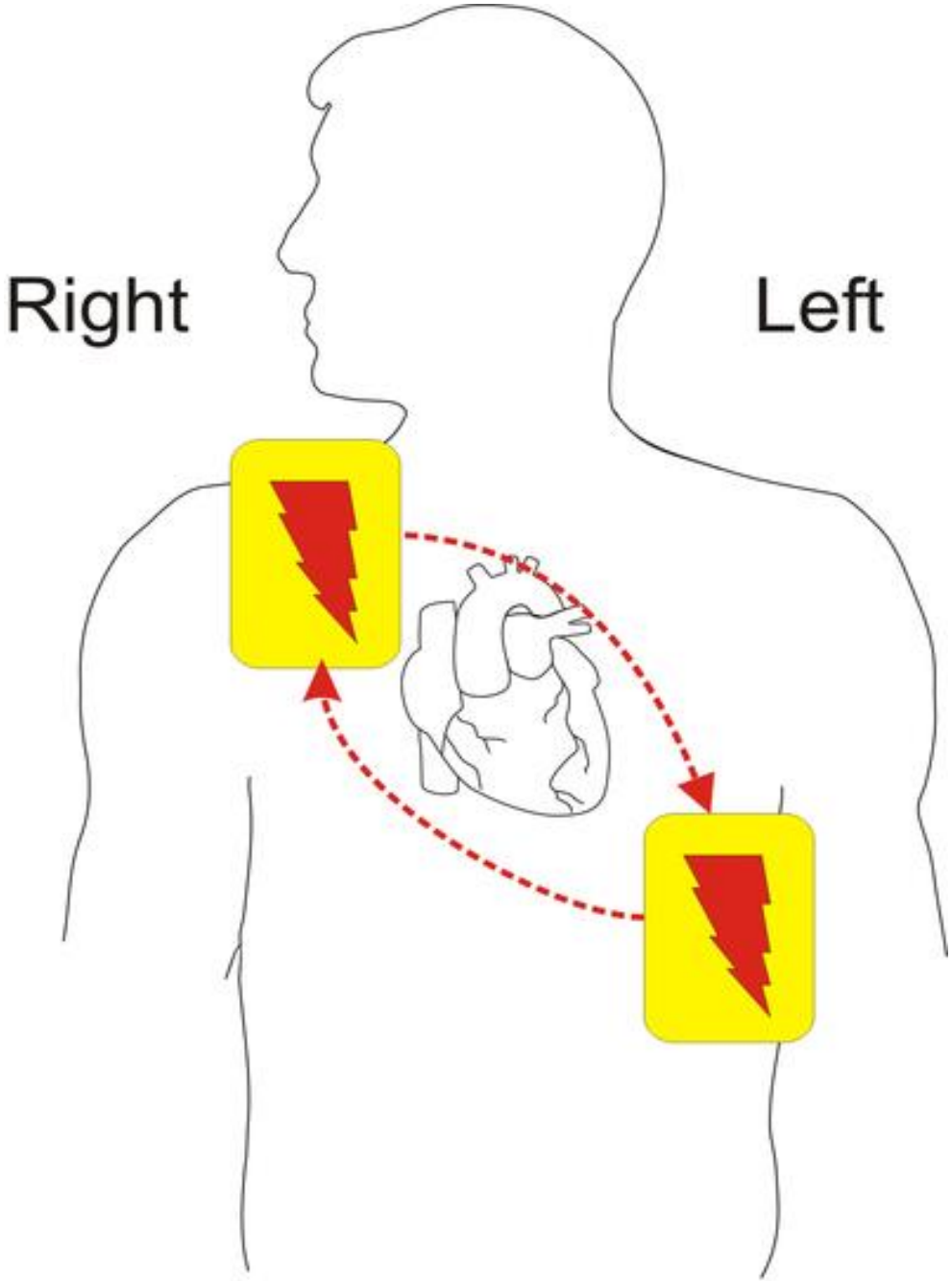
Important concepts and practices for the AHA Guidelines for BLS include:

- ●Minimizing interruptions in CPR
- ●For health care professional rescuers, taking no more than 10 seconds to check for a pulse
- ●For single untrained rescuers, encouraging performance of excellent chest compression-only CPR
- ●Using automated external defibrillators as soon as available
- ●Activating emergency medical services as soon as possible

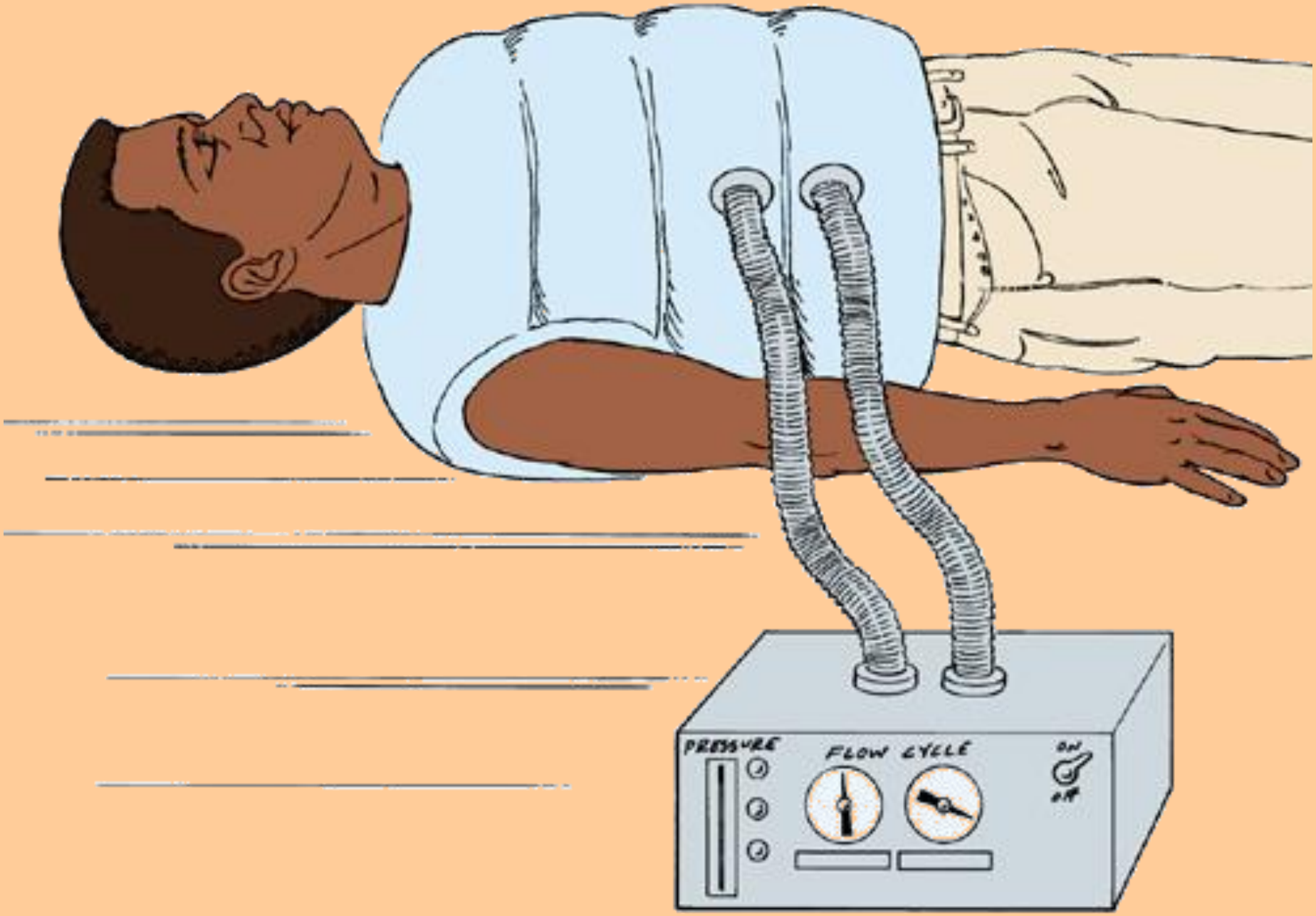

Lifeline VIEW
Give 30 Compressions
SHOCK

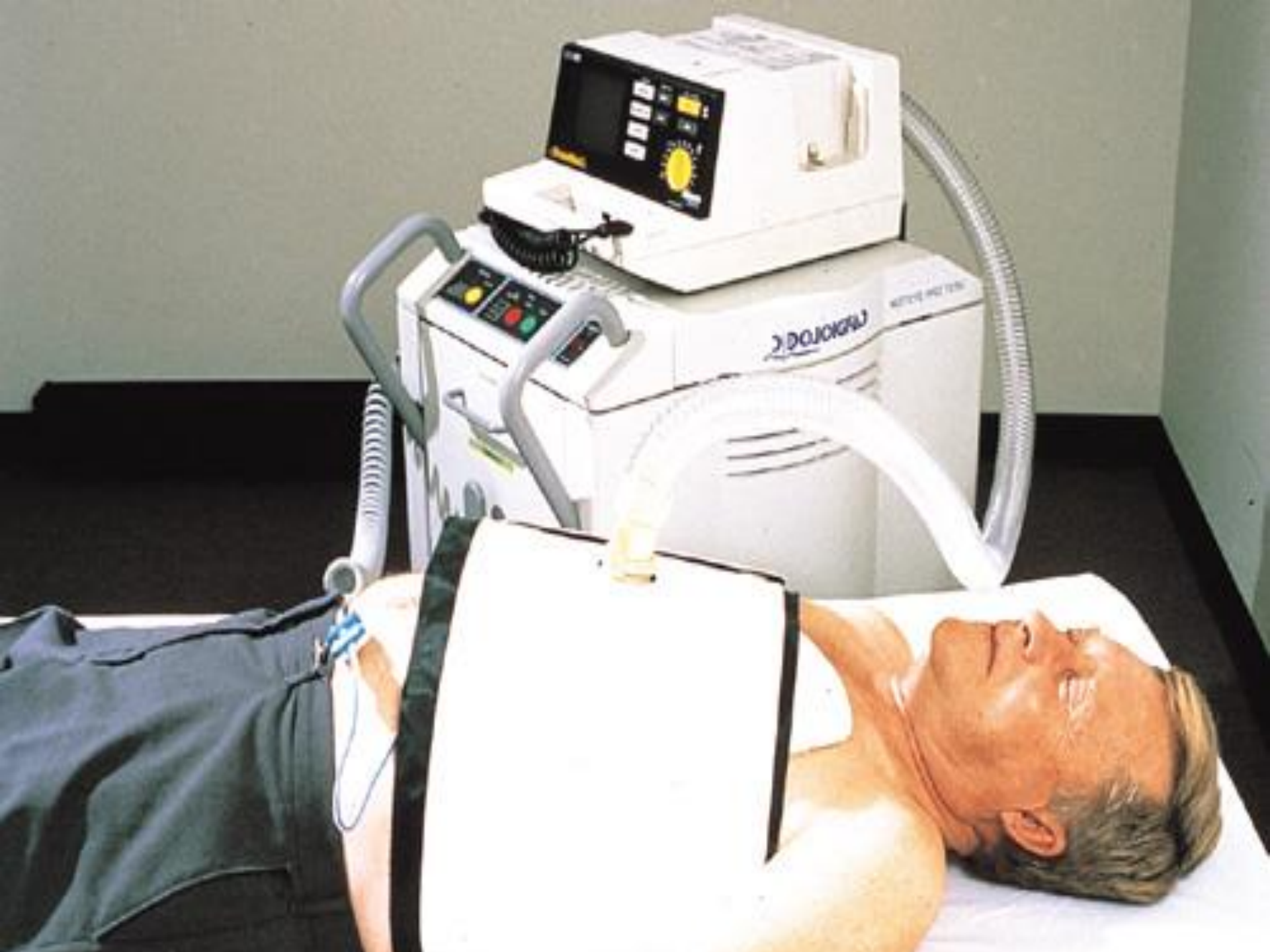

ZOLL
AED

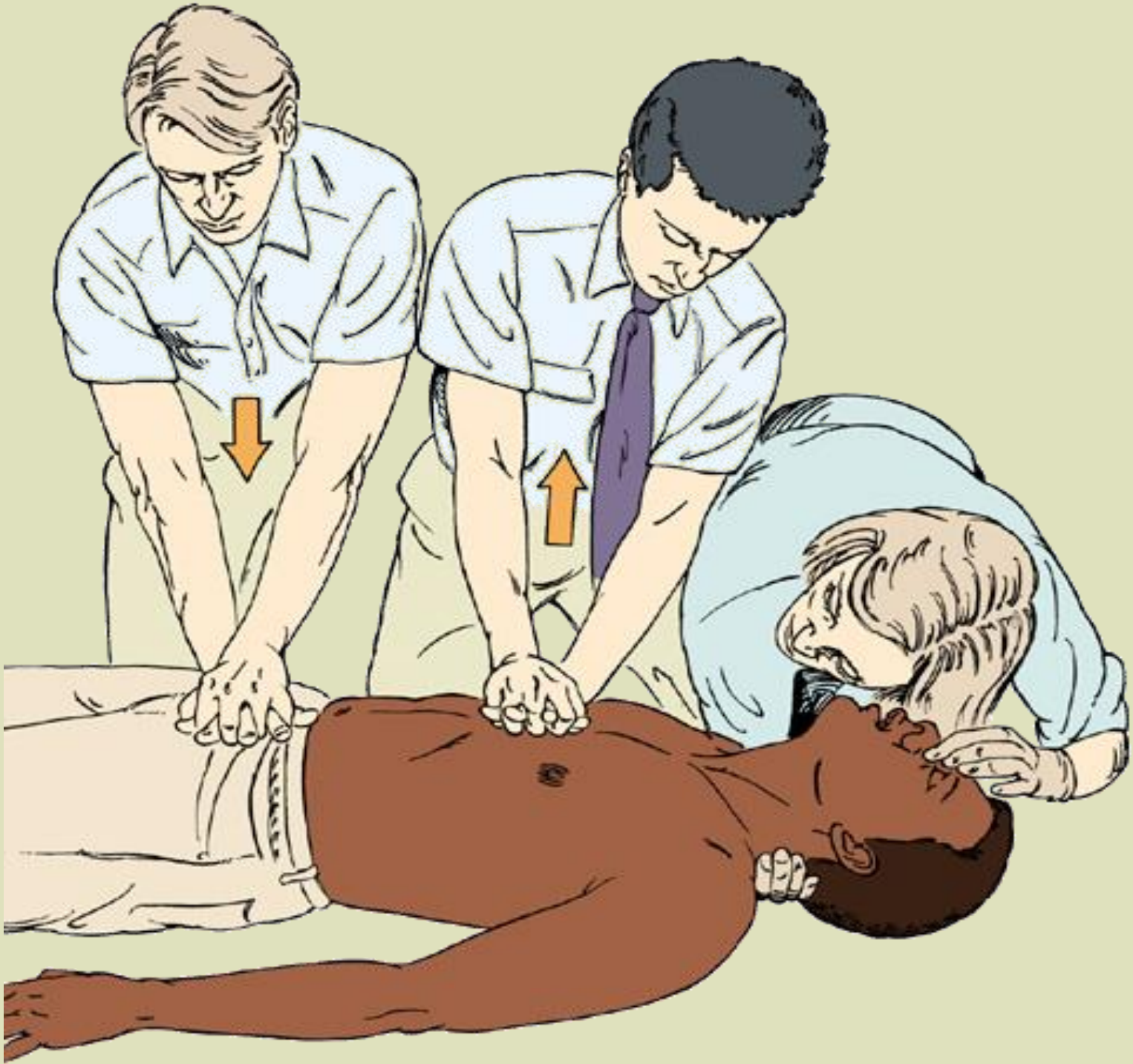

ZOLL
AED

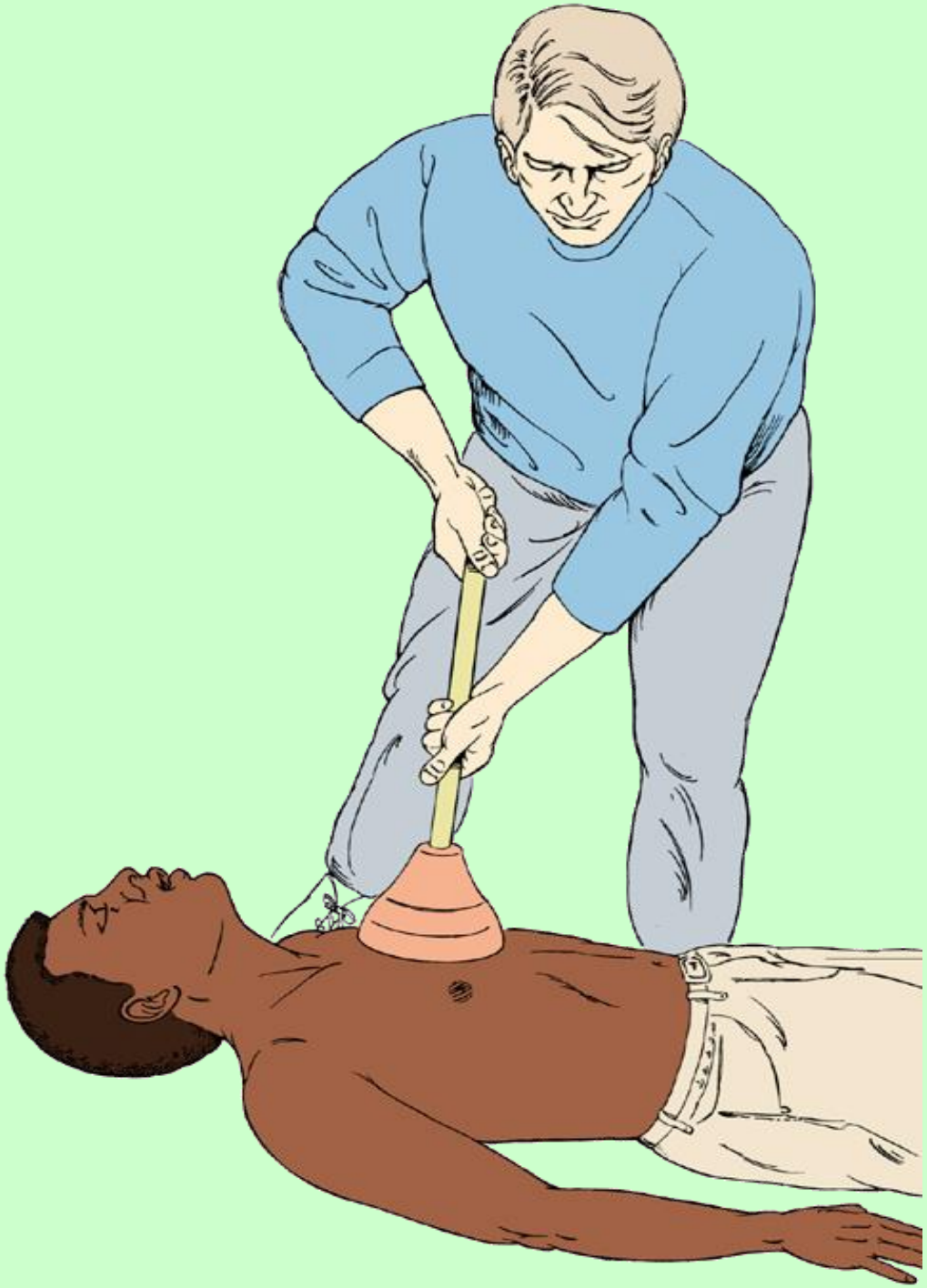

Right
Left

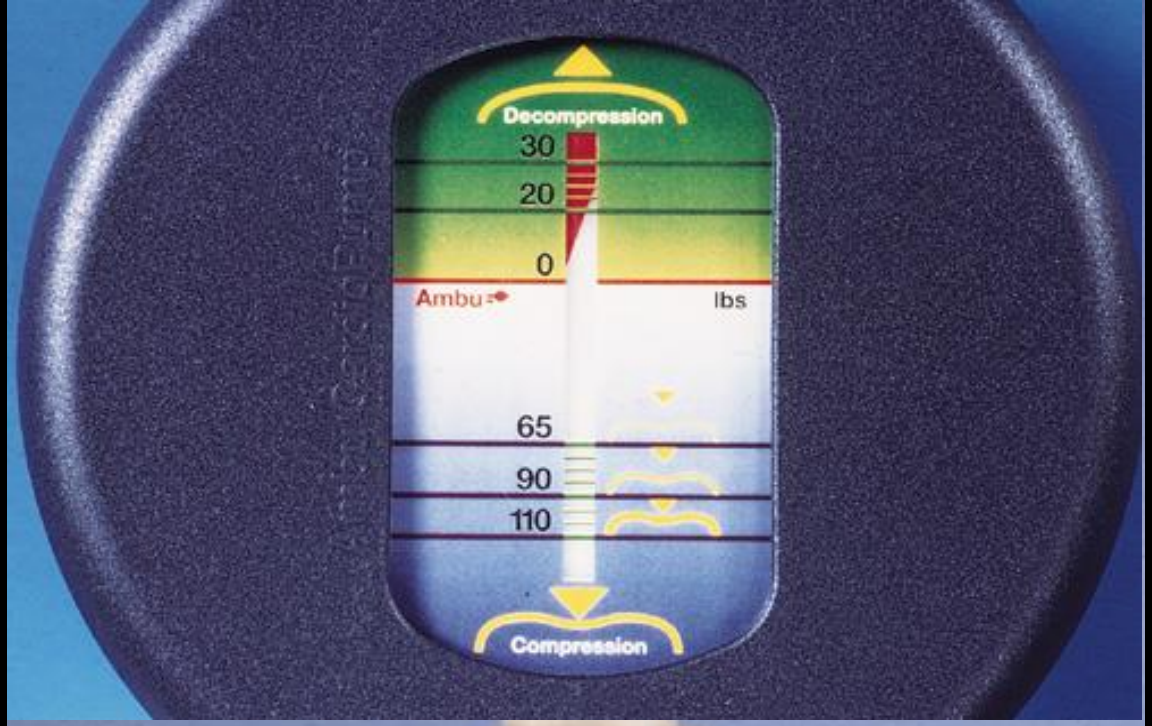

AED
AED

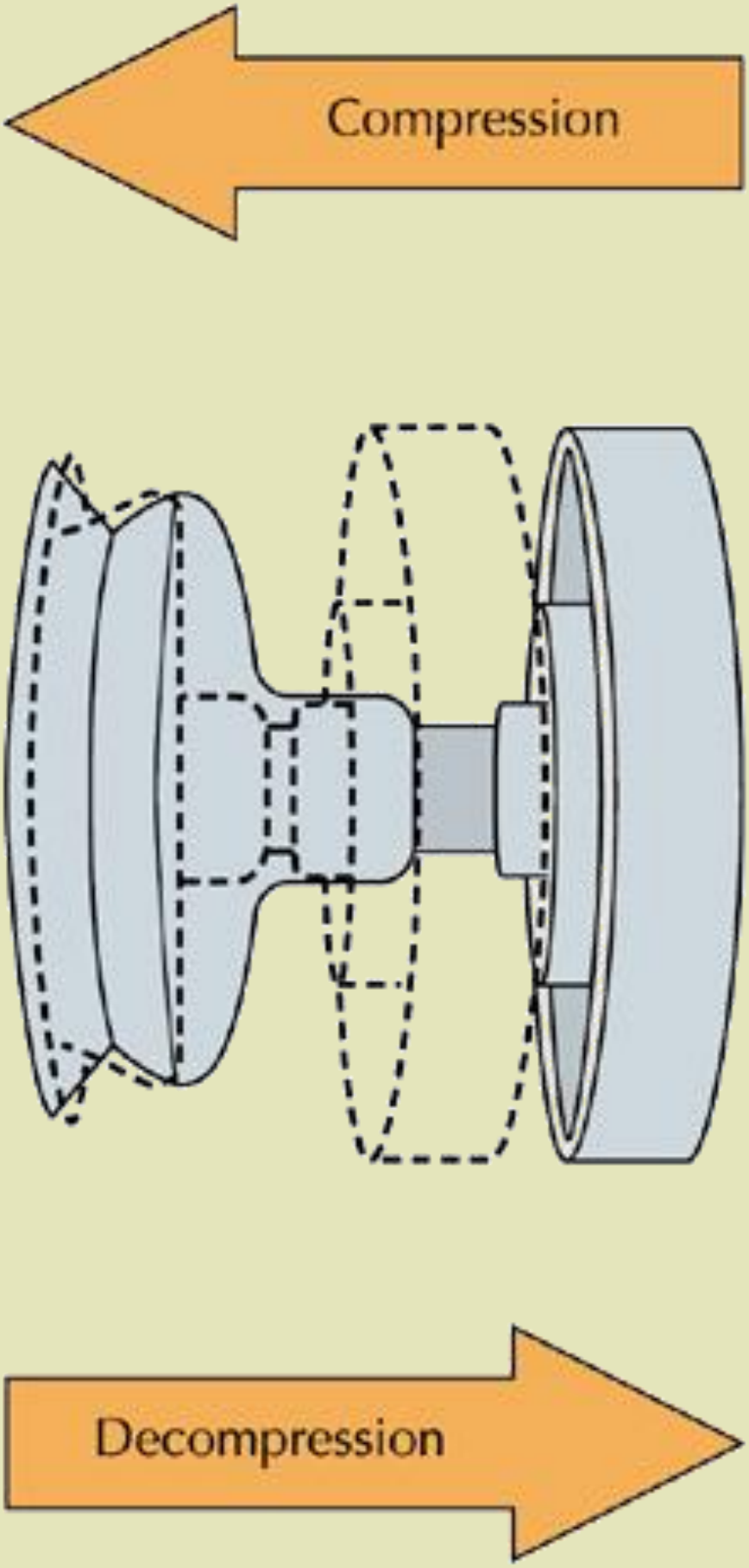

PRESSURE
FLOW CYCLE
ON
OFF








Decompression
30
20
0
Ambu
lbs
65
90
110
Compression


Compression
Decompression

Open-chest cardiac massage (*OCCM*)

1. There are clear indications of inadequate blood flow during standard CPR

2. Duration of arrest is less than 20 minutes

3. Clinician judges that potential exists for good neurologic outcome

Monitoring

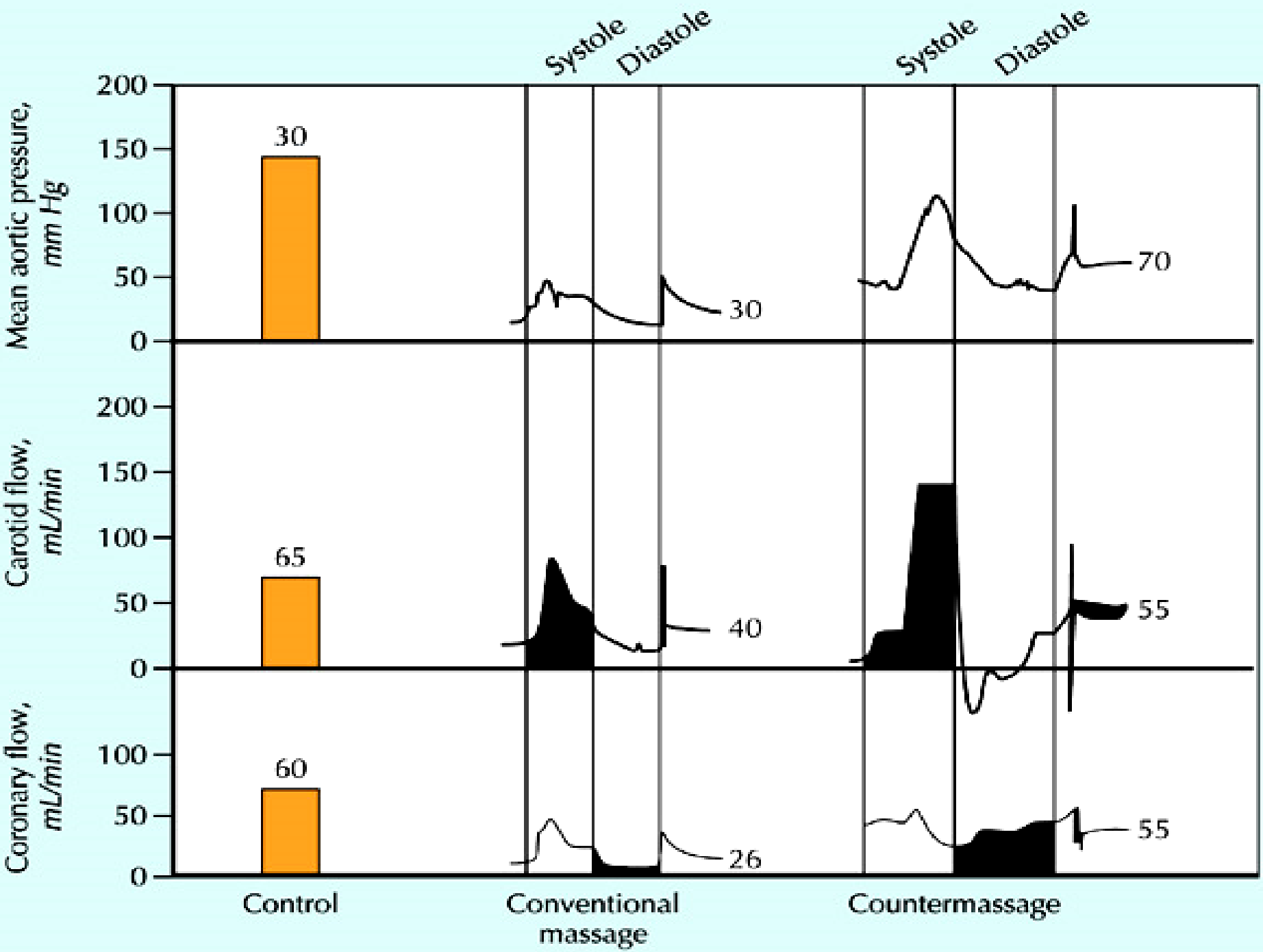
- **Arterial Blood Pressure and Coronary Perfusion Pressure**

  minimum CPP → 15 mm Hg

  arterial diastolic pressure → 40 mm Hg

- **$PetCO_2$** is a reliable indicator of cardiac output (CO) during CPR

  (**tension pneumothorax pericardial tamponade hypovolemia**)


Systole
Diastole
Systole
Diastole
Mean aortic pressure, mm Hg
Carotid flow, mL/min
Coronary flow, mL/min
200
150
100
50
0
30
30
70
65
40
55
60
26
55
Control
Conventional massage
Countermassage

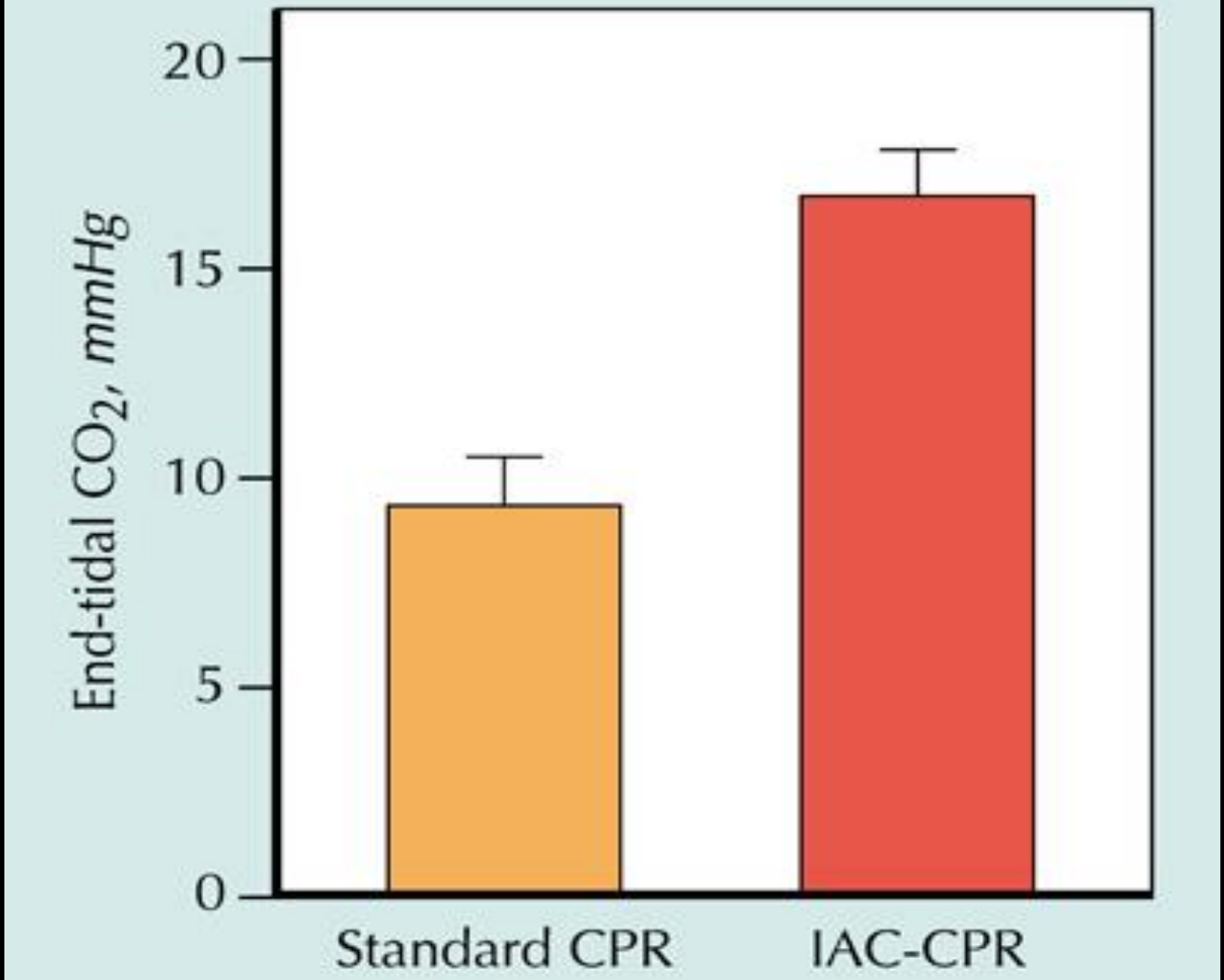
Monitoring

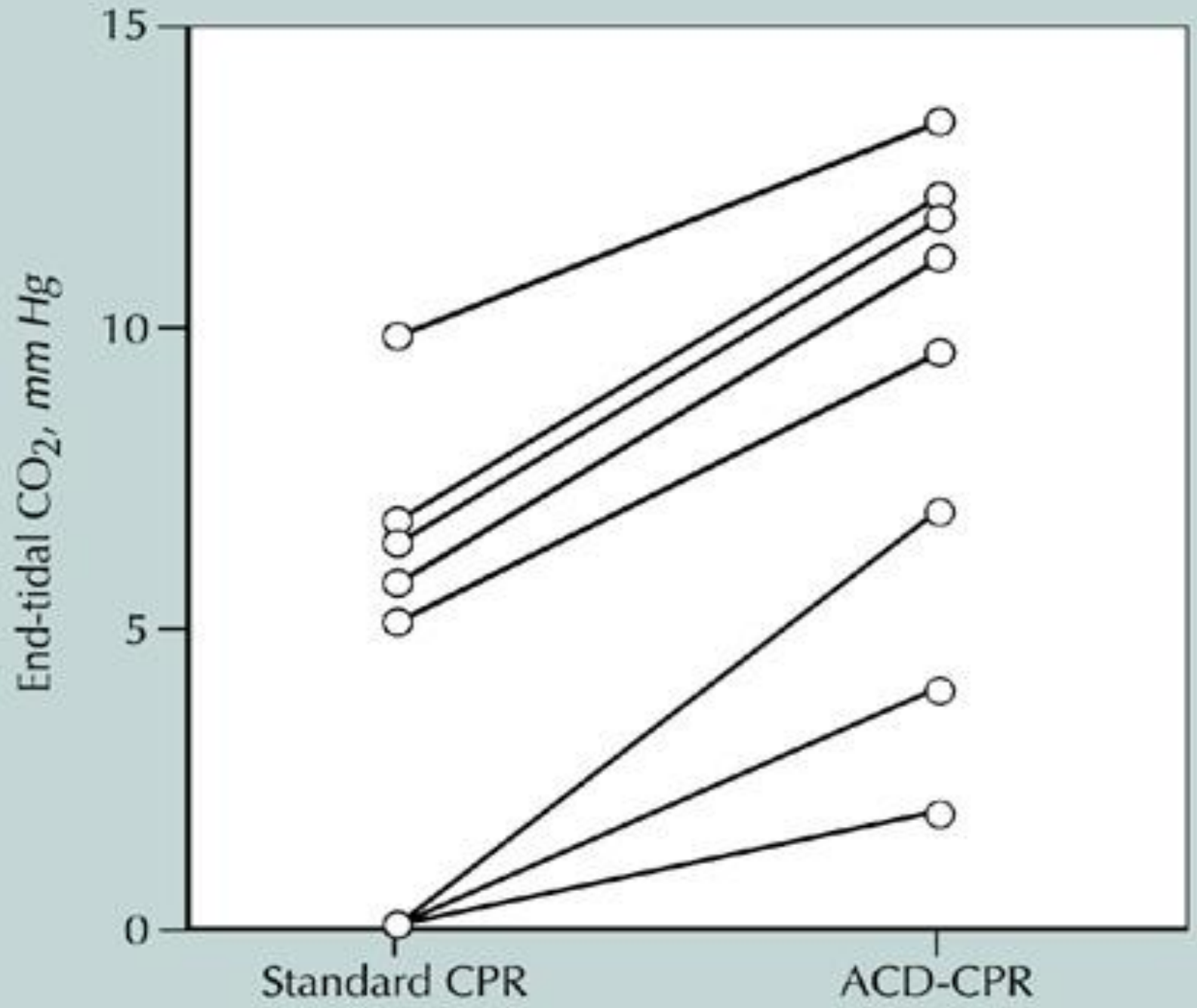
- **Central Venous Oxygen Saturation**
  ScvO2 values normally range from 60% to 80%

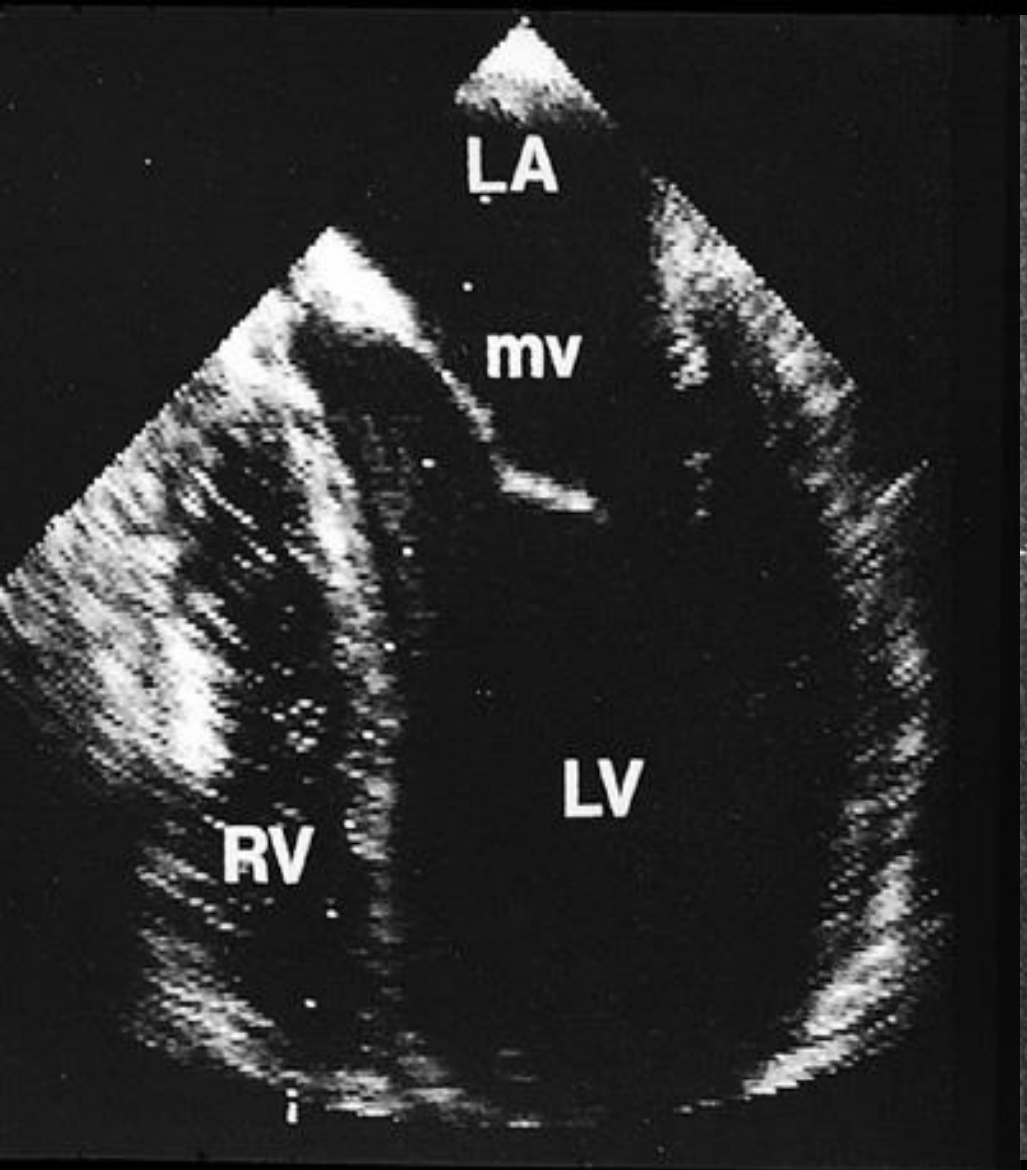
- **Echocardiography** Especially in patients with PEA helpful in diagnosing mechanical causes of PEA
- **Laboratory Testing**
  Intermittent arterial and venous blood sampling
  SaO2 is usually greater than 94% during CPR and of little value in titrating resuscitation therapy except in the case of massive PE or unrecognized esophageal intubation Serum electrolytes

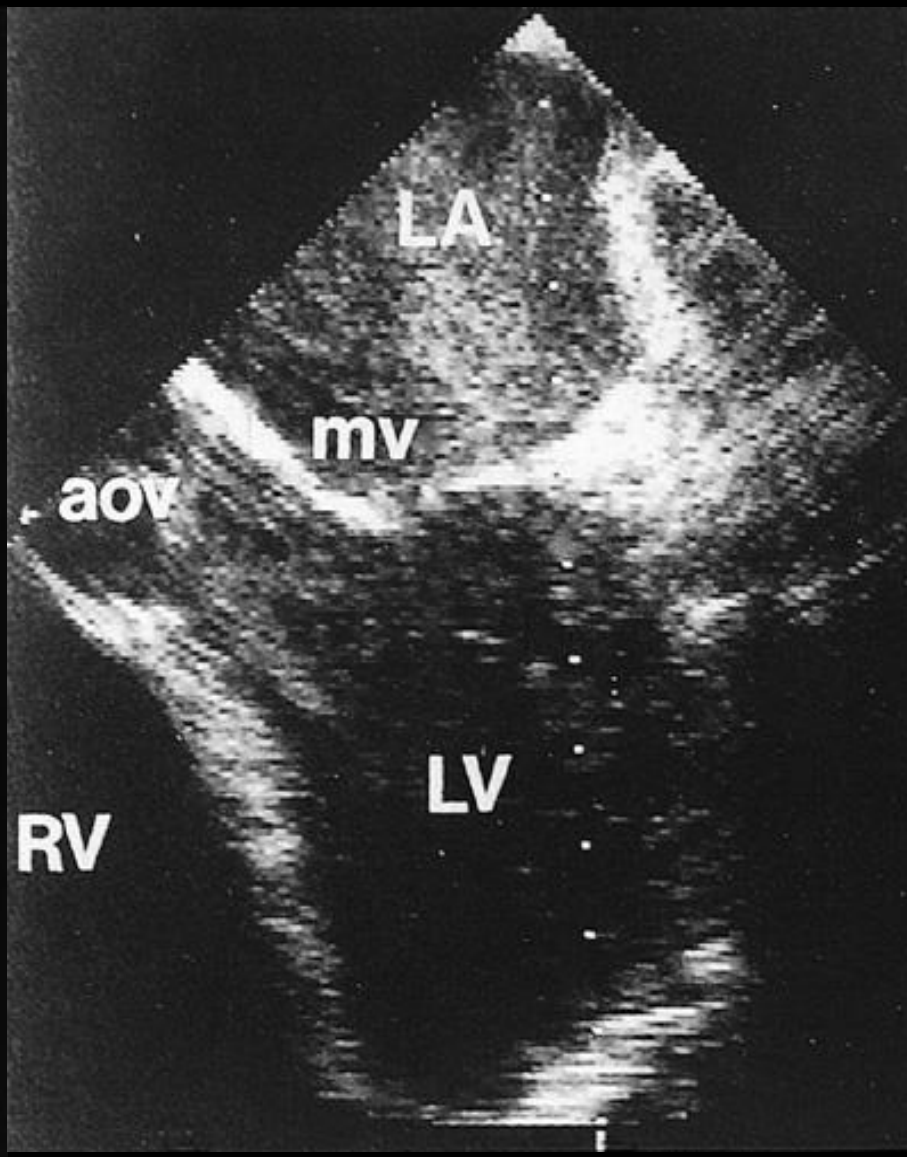
Monitoring

Monitoring technique	Indicator
Carotid or femoral pulse	Not palpable
CPP	<15 mm Hg
PetCO2 (before vasopressor)	<10 mm Hg
ScvO2	<40%


20
15
10
5
0
End-tidal CO_2, *mmHg*
Standard CPR
IAC-CPR


15
10
5
0
End-tidal CO_2, *mm Hg*
Standard CPR
ACD-CPR


LA
mv
LV
RV


LA
mv
aov
LV
RV

Common complications of standard CPR

1. **Aspiration**, *resulting in hypoxia, pneumonia, and adult respiratory distress syndrome (ARDS)*
2. **Hepatic trauma**, *which can lead to rapid exsanguination*
3. **Gastroesophageal damage**, *which carries the risk of fatal mediastinitis or hemorrhage*
4. **Cardiac trauma**, *including cardiac contusion, hemopericardium, and pericardial effusions*
5. **Bone trauma**, *including rib and sternal fractures, resulting in hemothorax and bone marrow emboli*

Defibrillation

- **Timing** AS soon as possible

  *-arrest durations longer than **4** minutes , reenergizing the heart before defibrillation*

- **Technique**

  -optimal current for defibrillation is 30 to 40 amps

  *-initial countershock of **200 watt-sec** is effective in most patients in VF*

  *-if unsuccessful is rapidly followed by a second countershock at **200 to 300 watt-sec** and a third at **360 watt-sec** without intercedent CPR*

  *-transthoracic impedance declines with rapid sequential countershocks **(8% with second shock)***

  *-Countershock with a **biphasic** current waveform*

  less energy required for successful defibrillation

  reduced postcountershock myocardial dysfunction

Defibrillation

- *The **precordial thump** has been reported to have a **10% to 25%** efficacy in converting **pulseless VT** to a perfusing rhythm.* **VF, however, is rarely converted by this method**. *Administration of a precordial thump is an optional technique **if an arrest is witnessed and no defibrillator is available***
- However, a precordial thump should not be administered to patients with VT and a pulse ***in the absence of a defibrillator*** because it can result in deterioration to a more malignant rhythm such as VF, asystole, or EMD.

Pharmacologic Therapy

- **Drug Administration Routes**
  peripheral and central venous, endotracheal, intraarterial, intracardiac, and intraosseous
- **by ET tub(epinephrine, atropine, and lidocaine)**
  -2 to 2.5 times the recommended dosage
  -diluted in 10 ml of normal saline or distilled water
  -passing a catheter beyond the tip of the ET tube
  -temporarily holding chest compressions
  -several deep breaths with the ambu-bag

peripheral and central venous

- **internal jugular** or **supraclavicular** routes are preferred over the **subclavian** route because CPR does not have to be interrupted

- Because subdiaphragmatic blood flow is minimal during CPR, **femoral vein** cannulation should be avoided unless a cannula long enough to pass above the diaphragm is used

- **Intracardiac** injection is recommended only when other routes are not readily available or during **open-chest CPR**, when the heart can be directly visualized

Antidysrhythmics

- ***Amiodarone*** is considered a class III antidysrhythmic agent but has characteristics of all four classes
- Significant side effects of amiodarone therapy included **bradycardia** and **hypotension**
- The **300-mg** bolus given during cardiac arrest is expected to provide sufficient levels for **30 minutes**
- amiodarone may become the **first-line antidysrhythmic** in the treatment of **refractory VF and pulseless VT**.

Antidysrhythmics

- ***Lidocaine*** is an amide local anesthetic and class IB antidysrhythmic agent.
- Recommended as the **first-line antidysrhythmic** for treatment of VF and pulseless VT refractory to defibrillation
- **Increases the VF threshold**, which decreases the likelihood of fibrillation
- **Increases the defibrillation threshold**, or minimum electrical dose, required for defibrillation
- The dosage of lidocaine during VF or pulseless VT refractory to defibrillation is a ***1.5-mg/kg initial bolus*** followed by ***0.5-to 1.5-mg/kg boluses every 5 to 10 minutes*** if necessary, up to a total dose of ***3.0 mg/kg***

Antidysrhythmics

- *Magnesium deficiency is associated with* **sudden cardiac death**, *and hypomagnesemia can precipitate* **refractory VF**
- *In the event of* **torsades de pointes** *and severe magnesium deficiency,* **1 to 2 g** *of magnesium sulfate diluted in 100 ml of 5% dextrose in water (D5W) may be administered over* **1 to 2 minutes**

Atropine

- Atropine acts as a competitive antagonist of acetylcholine (ACh) at the **muscarinic receptor**.
- The use of atropine in **bradyasystolic arrest** is based on the belief that parasympathetic tone is increased as a result of vagal stimulation, possibly resulting from **hypoxia and acidosis** of the carotid body
- Initial treatment is with atropine, **0.5 to 1.0 mg** intravenously and repeated as needed at **3- to 5-minute** intervals to **0.04 mg/kg**

Administration of Adenosine

- **Adenosine** induces slowing of AV nodal conduction and prolongation of refractoriness and therefore is very effective in **terminating PSVT**, the most common cause of which is reentry within the AV node
- **Peripheral** (antecubital): 6 mg, then 12 mg if needed
- **Central**: 3 mg, then 6 mg if needed
- If taking **theophylline**-containing drugs: 9 mg peripherally, 6 mg centrally
- If taking **dipyridamole**: 2 mg peripherally, 1 mg centrally
- Use with caution in **asthmatic** patients
- Caution in patients taking **carbamazepine**

Adenosine Antagonists

- **Hypoxia and ischemia lead to the interstitial accumulation of adenosine in the myocardium.**
- **Adenosine is known to depress automaticity of the sinus node, AV junction, His bundle, and Purkinje fibers, as well as conduction through the AV node.**
- ***Aminophylline*** is a competitive antagonist of adenosine. Aminophylline **(250 mg IV)** given to patients who fail ACLS with more than 2 mg of epinephrine and 2 mg of atropine

Buffers

- Controversy surrounds the use of buffer therapy in CPR
- Correct persistent metabolic acidosis
- Improving vascular response to pressors
- Lowering the ventricular defibrillation threshold

In cases of

hyperkalemia,

tricyclic antidepressant overdose

after prolonged untreated cardiac arrest (10 minutes or longer)

prolonged CPR (10 minutes or more)

in conjunction with repeated or large doses of epinephrine (0.05 mg/kg or more)

Buffers

- When indicated, the recommended dosage of $NaHCO_3$ is **0.5 to 1 mEq/kg** every **10 minutes** during CPR or as guided by arterial blood gases

Calcium Chloride

- Calcium administration is likely to be beneficial in cases of **hyperkalemia**, **hypocalcemia**, **calcium channel blocker toxicity**
- If required, **4 mg/kg** of calcium chloride (0.04 ml/kg of 10% solution) may be administered every **10** minutes

Ventricular Fibrillation & Pulseless Ventricular Tachycardia

- **Both VF and pulseless VT are treated identically**
- A patient who develops VF or pulseless VT while on a cardiac monitor may remain ***conscious for 15 to 30 seconds***. The patient should be encouraged to ***cough vigorously*** until a defibrillator is available .
- If the duration of untreated arrest is prolonged ***(>4 minutes)*** or unknown, a brief period of CPR (60 to 90 seconds) before defibrillation may help achieve ROSC

Ventricular Fibrillation & Pulseless Ventricular Tachycardia

- Cough vigorously (at least one cough every 2 seconds)
- Defibrillation without antecedent CPR
- Chest compression
- Intravenous (IV) access and intubation
- Vasopressor therapy
  (epinephrine or vasopressin)
- Antidysrhythmic agent
  (e.g., amiodarone)
- Defibrillation should be repeated 2 minutes after Vasopressor therapy

Ventricular Fibrillation & Pulseless Ventricular Tachycardia

- **Magnesium sulfate**
  Torsades de pointes and suspected hypomagnesemia
- **NaHCO3**
  hyperkalemia, tricyclic antidepressant overdose, and preexisting metabolic acidosis.
  prolonged cardiac arrest (>10 minutes)
  prolonged CPR (>10 minutes)
  after high dosages of epinephrine (>0.05 mg/kg)

Pulseless Electrical Activity

- PEA is defined as **coordinated electrical activity** of the heart (other than VT/VF) **without a palpable pulse.**
- This group of dysrhythmias includes **EMD**, in which no myocardial contractions occur, and **pseudo-EMD**, in which myocardial contractions occur but no pulse can be palpated
- **In most cases of primary PEA there is a natural progression from hypotension to pseudo-EMD to EMD.**

Pulseless Electrical Activity

- It is most often associated with ***global myocardial energy depletion*** and ***acidosis*** resulting from ***ischemia or hypoxia***.
- True EMD typically occurs
  - ***after defibrillation***
  - ***hyperkalemia,***
  - ***hypothermia,***
  - ***drug overdose.***

Pulseless Electrical Activity

- ***Cardiac cause of pseudo-EMD :***

  Papillary muscle rupture
  Myocardial wall rupture

- ***extracardiac causes of pseudo-EMD :*** **Hypovolemia,**
  Tension pneumothorax,
  Pericardial tamponade,
  Massive PE

Diagnosis and Treatment of Common Causes of Pulseless Electrical Activity

Cause	Diagnosis	Palliative therapy	Definitive therapy
Hypovolemia	Response to volume infusion	Volume infusion, consider OCCM	Hemostasis if hemorrhage
Hypoxia	Response to oxygenation	Oxygenation, assisted ventilation	Treat underlying cause
Cardiac tamponade	Echocardiography, pericardiocentesis	Pericardiocentesis	Thoracotomy and pericardiotomy
Tension pneumothorax	Asymmetric breath sounds, tracheal deviation	Needle thoracostomy	Tube thoracostomy
Hypothermia	Rectal temperature		Warm peritoneal or thoracic lavage, OCCM or CPB

Diagnosis and Treatment of Common Causes of Pulseless Electrical Activity

Cause	Diagnosis	Palliative therapy	Definitive therapy
Drug overdose	History of drug ingestion	Drug specific	Drug specific
Hyperkalemia	History of renal failure or elevated serum potassium	Calcium chloride, insulin and glucose, sodium bicarbonate	Hemodialysis
Acidosis	Arterial blood gas	Hyperventilation, sodium bicarbonate	Treat underlying cause
Pulmonary embolus	Risk factors or evidence of deep venous thrombosis	OCCM or CPB	Lytic therapy, pulmonary embolectomy

Asystole

- Confirmed in at least two limb leads.
- Extremely fine VF
- Asystole may occur early in cardiac arrest
- Asystole generally represents the end-stage rhythm after prolonged cardiac arrest caused by VF or PEA

Treatment of asystole

All general resuscitation measures,

- **CPR**
- **Intubation with assisted ventilation**
- **IV access**
- **Repeated administration of vasopressors**
- **Atropine with the first dose of vasopressor**

  and repeated up to a total dose of 0.04 mg/kg
- **Pacing must be initiated within several minutes of arrest**