CPR

And cardiac events in athlets

Dr Sirous Krdrostami

Special consideration in athlets

The athlete's heart

- Systematic engagement in regular exercise results in predictable changes to the cardiac architecture, termed "structural" and "functional" remodelling. The physiologic adaptations that occur in response to exercise are often evident on standard 12-lead electrocardiography. Such findings are considered normal in an athlete and typically do not warrant further investigation.

Exercise-induced remodelling

- Exercise-induced cardiac remodelling occurs as an adaptation to permit and enhance the increased cardiac output required during exercise.
- Such changes are specific to the exercise loads encountered in a particular sport.

Structural remodelling

- myocardial wall thickness (concentric hypertrophy)
- chamber size (eccentric hypertrophy)
- This results in normalization of the left ventricular mass-to-volume ratio with prolonged training.
- Functional remodelling results in higher vagal tone and slowing of resting heart rate, more rapid heart rate recovery after exercise and improved diastolic function.
- **Does physiologic remodelling from exercise cause harm?**

Does physiologic remodelling from exercise cause harm?

- In a small minority the degree of structural adaptation resembles structural changes seen in pathologic states
- hypertension-induced left ventricular hypertrophy
- hypertrophic cardiomyopathy
- arrhythmogenic right ventricular cardiomyopathy.

Causes of sudden cardiac arrest in athletes

- **Age group ≤ 35 yr**
- Primary electrical disease with no specific cause identified
- Idiopathic left ventricular hypertrophy
- Anomalous origin of the coronary arteries
- Heritable cardiomyopathies:
  - Hypertrophic cardiomyopathy
  - Arrhythmogenic right ventricular cardiomyopathy
- Myocarditis
- Electrical disorders:
  - Long QT syndrome, catecholaminergic polymorphic ventricular tachycardia

Causes of sudden cardiac arrest in athletes

- **Age group > 35 yr**
- Coronary artery disease
- Idiopathic left ventricular hypertrophy
- Heritable cardiomyopathies:
  - Hypertrophic cardiomyopathy
  - Arrhythmogenic right ventricular cardiomyopathy
- Primary electrical disease with no specific cause identified
- Myocarditis

Definition of sudden cardiac death in athletes

SCD = sudden and unexpected death occurring during, or shortly after, exercise (with varying time intervals up to 24 h used by different investigators), if witnessed by a bystander and/or happening in an individual who was otherwise known to be healthy.

Incidence of SCD in athletes

- Lack of rigorous studies
- Best estimate is 1–2/100,000 athletes aged 12–35 years [1–4]
- A robust Italian study suggested incidence 3x higher in athletes vs. non-athletes,[2] but a Danish study found decreased rates of SCD in athletes [5]
- Incidence similar in competitive and recreational athletes [4]
- 2–25x higher incidence in males than females [1–6]
- 5–10x higher incidence in age >35 years [1,4–6]
- High incidence in black athletes (5.5/100,000) and male basketball players (>10/100,000) [1,6]

Causes of SCD in athletes

Hypertrophic cardiomyopathy (HCM)

- HCM is defined as left ventricular hypertrophy (LVH) that cannot be solely explained by abnormal loading conditions
- HCM prevalence is 1:500 in European, American, Asian and African populations [1,2]
- Clinical manifestations include heart failure (HF) and atrial fibrillation, although patients can be asymptomatic and SCD may the be first manifestation [2]
- In most series, HCM is the most common cause of SCD in young people engaged in competitive sport [3,4]

Causes of SCD in athletes

Arrythmogenic right ventricular cardiomyopathy (ARVC)

- ARVC is a progressive inherited heart muscle disease causing ventricular electrical instability that may lead to arrhythmic SCD
- Prevalence 1:1,000 to 1:5,000 [1]
- Characterised by progressive loss of myocardium, myocyte death and fibro-fatty scar, which predispose to ventricular arrhythmia [1,2]
- Competitive sports confer 5x increased risk of SCD in young people with ARVC [3]
- Exercise may accelerate disease progression [4,5]

Causes of SCD in athletes

Coronary congenital abnormalities

- Most common are coronary arteries arising from anomalous sinus in aortic root
- Affects 0.5–1.0% of individuals [1]
- Most are clinically silent, but some develop ventricular ischaemia, and SCD is the first manifestation in a minority of cases [1]
- Higher risk of SCD in patients with left coronary artery originating in right coronary sinus or coronaries coursing between aorta and pulmonary artery [1]
- Exercise may trigger ventricular ischaemia, electrical instability and VF
- Some studies suggest this is the 2nd most common cause of SCD in athletes [2,3]
- Nearly half of athletes who suffer SCD from coronary arteries with anomalous origin were previously asymptomatic [4]

Causes of SCD in athletes

Ventricular pre-excitation

- Atrioventricular (AV) bypass tracts cause early, anomalous ventricular activation before normal activation through the AV node
- Estimated prevalence 1:1,000 [1]
- Can be asymptomatic or present with paroxysmal supraventricular tachycardia symptoms – palpitations, syncope
- Symptomatic patients at high risk of SCD (≈0.15%/yr)
- Ventricular pre-excitation seen in ≈1% of athletes with SCD

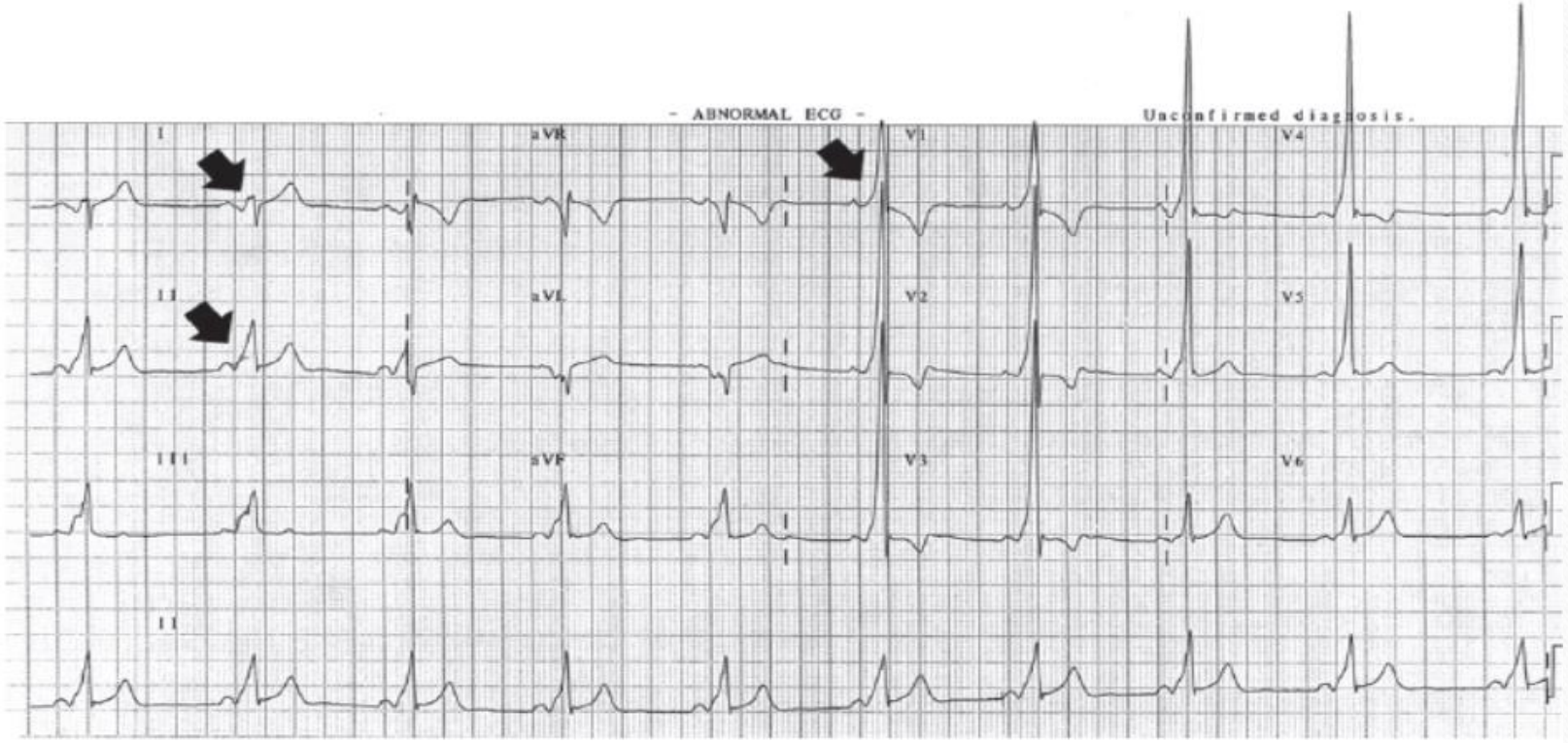
Causes of SCD in athletes

Ventricular pre-excitation: diagnosis

- ECG with characteristic delta-wave pattern
- Delta waves have slurred upstroke QRS complexes and short PR intervals (<120 ms)
- Pre-excitation may be masked in adrenergic settings when AV node conduction is fast

Causes of SCD in athletes

Representative ECG from a patient with a pre-excitation syndrome



Causes of SCD in athletes

Valvulopathies

- Bicuspid aortic valves are found in ≈1% of the general population[1] and confer risk of aortic stenosis or regurgitation, ascending aorta dilation and dissection
- Mitral valve prolapse is the most common cause of primary mitral regurgitation in young to middle-aged individuals
- Both may lead to LV dysfunction and ventricular arrhythmias during exercise
- No evidence that exercise accelerates valvulopathy progression, but valvulopathies are sometimes a cause of SCD in athletes
- Diagnosis by physical examination (cardiac murmur) and cardiac imaging test

Causes of SCD in athletes

Inherited primary arrhythmia syndromes

Genetically determined, primary arrhythmic disorders in absence of clinically determined structural heart disease.[1] These include:

- Long QT syndrome
- Short QT syndrome
- Catecholaminergic polymorphic ventricular tachycardia
- Brugada syndrome

Causes of SCD in athletes

Catecholaminergic polymorphic ventricular tachycardia (CPVT)

- Characterised by adrenergic-induced bidirectional or polymorphic ventricular tachycardia
- Prevalence around 1:10,000
- Two-thirds of cases due to known genetic mutations [1]
- Silent mutations in 20% [2]
- If undiagnosed, mortality rates are 30–50% by age 40 [1,2]
- Physical activity is a common trigger of ventricular arrhythmias in patients with CPVT
- Diagnosis – structurally normal heart, normal ECG. Effort or emotion trigger ventricular premature beats that increase in complexity with heart rate

Causes of SCD in athletes

Brugada syndrome

- Characterised by right bundle branch block, persistent ST segment elevation and sudden death due to polymorphic ventricular tachycardia (PVT) and/or VF in absence of other cardiomyopathies.[1] May be accompanied by mild RV abnormalities
- Inherited disease, but may be sporadic in up to 60% of cases [2]
- Prevalence of 1:1,000 in Asia, but <1:10,000 in Europe and America [3]
- Ventricular arrhythmias generally occur at rest. Chronic athletic conditioning or raised body temperature during exercise may exacerbate the condition

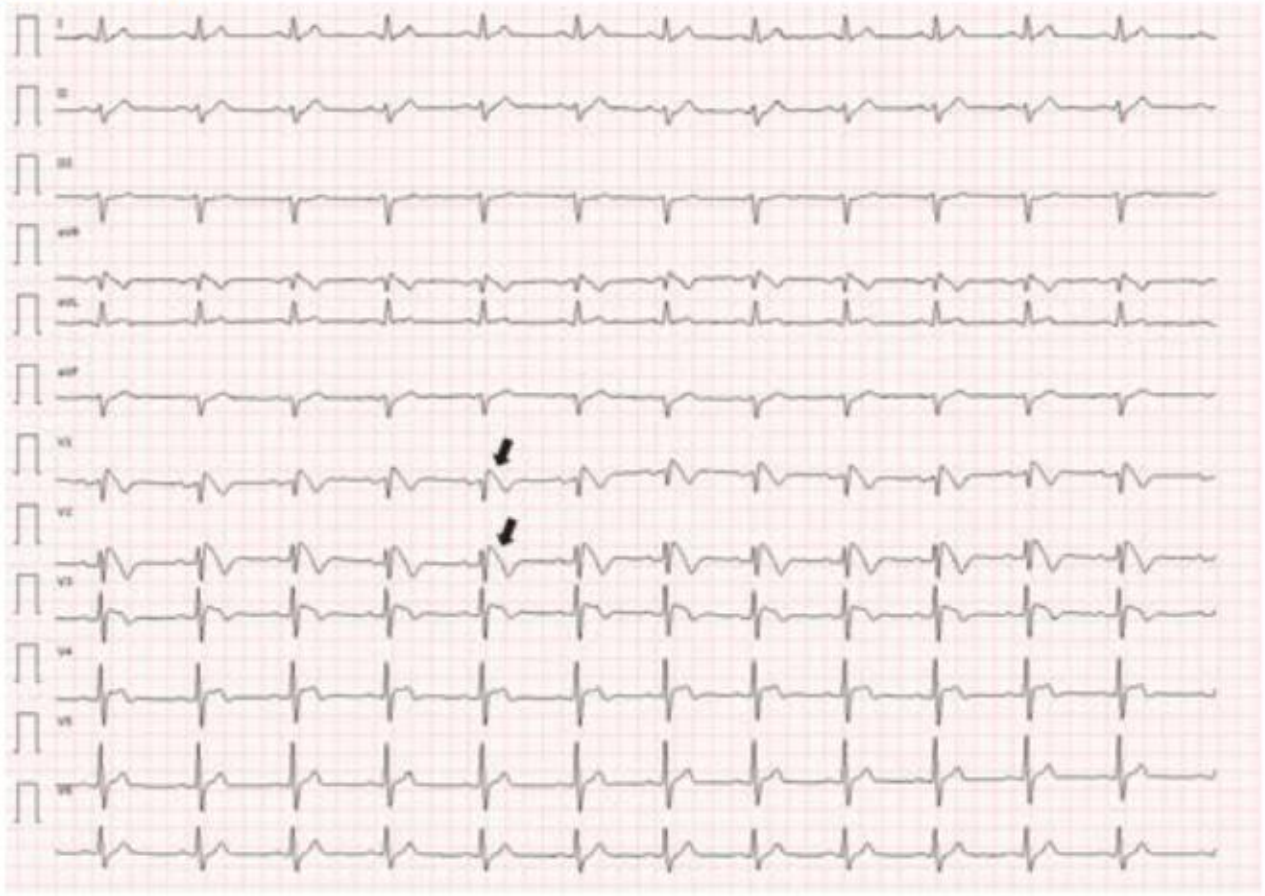
Causes of SCD in athletes

Brugada syndrome: diagnosis

- Brugada syndrome is diagnosed in the presence of type 1 ST elevation either spontaneously or after intravenous administration of a sodium channel blocker in at least one right precordial lead (V1 and V2), placed in a standard or superior position (up to the second intercostal space)
- Other signs might also be present:
  - Attenuation of ST elevation during exercise with reappearance during recovery
  - First-degree AV block and left axis deviation
  - Atrial fibrillation
  - Late potentials in high resolution ECG
  - QRS fragmentation
  - Ventricular refractory period <200 ms and HV interval >60 ms in electrophysiological study
  - Absence of diagnosis of alternative cardiomyopathy

Causes of SCD in athletes

Brugada syndrome



- Survival to hospital discharge presently approximately 5-10 - 14%
- Bystander CPR = vital intervention before arrival of emergency services
- Early resuscitation and prompt defibrillation (within 1-2 minutes) can result in >60% survival

- In first **4 minutes** – brain damage is unlikely, if CPR started
- **4 – 6 minutes** – brain damage possible
- **6 – 10 minutes** – brain damage probable
- **> 10 minutes** – severe brain damage certain

Cells of the brain cortex

- Most sensitive for the stop of pefusion and oxygenation

Without perfusion and oxygenation

➔ **irreversibly damaged after 3-5 minutes**

Ventricullar fibrilation – better than asystole
- in case of immediate CPR

Special emphasis

⬇

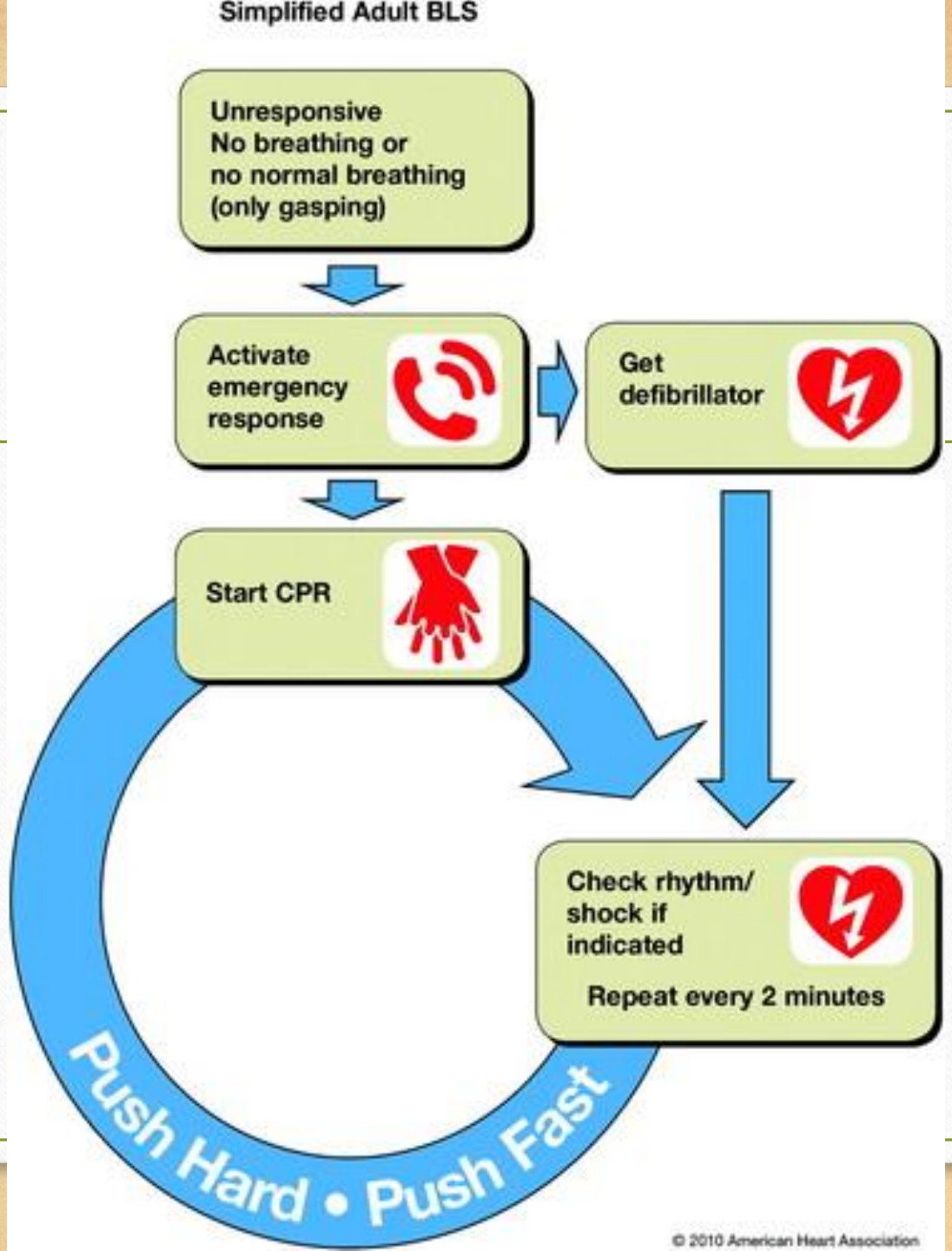
Soon defibrilation

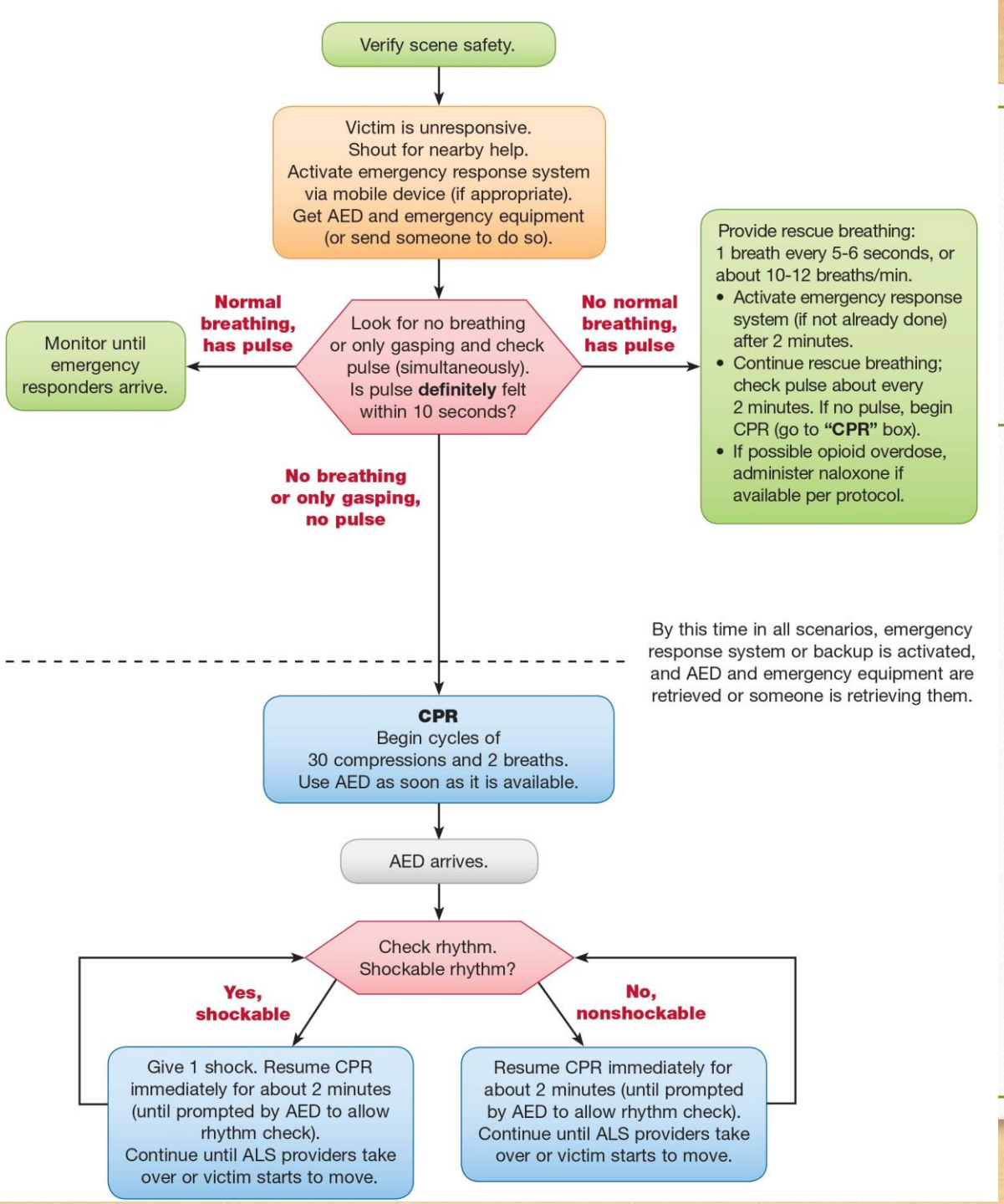
- **1 minute - survival - 90%,**
- **5 minutes - survival - 50%,**
- **7 minutes - survival - 30%**
- **10 - 12 minutes - survival - 2 – 5%.**

Management

- (1) initial assessment by a witness/bystander and summoning of an emergency response team
- (2) basic life support (BLS)
- (3) early defibrillation by a first responder (if available)
- (4) advanced life support(ALS,ACLS)
- (5) post–cardiac arrest care

BLS

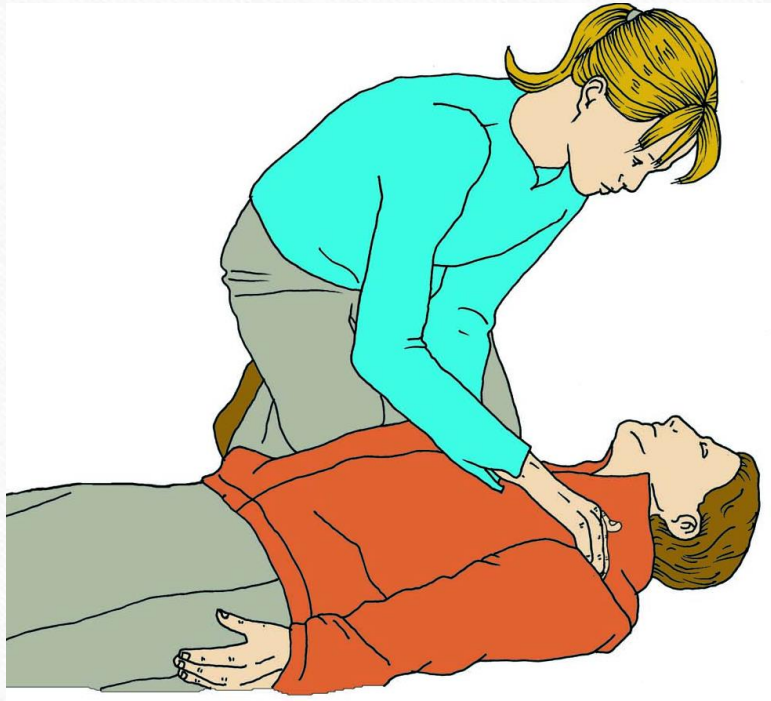

Simplified Adult BLS
Unresponsive
No breathing or
no normal breathing
(only gasping)
Activate
emergency
response
Get
defibrillator
Start CPR
Check rhythm/
shock if
indicated
Repeat every 2 minutes
Push Hard • Push Fast
© 2010 American Heart Association


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
No, nonshockable
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.
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.

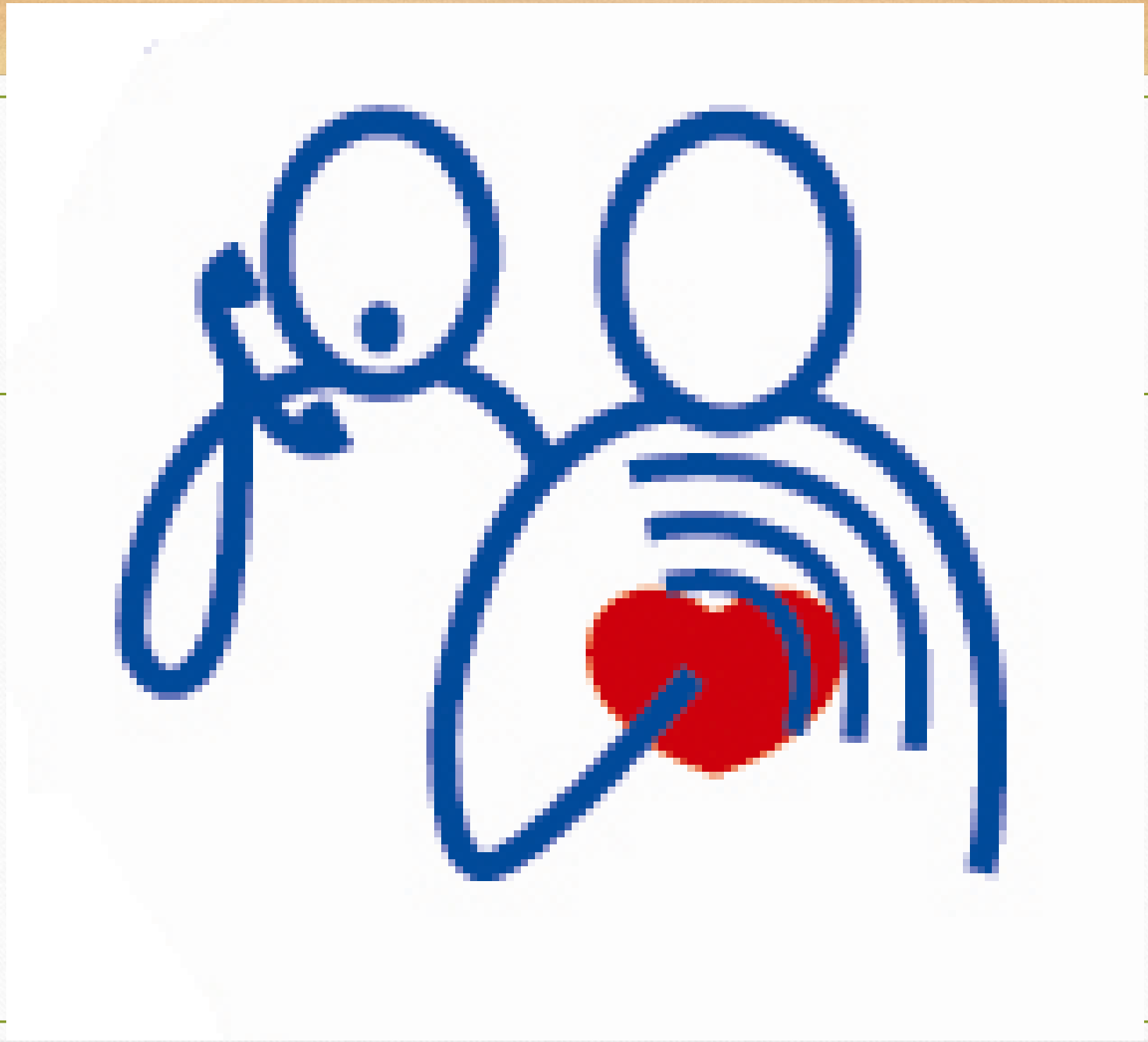
SRH

- Safety
- Responsiveness
- Help

Responsiveness



Help



BLS

- environmental safety of the site
- confirm that the victim is unresponsive
- call for nearby help
- activate an emergency response system and send for an AED.
- The previous sequence of the “ABC” of basic life support—airway, breathing, compression—has been changed to “CAB”—compression, airway, breathing—based on the recognition that compression alone is the better strategy

CPR is as easy as

C-A-B

Compressions

Push hard and fast on the center of the victim's chest

Airway

Tilt the victim's head back and lift the chin to open the airway

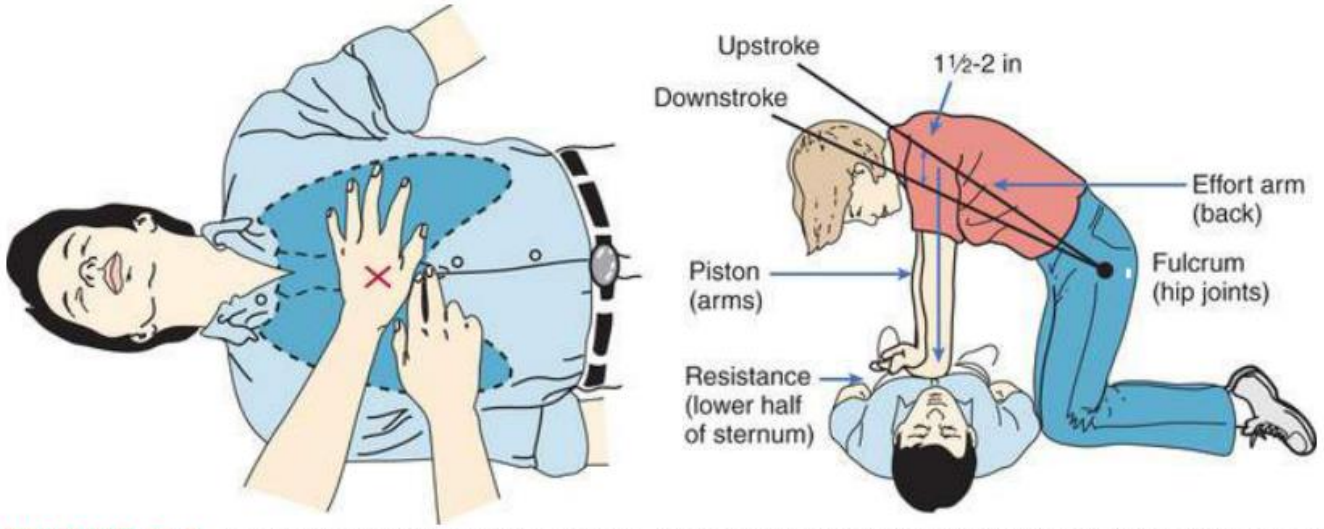
Breathing

Give mouth-to-mouth rescue breaths

American Heart Association

Learn and Live

Circulation

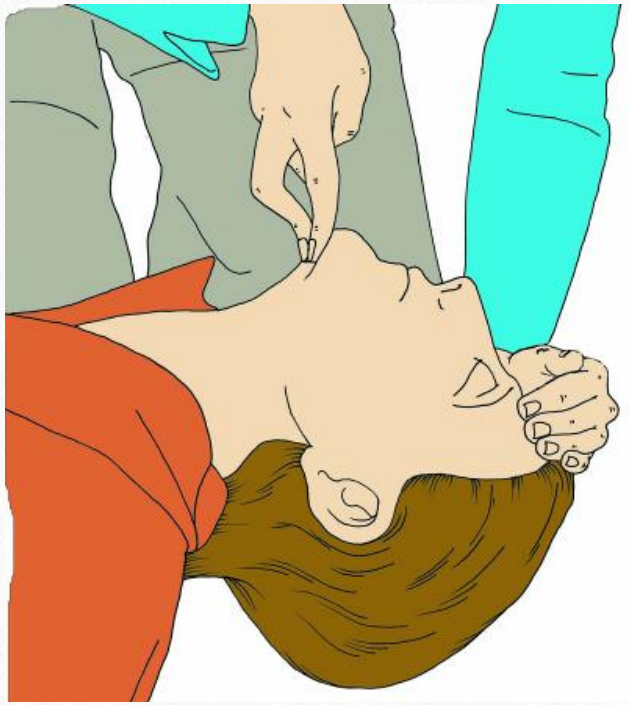


EFIGURE 42.4 External chest compression. **Left,** Locating the correct hand position on the lower half of the sternum. **Right,** Proper position of the rescuer, with the shoulders directly over the victim's sternum and the elbows locked. (From National Academy of Sciences, National Research Council. Standards and guidelines for cardiopulmonary resuscitation [CPR] and emergency cardiac care [ECC]. JAMA 1986;255:2906.)

Circulation

- Place the heel of one hand in the centre of the chest
- Place other hand on top
- Interlock the fingers
- **Compress the chest**
  - **Rate 100 min^{-1}**
  - **Depth 4-5 cm**
  - **Equal compression : relaxation**
- **When possible (2 or more rescuers) change CPR operator every 2 min. to prevent fatigue**

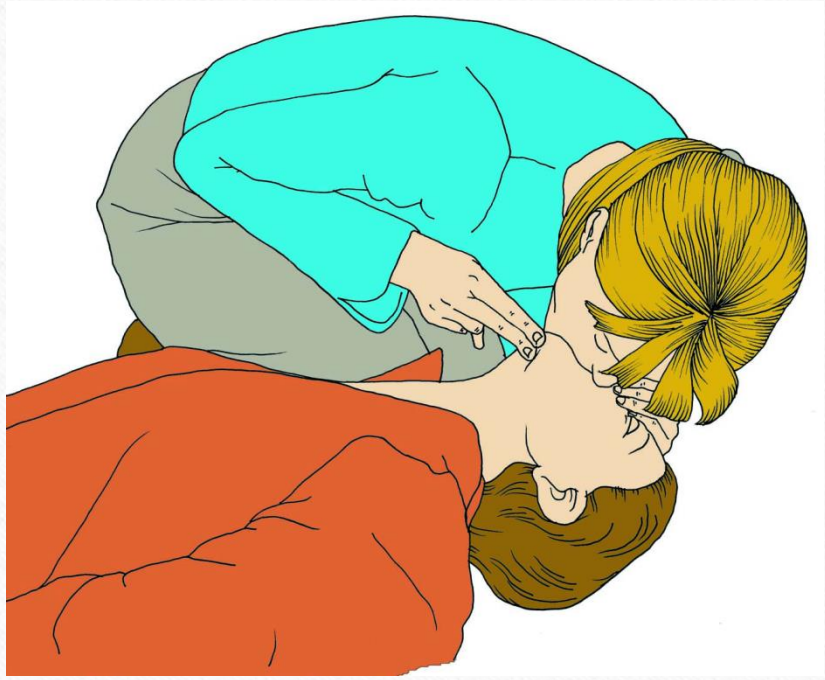
Airway



Airway

- tilting the head backward
- lifting the chin
- exploring the airway for foreign bodies, including dentures, and removing them
- The Heimlich maneuver should be performed if there is reason to suspect that a foreign body is lodged in the oropharynx

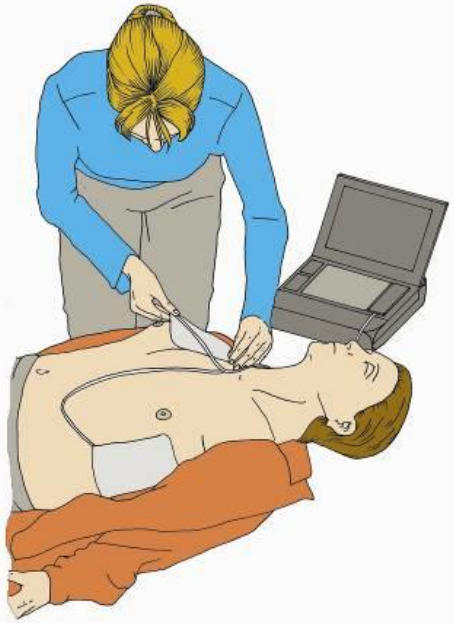
Breathing



- For single responders to victims from infants to adults, and for two-rescuer response to adults, a compression/ventilation ratio of 30 : 2

- For two-rescuer CPR in infants and children, compression/ventilation ratio of 15 : 2

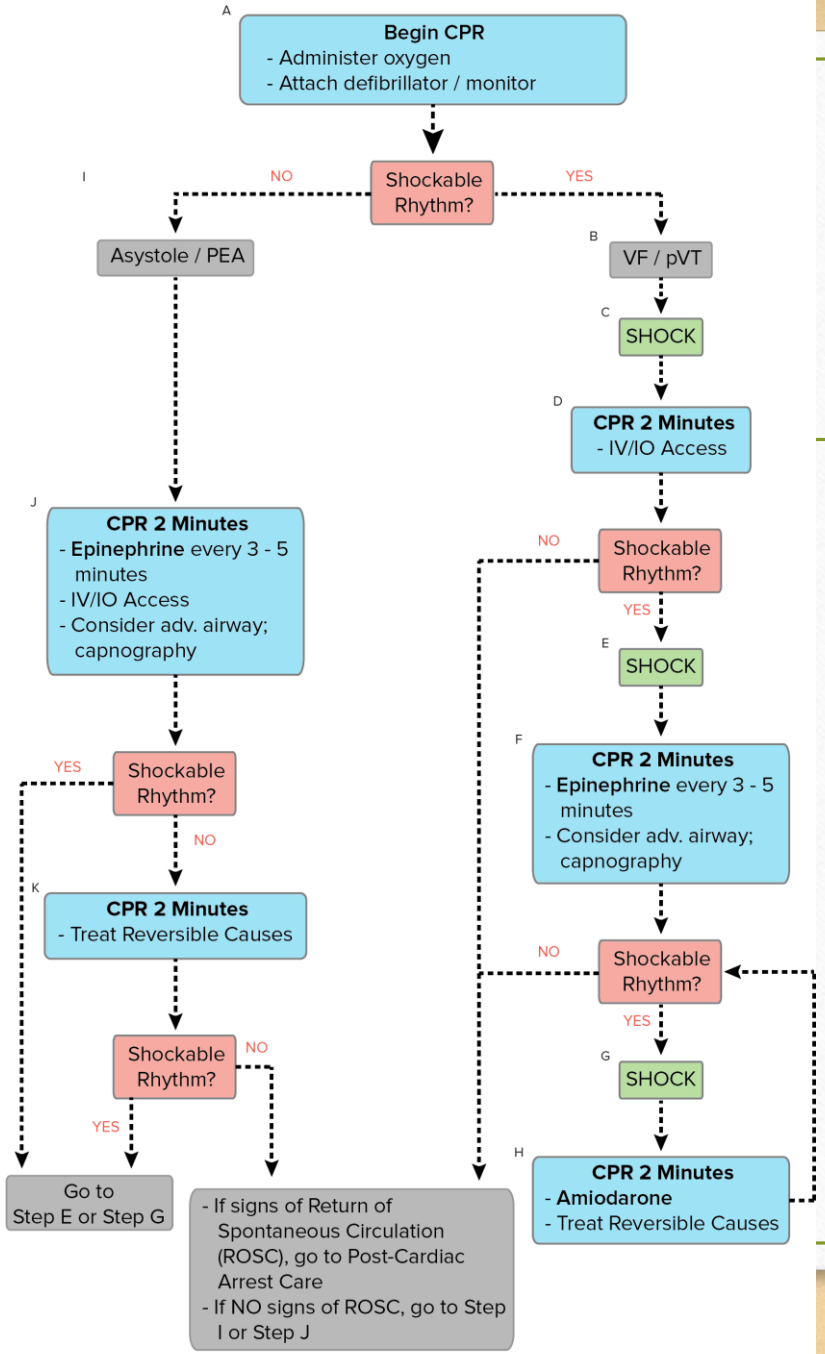
Early defibrilation



Advanced Cardiac Life Support(ACLS)

- The general goals of ACLS are to restore cardiac rhythm to hemodynamic effectiveness, to optimize ventilation, and to maintain and support the restored circulation
- After the initial attempt to restore a hemodynamically effective rhythm, the patient is intubated and oxygenated, if needed, and the heart is paced if bradyarrhythmia or asystole occurs. An intravenous (IV) line is established to deliver medications

Adult Cardiac Arrest Algorithm



CPR Quality
- Push hard (> 2 in.) and fast (≥ 100 - 120 per min.)
- Allow complete chest recoil, and reduce interruptions between compressions
- Avoid over-ventilation
- If no advanced airway, 30:2 compression - to - ventilation ratio
- Quantitative Waveform Capnography
  - If $PETCO_2$ < 10 mm Hg, try to improve CPR Quality
- Intra-Arterial Pressure
  - If diastolic pressure < 20 mm Hg, try to improve CPR quality

Advanced Airway
- Endotracheal intubation or Supraglottic Advanced Airway
- Waveform capnography to confirm / monitor ET Tube placement
- 10 breaths/min. with continuous chest compressions

Shock Energy
- **Biphasic**: Use manufacturer recommendation (i.e. 100 - 200J initial dose); If unknown, use max accessible. Second / subsequent doses should be equivalent, higher doses may be considered.
- **Monophasic**: 360J

CPR Drug Therapy
- **Amiodarone IV/IO Dose:**
  First dose: 300 mg bolus
  Second dose: 150 mg
- **Epinephrine IV/IO Dose:**
  1 mg every 3 - 5 mins.

ROSC
- Blood pressure (BP) and pulse
- Spontaneous arterial pressure waves with intra-arterial monitoring
- Sudden continuous increase in $PETCO_2$ (commonly ≥ 40 mm Hg)

Reversible Causes
- **H**ypoxia
- **H**ypovolemia
- **H**ydrogen Ions (Acidosis)
- **H**yper / **H**ypokalemia
- **H**ypothermia
- **T**ension Pneumothorax
- **T**amponade
- **T**oxins
- **T**hrombosis (Pulmonary Embolus)
- **T**hrombosis (Acute Coronary Syndrome)

Is there value in pre-participation screening to prevent sudden cardiac arrest?

- Unfortunately, the first premise has major limitations, and the second is unproven.
- For "prediction" to be effective, screening needs to be sensitive and allow the detection of conditions most commonly responsible for sudden death.
- False-positive findings can lead to unnecessary additional testing and the unfortunate restriction of athletes who are actually at low risk.
- For the promise of "prevention" to be realized, it is necessary to show that sports restriction truly reduces the risk of death.

Is there value in pre-participation screening to prevent sudden cardiac arrest?

- Many guidelines from different organizations have made recommendations regarding the process of preparticipation screening.
- Medical and family history and a corresponding physical examination are recommended as part of all routine screening programs
- the use of electrocardiography (ECG) in the screening process remains controversial

What is the best approach to screening athletes in a primary care setting?

- History and physical examination

it is important to routinely ask athletes the following questions

- Have you ever felt severely dizzy or faint, unexpectedly short of breath, or had chest pain during or immediately after exercise?
- Do you have any first-degree relatives who died suddenly or had severe cardiac disease under age 60?
- physical examination should emphasize measurement and comparison of blood pressure in both arms, auscultation for heart murmurs, and examination for physical features of **Marfan syndrome**

12-lead ECG

- 2%–4% rate of abnormalities requiring further testing,
- rate of clinically significant abnormalities in about 0.3% of athletes
- routine use of ECG as a screening strategy for athletes carries low specificity and requires the use of strict interpretation criteria
- many of the disorders that can cause sudden cardiac arrest in athletes, including primary electrical disease, anomalous origin of the coronary arteries, and premature coronary artery disease, will not be detected on standard ECG.

12-lead ECG

- Among younger patients, the ECG is less sensitive in detecting cardiomyopathies
- typical abnormalities in only 25% of adolescents with arrhythmogenic right ventricular cardiomyopathy
- 50%–75% of asymptomatic young patients with hypertrophic cardiomyopathy

Role of screening techniques in SCD

ECG: European Society of Cardiology (ESC) recommendations

Physiological adaptation

Do not require further revaluation

- Sinus bradicardia *(>30bpm) -51% to 68%-*
- Sinus arrhythmia *-20% to 23%-*
- Ectopic atrial rhythm *-1%-*
- Junctional escape rhythm *-0.4%-*
- 1st degree AV block *(PR interval > 200 ms) -7%-*
- Mobitz Type I *(Wenckebach)* 2nd degree AV block – *0.1%-*
- Incomplete RBBB *-24% to 27%-*
- Isolated QRS voltage criteria for LVH *-23% to 36%-*
- Early repolarization *(ST elevation, J-point elevation, J-waves or terminal QRS slurring) -37% to 72%-*
- Convex ('domed') ST segment elevation combined with T-wave inversion in leads V1–V4 in black/African athletes

Abnormal ECG findings

Require further evaluation

- T-wave inversión *(>1 mm V2-V6, II and aVF, I and aVL)*
- ST segment depression *(>0,5 mm)*
- Pathologic Q waves *(>3 mm depth/>40 ms duration)*
- Complete LBBB
- Intraventricular conduction delay *(≥140 ms)*
- Left axis deviation *(-30º to -90º)*
- Left atrial enlargement *(P>120 ms in I or II; negative portion of P≥1mm and ≥40 ms in V1)*
- Right ventricular hypertrophy *(R-V1+S-V5>10,5 mm AND right axis deviation)*
- Ventricular pre-excitation
- Long QT interval *(QTc≥470 in male; ≥480 ms in female)*
- Short QT interval *(QTc≤320 ms)*
- Brugada-like ECG pattern
- Profound sinus bradicardia *(<30bpm or pauses≥3 s.)*
- Atrial tachyarrhythmias
- Premature ventricular contractions *(≥2 PVCs/10s)*
- Ventricular arrhythmias *(couplets, triplets and NSVT)*

a sudden cardiac arrest incidence of 6/100 000 per year

- 6 out of 8 sudden cardiac deaths occurred in athletes who had a negative screen on history, physical examination and ECG (as well as on echocardiography) and were deemed “healthy.”

What can optimize survival following sudden cardiac arrest in athletes?

- Adequately trained personnel
- education of trainers and bystanders
- access to AEDs at a sports venue