

# ΕΞΕΙΔΙΚΕΥΜΕΝΗ ΥΠΟΣΤΗΡΙΞΗ ΖΩΗΣ

## Καρδιολογική υποστήριξη



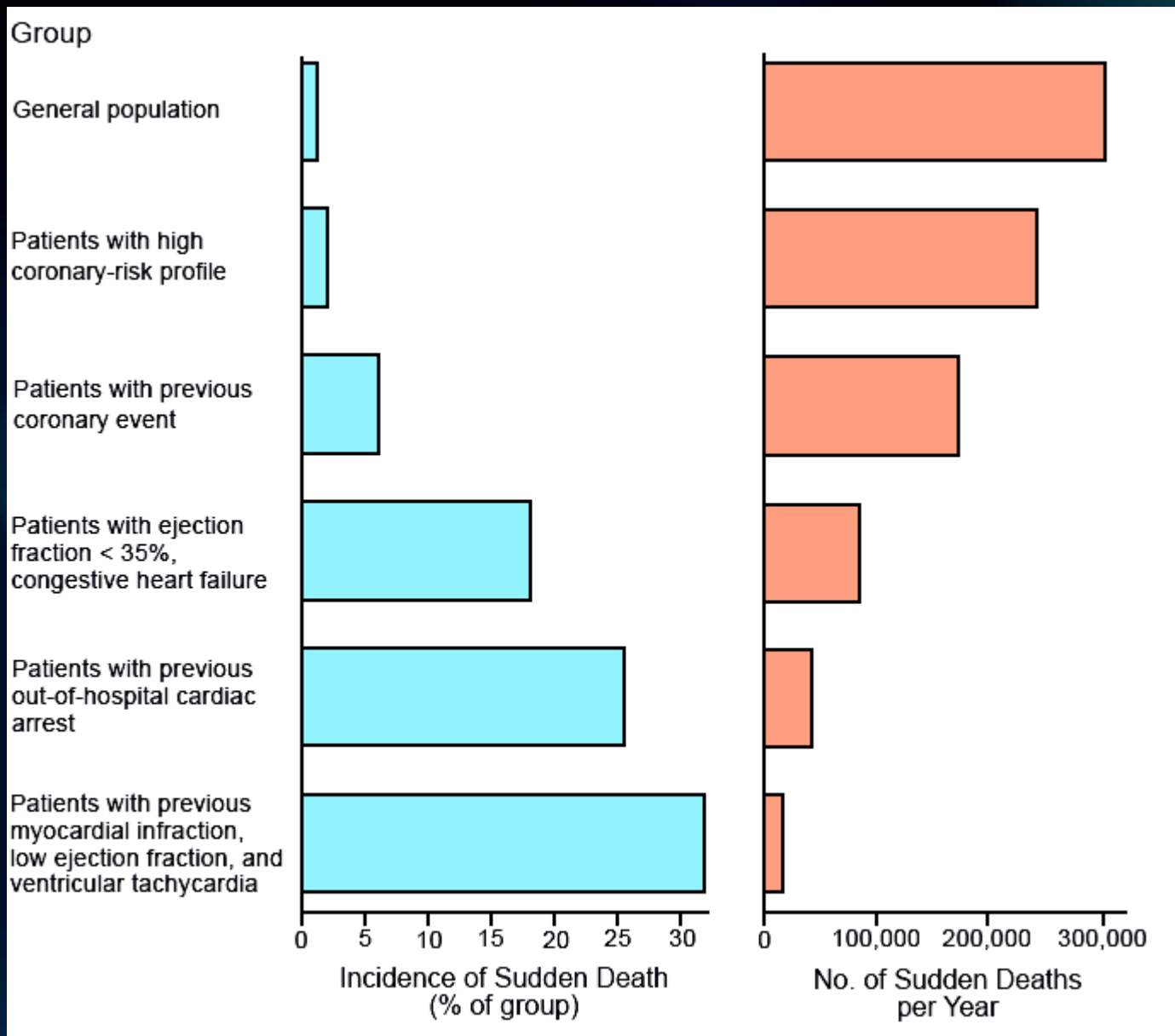
Αργύρης Σ. Νταλιάνης  
Καρδιολόγος



Γ' Καρδιολογική Κλινική Πανεπιστημίου Αθηνών



# Incidence of sudden death





# Causes of Cardiac Arrest

1. Ventricular fibrillation (VF)
2. Pulseless ventricular tachycardia (VT)
- 3.
4. Pulseless electric activity (PEA)
5. Asystole



# Cardiac causes of cardiac arrest

Ischaemic cardiac disease (coronary artery disease)

Ischaemic cardiomyopathy

Dilated cardiomyopathy

Hypertrophic cardiomyopathy

Non-atherosclerotic disease of coronary arteries

Valvular heart disease

Arrhythmogenic right ventricular cardiomyopathy

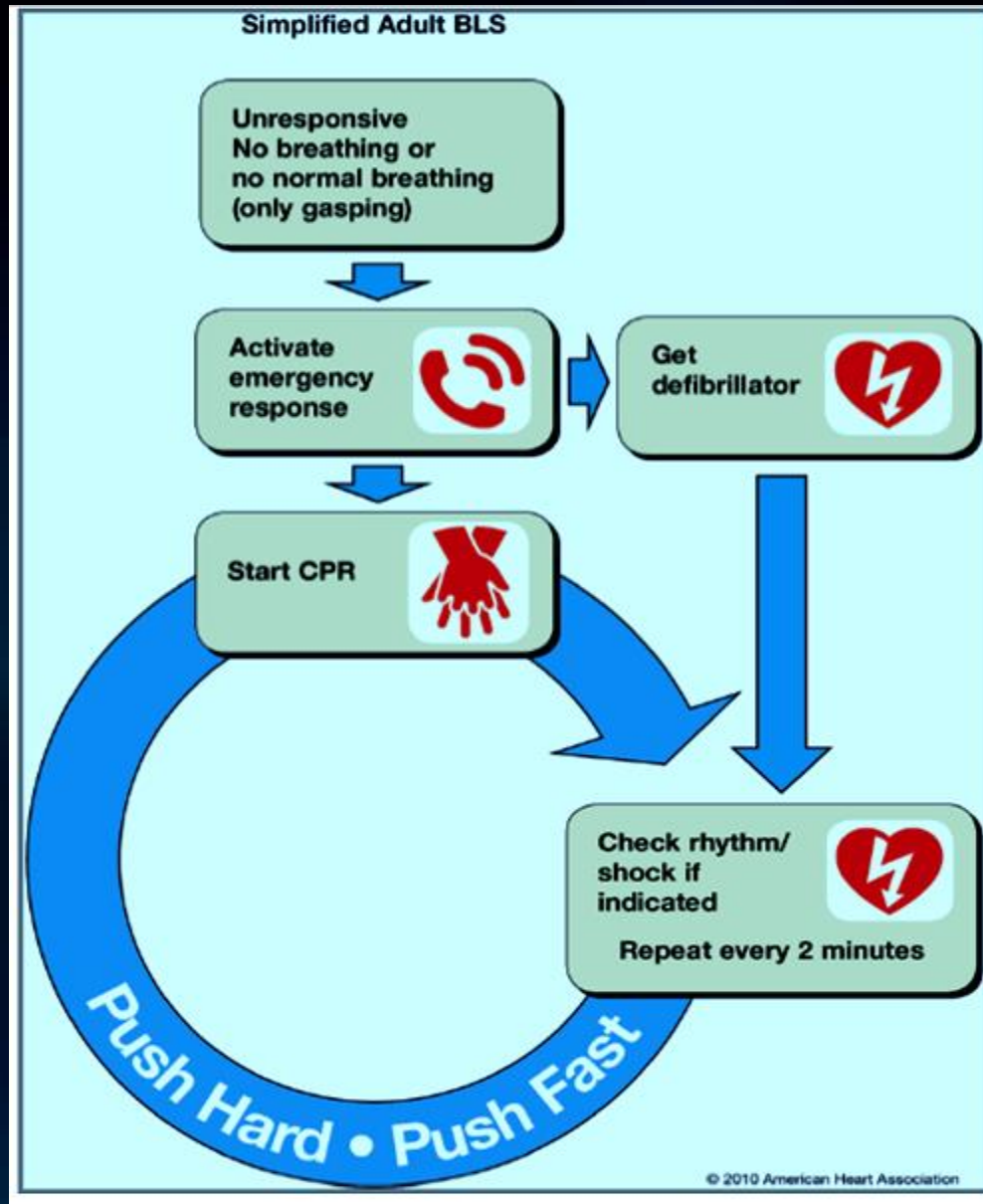
Infiltrative and inflammatory myocardial disease

Congenital heart disease

Primary cardiac electrical abnormalities

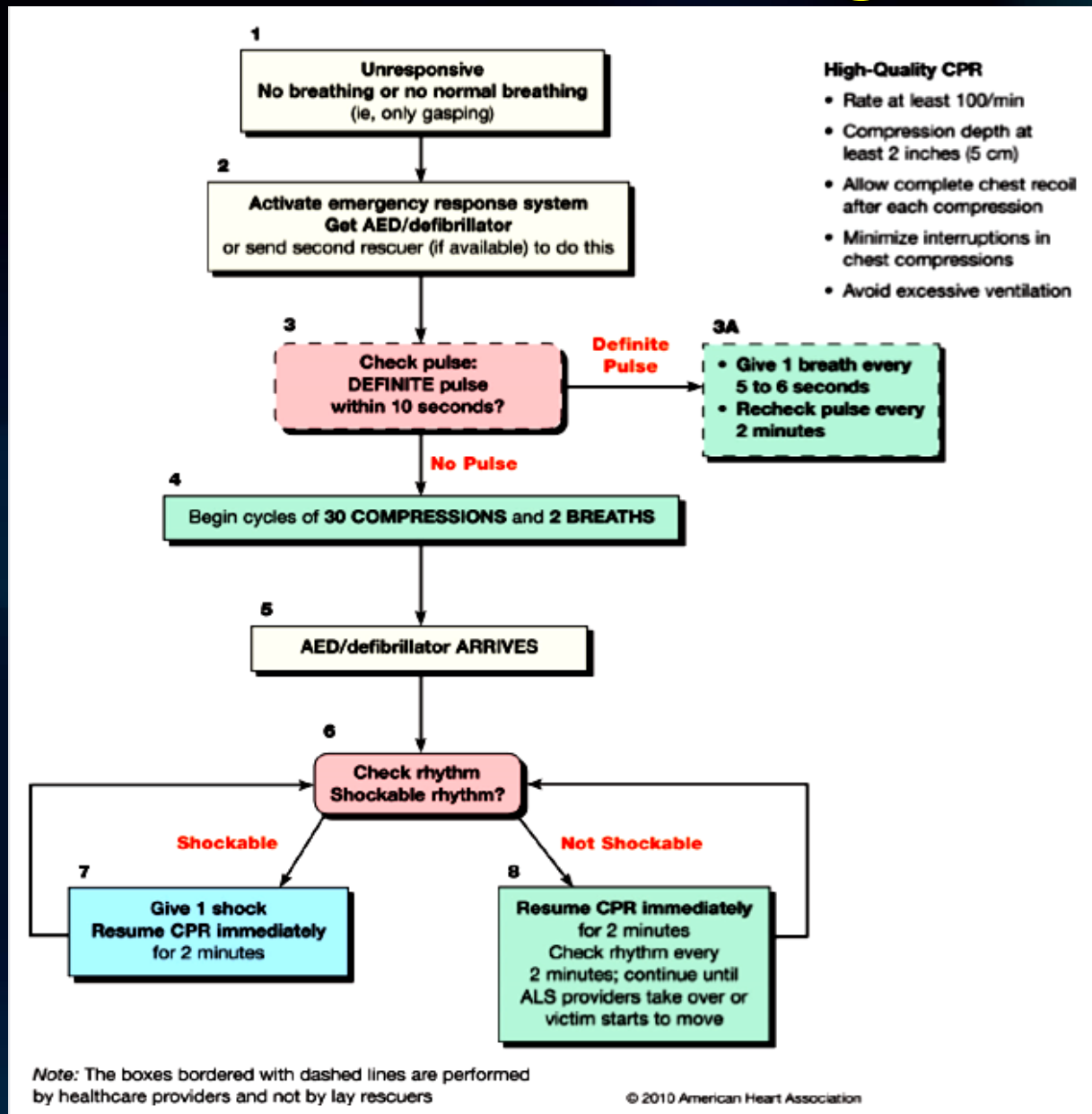


# Simplified Basic Life Support Algorithm



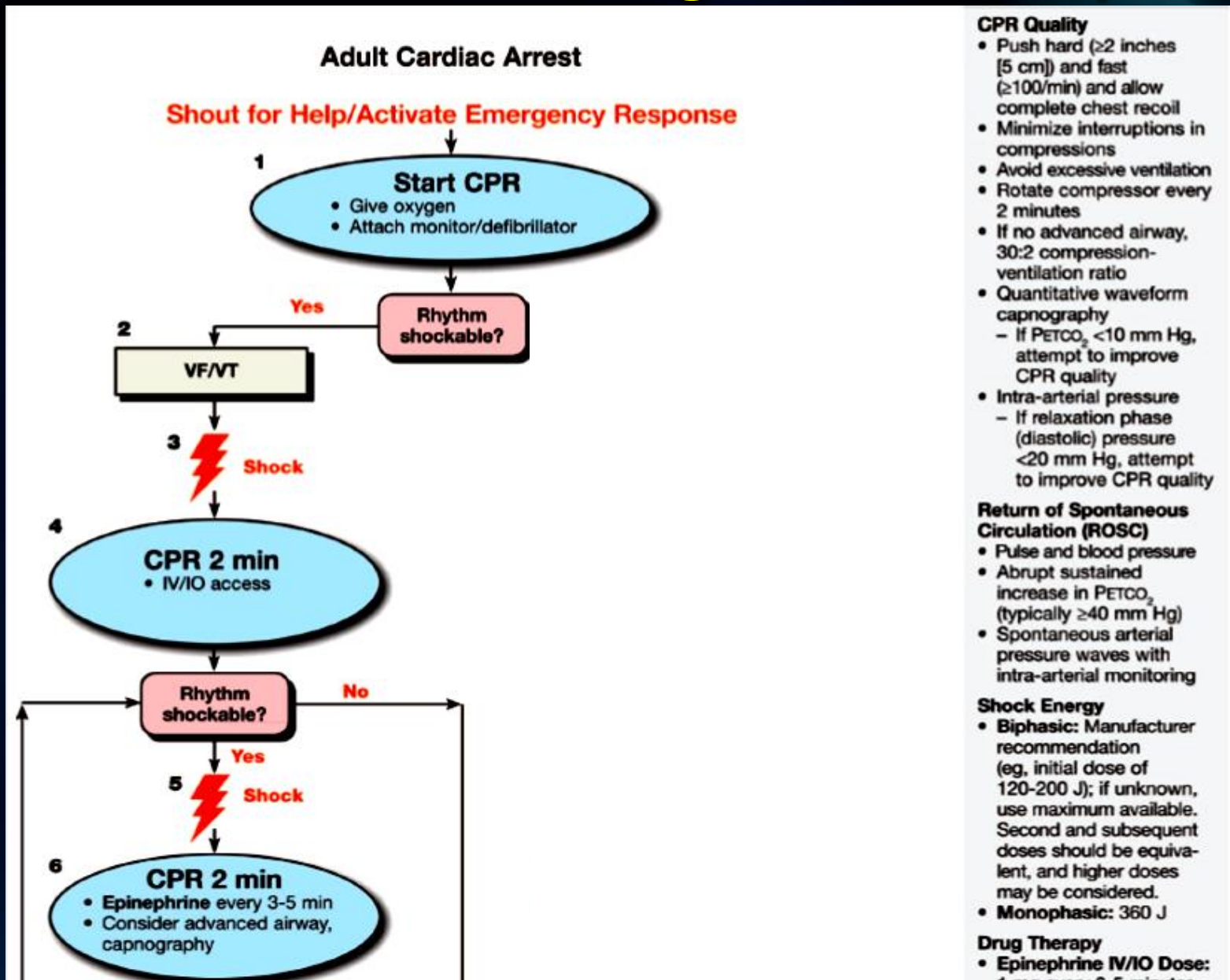


# Health Care Provider Algorithm



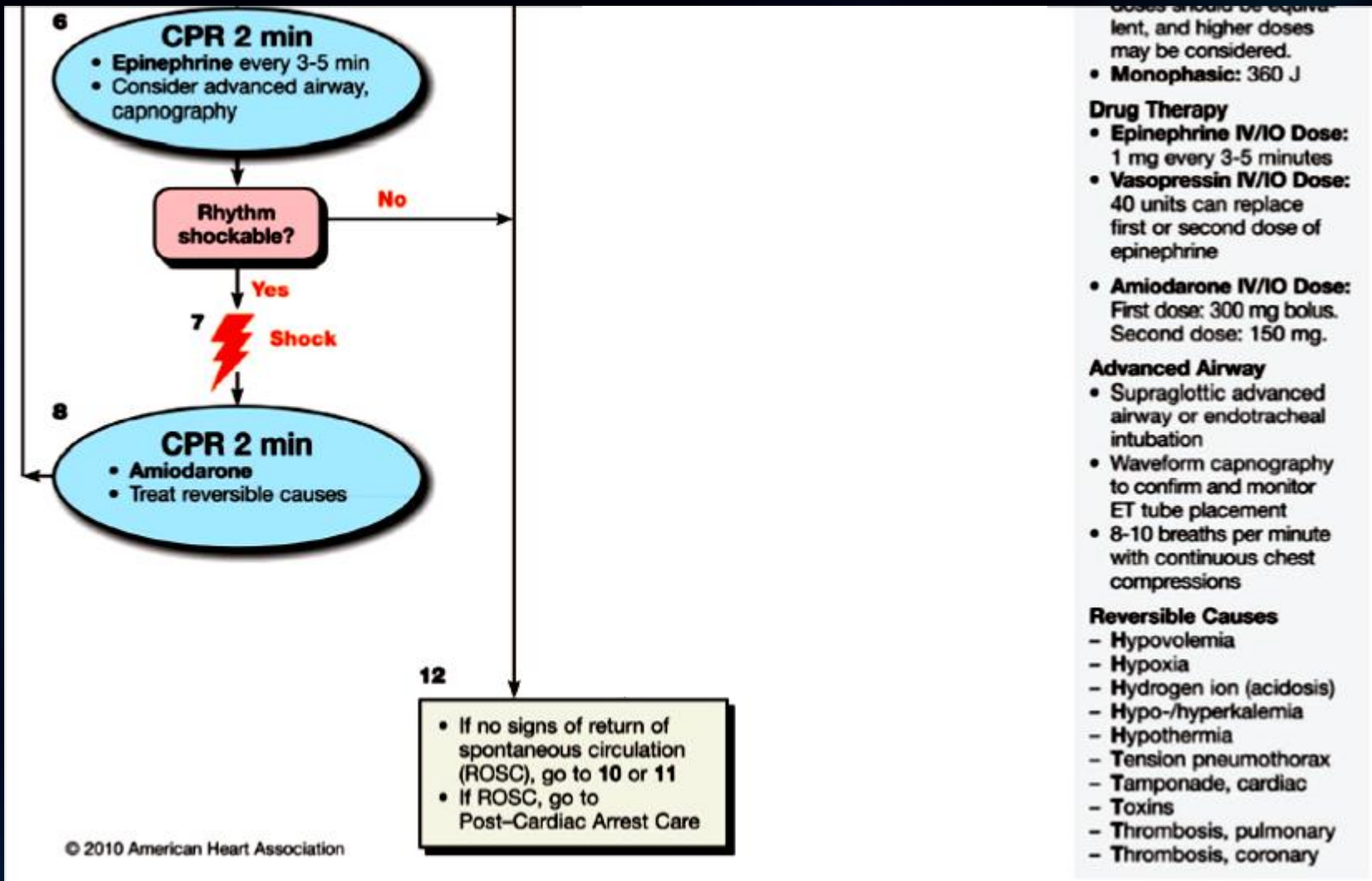


# Cardiac Arrest Algorithm – VF/VT





# Cardiac Arrest Algorithm – VF/VT (con't)



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

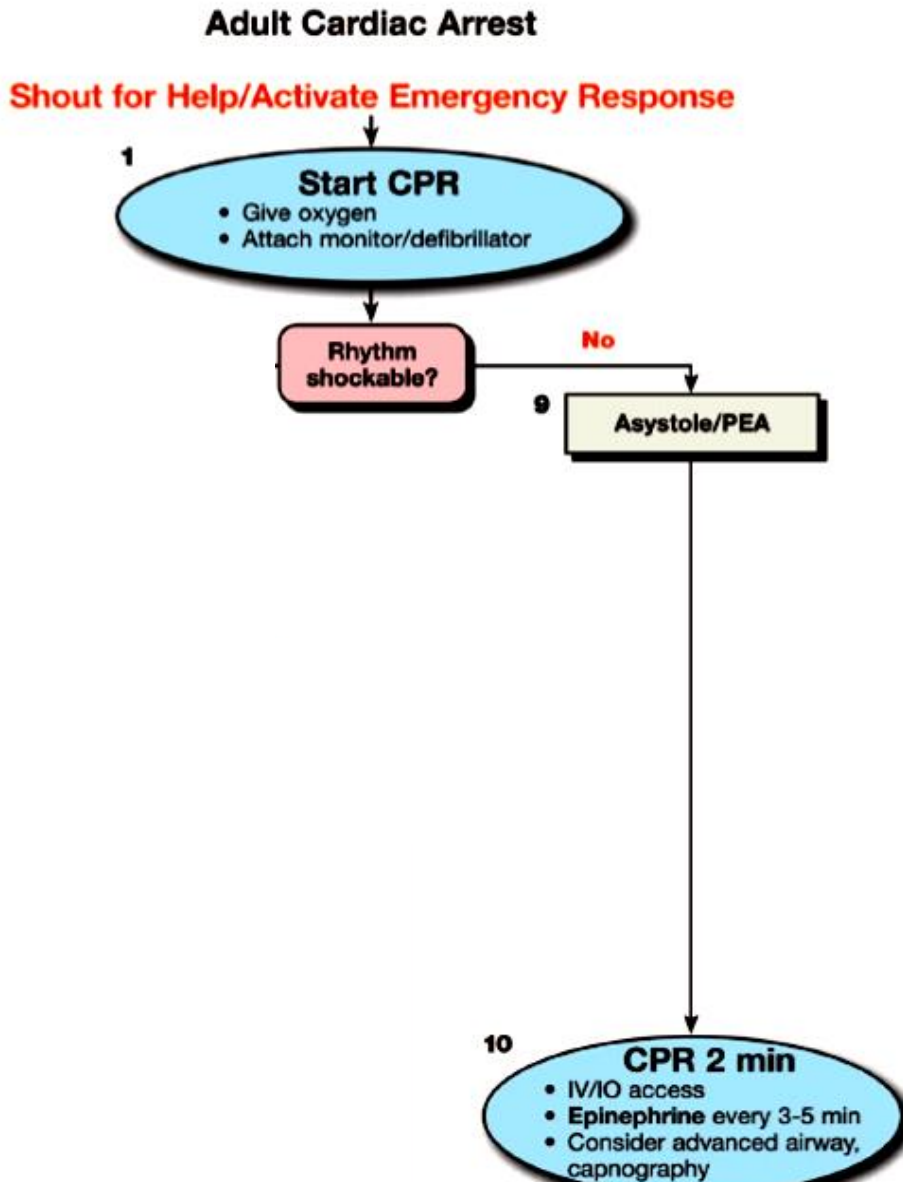
### Reversible Causes

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





# Cardiac Arrest Algorithm - Asystole



## CPR Quality

- Push hard ( $\geq 2$  inches [5 cm]) and fast ( $\geq 100$ /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  $PETCO_2 < 10$  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  $PETCO_2$  (typically  $\geq 40$  mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

## Shock Energy

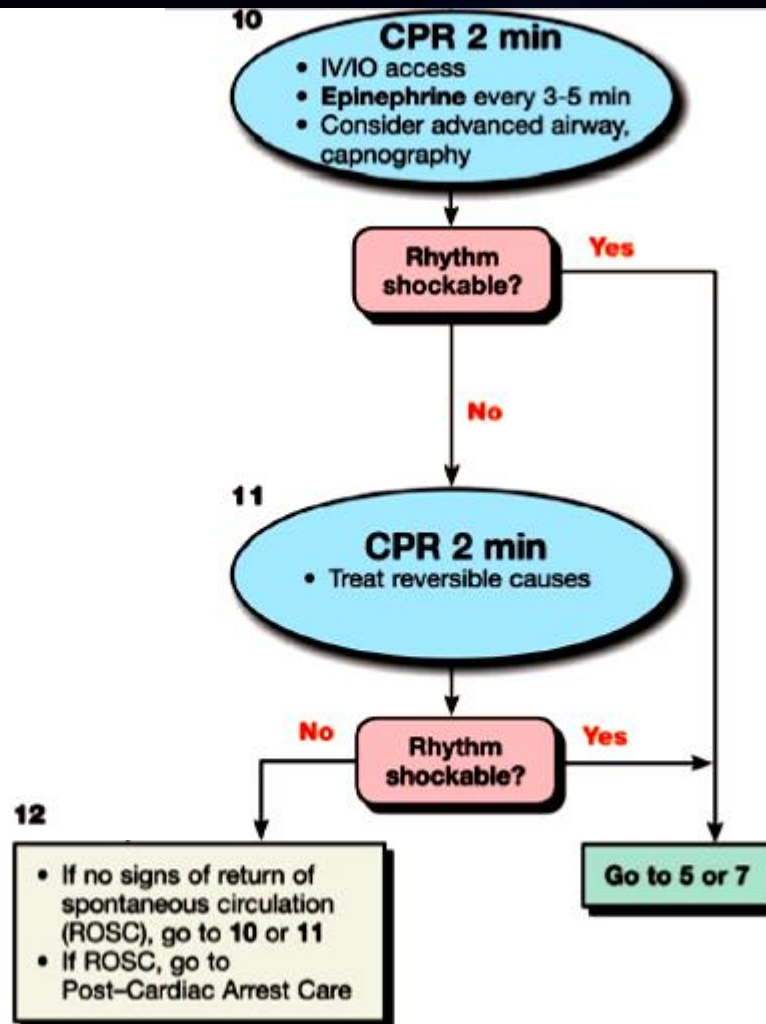
- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

## Drug Therapy

- Epinephrine IV/IO Dose: 1 mg every 3-5 minutes



# Cardiac Arrest Algorithm - Asystole (cont'ed)



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
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#### Advanced Airway

- Supraglottic advanced airway or endotracheal intubation
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#### Reversible Causes

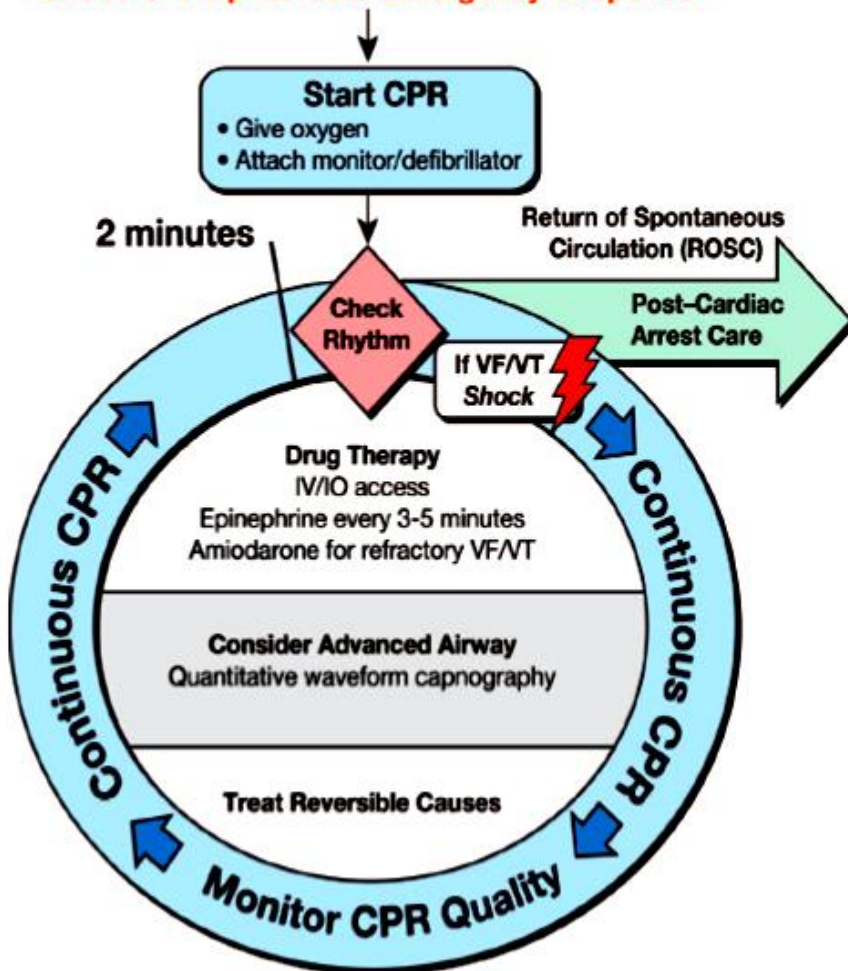
- Hypovolemia
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- Hypothermia
- Tension pneumothorax
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- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary



# Cardiac Arrest Circular Algorithm

## Adult Cardiac Arrest

Shout for Help/Activate Emergency Response



### CPR Quality

- Push hard ( $\geq 2$  inches [5 cm]) and fast ( $\geq 100$ /min) and allow complete chest recoil
- Minimize interruptions in compressions
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# Prognosis of CPR – CPR technique

Early CPR and rapid defibrillation can significantly increase the chance for survival to hospital discharge (THE ONLY PROVED LIFE SAVING TECHNIQUES!!)

In the absence of an advanced airway, a synchronized compression–ventilation ratio of 30:2 is recommended at a compression rate of at least 100 per minute.

After placement of a supraglottic airway or an endotracheal tube, the provider performing chest compressions should deliver at least 100 compressions per minute continuously without pauses for ventilation. The provider delivering ventilations should give 1 breath every 6 to 8 seconds (8 to 10 breaths per minute) and should be particularly careful to avoid delivering an excessive number of ventilations

The provider performing chest compressions should switch every 2 minutes

Vascular access, drug delivery, and advanced airway placement should not cause significant interruptions in chest compression or delay defibrillation

Neumar R et al. CIRCULATION 2010, CPR AHA guidelines



# Monitoring CPR

1. **End-tidal CO<sub>2</sub>** is the concentration of carbon dioxide in exhaled air at the end of expiration. Persistently low PETCO<sub>2</sub> values (<10 mm Hg) during CPR in intubated patients suggest that resuscitation is unlikely
2. **Coronary perfusion pressure**: aortic relaxation [“diastolic”] pressure minus right atrial relaxation [“diastolic”] pressure)  
In one human study resuscitation did not occur unless a CPP  $\geq$ 15 mm Hg was achieved during CPR
3. **Central Venous Oxygen Saturation**. In one clinical study the failure to achieve ScvO<sub>2</sub> of 30% during CPR was associated with failure to achieve resuscitation
4. Echocardiography: absence of cardiac motion on sonography during resuscitation of patients in cardiac arrest was highly predictive of inability to achieve resuscitation



# Defibrillation strategies

Biphasic defibrillator: initial energy dose of 120 to 200 J for terminating VF (Class I, LOE B)

Second and subsequent energy levels should be at least equivalent, and higher energy levels may be considered if available (Class IIb, LOE B)

Monophasic defibrillator: initial shock of 360 J and use that dose for all subsequent shocks. If VF is terminated by a shock but then recurs later in the arrest, deliver subsequent shocks at the previously successful energy level

**Amiodarone** is the first-line antiarrhythmic agent given during cardiac arrest → improves the rate of Resuscitation & hospital admission in adults with refractory VF/pulseless VT. Amiodarone may be considered when VF/VT is unresponsive to CPR, defibrillation, and vasopressor therapy (Class IIb, LOE A)

If amiodarone is unavailable, **lidocaine** may be considered, (Class IIb, LOE B)

**Magnesium sulfate** → only for torsades de pointes (long QT ) (Class IIb, LOE B).



# Treatable Causes of Cardiac Arrest

Hypoxia → **VENTILATION**

Hypovolemia → **FLUID/BLOOD  
ADMINISTRATION**

Hydrogen ion (acidosis) → **NaHCO<sub>3</sub>**

Hypo-/hyperkalemia → **Correct** ↓↑**K<sup>+</sup>**

Hypothermia → **Rewarming**

Toxins → **charcoal/antidotes**

Tamponade (cardiac) → **pericardiocentesis**

Tension pneumothorax → **thoracocentesis**

Thrombosis, pulmonary → **thrombolysis**

Thrombosis, coronary → **PCI**

# Diagnosis & Management of Tachycardia





# Narrow-complex tachycardia

## SVT

QRS <0.12 seconds

In order of frequency:

- Sinus tachycardia
- Atrial fibrillation
- Atrial flutter
- AV nodal reentry
- Accessory pathway–mediated tachycardia
- Atrial tachycardia (including automatic and reentry forms)
- Multifocal atrial tachycardia (MAT)
- Junctional tachycardia (rare in adults)



# Narrow-complex tachycardia

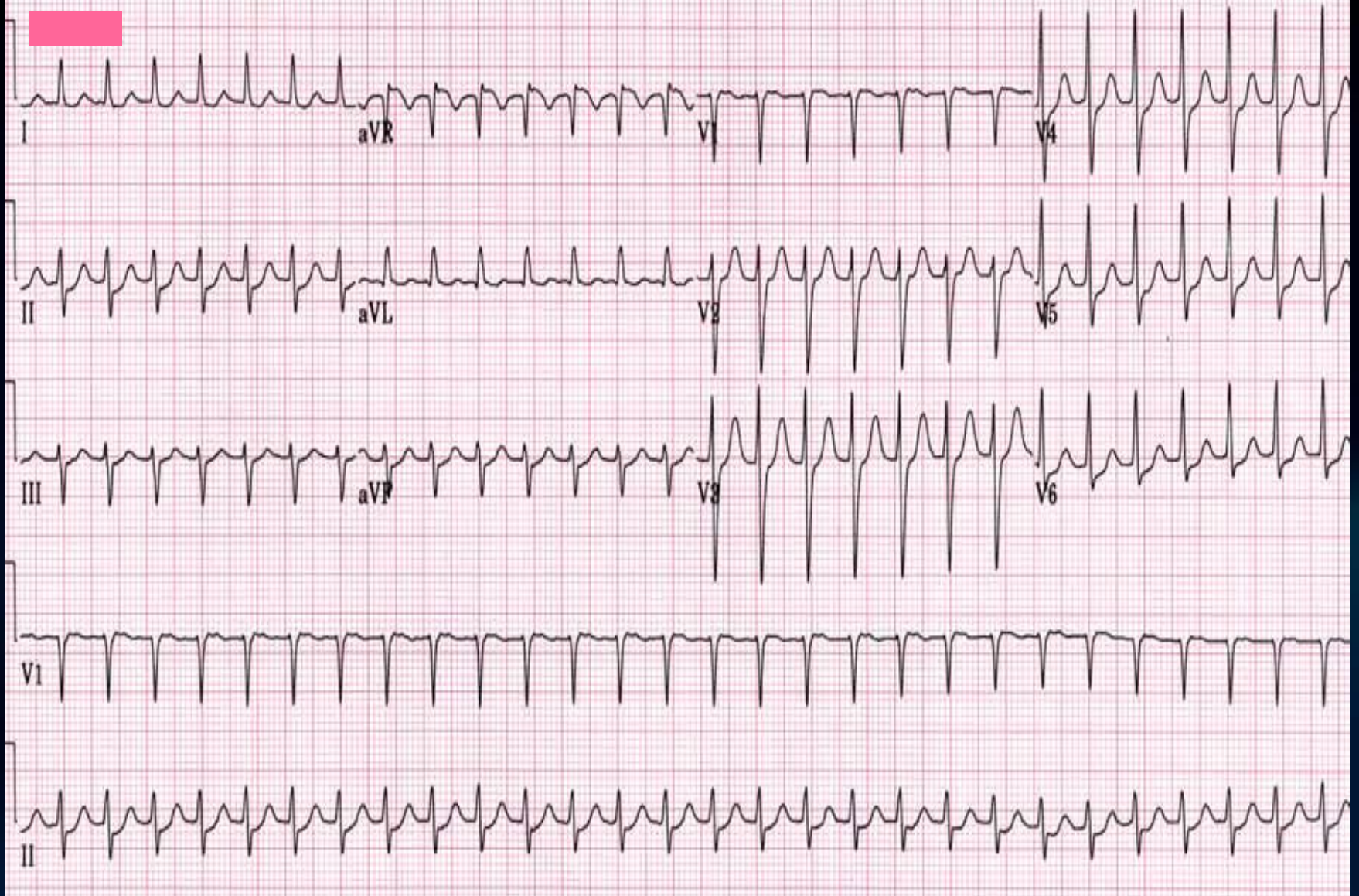
## SVT

QRS  $\geq 0.12$  seconds

- Ventricular tachycardia (VT) and ventricular fibrillation (VF)
- SVT with aberrancy
- Pre-excited tachycardias (Wolff-Parkinson-White [WPW] syndrome)
- Ventricular paced rhythms



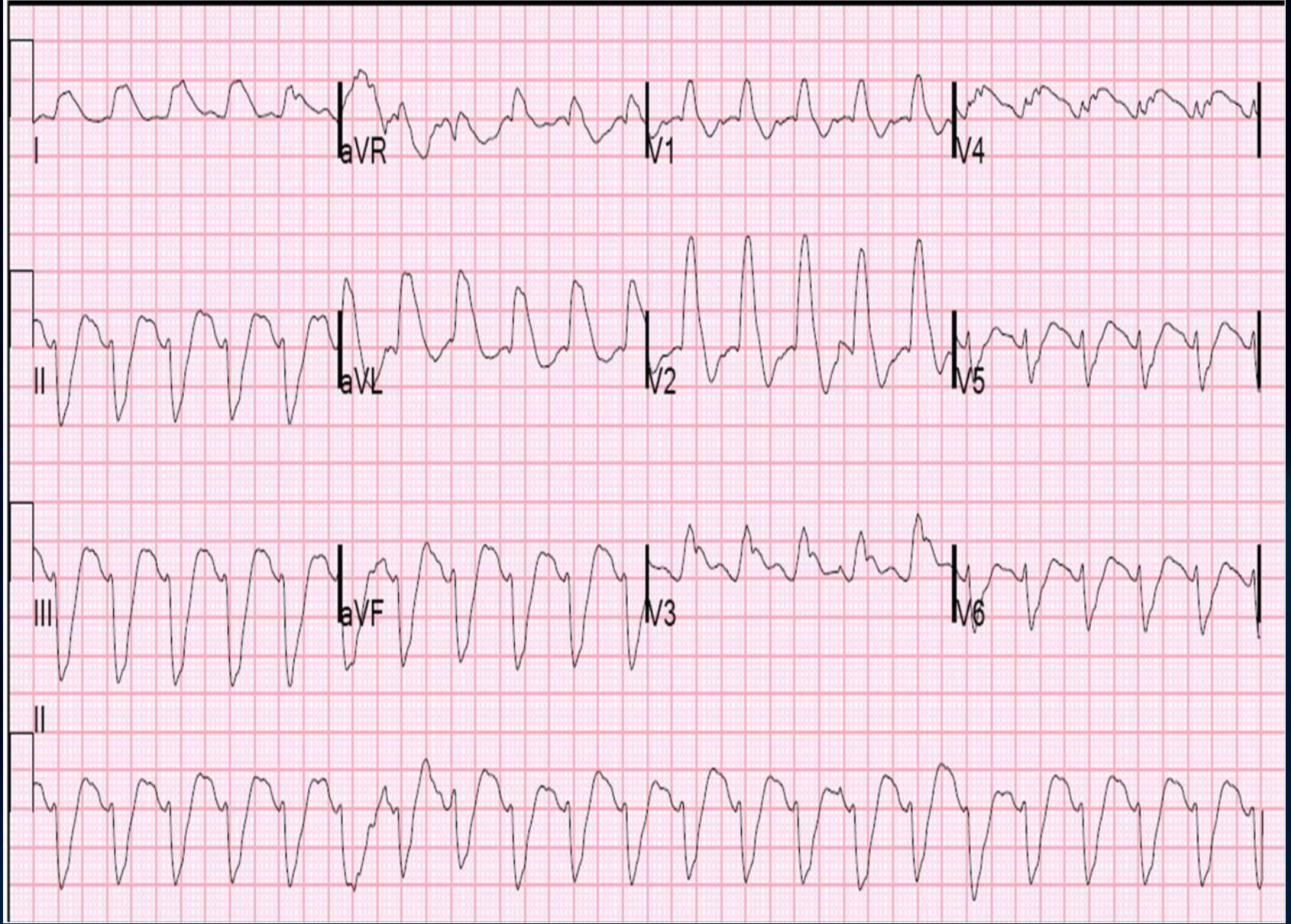
# Narrow-complex tachycardia -SVT





# Wide-complex tachycardia

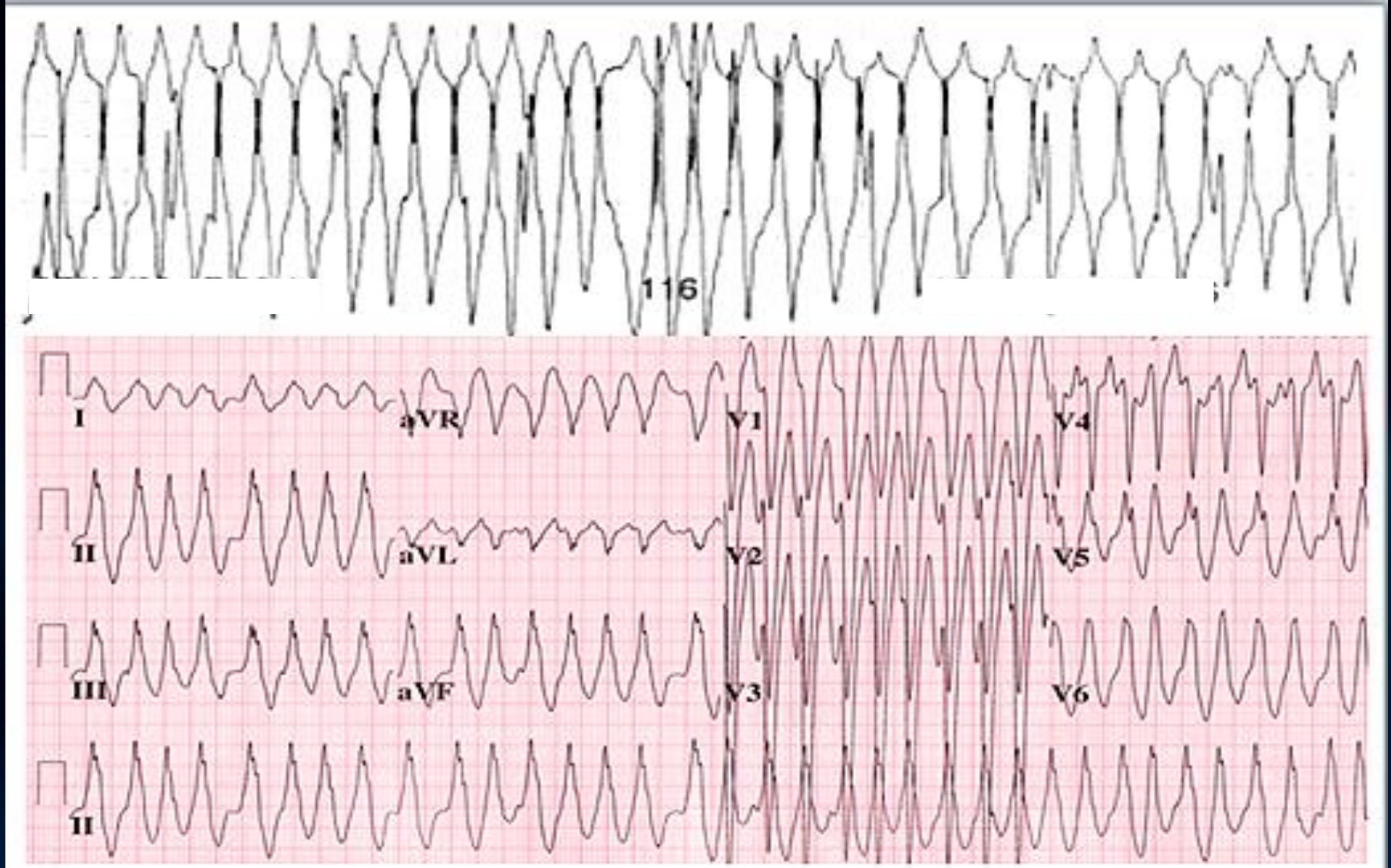
## Monomorphic VT





# Wide-complex tachycardia

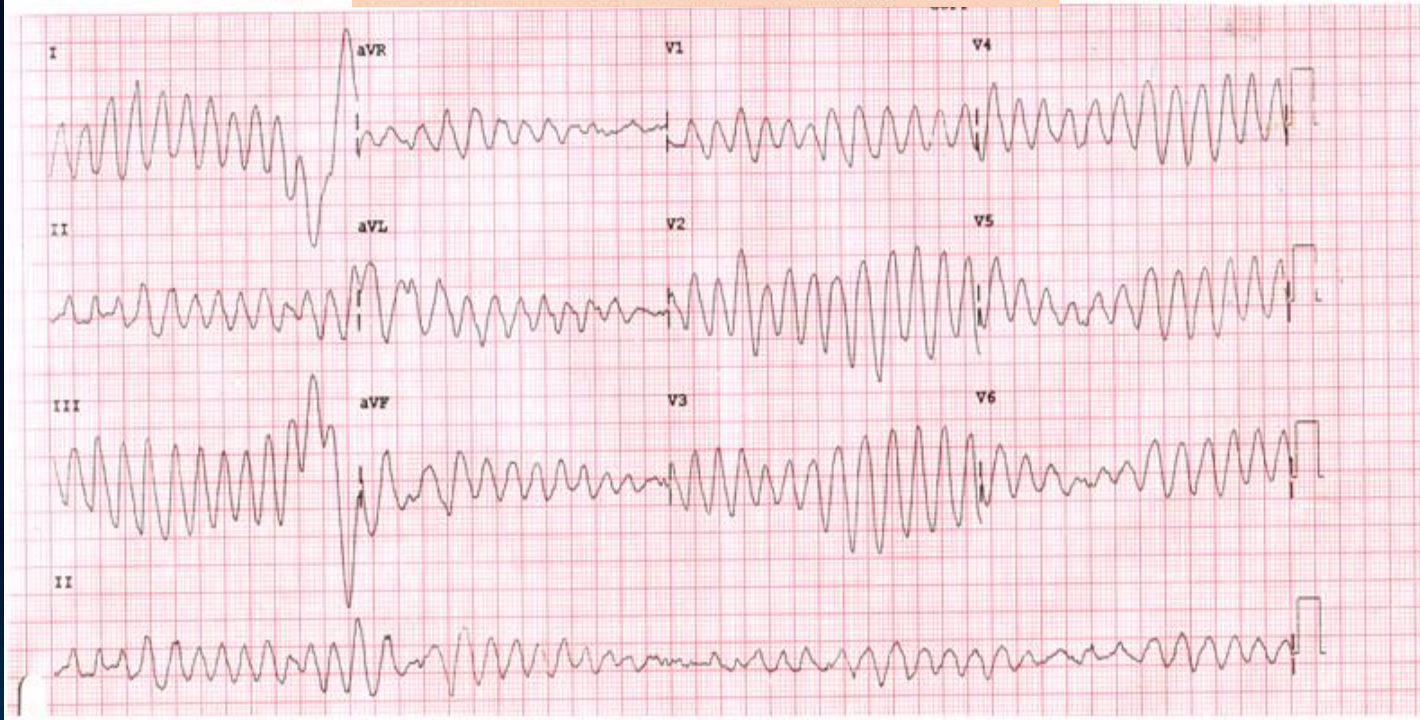
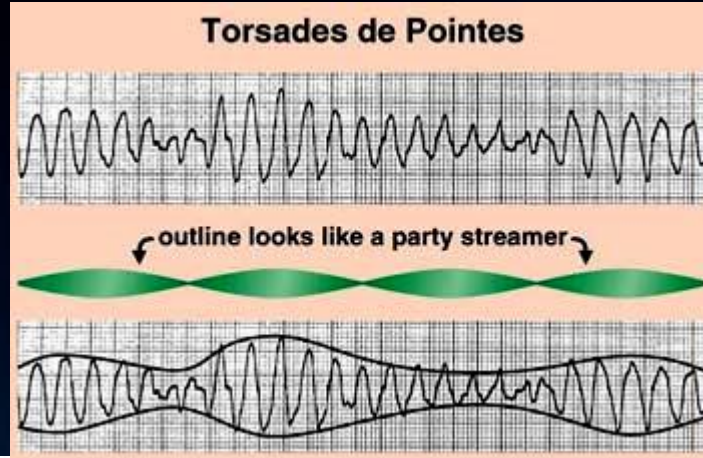
## Polymorphic VT





# Wide-complex tachycardia

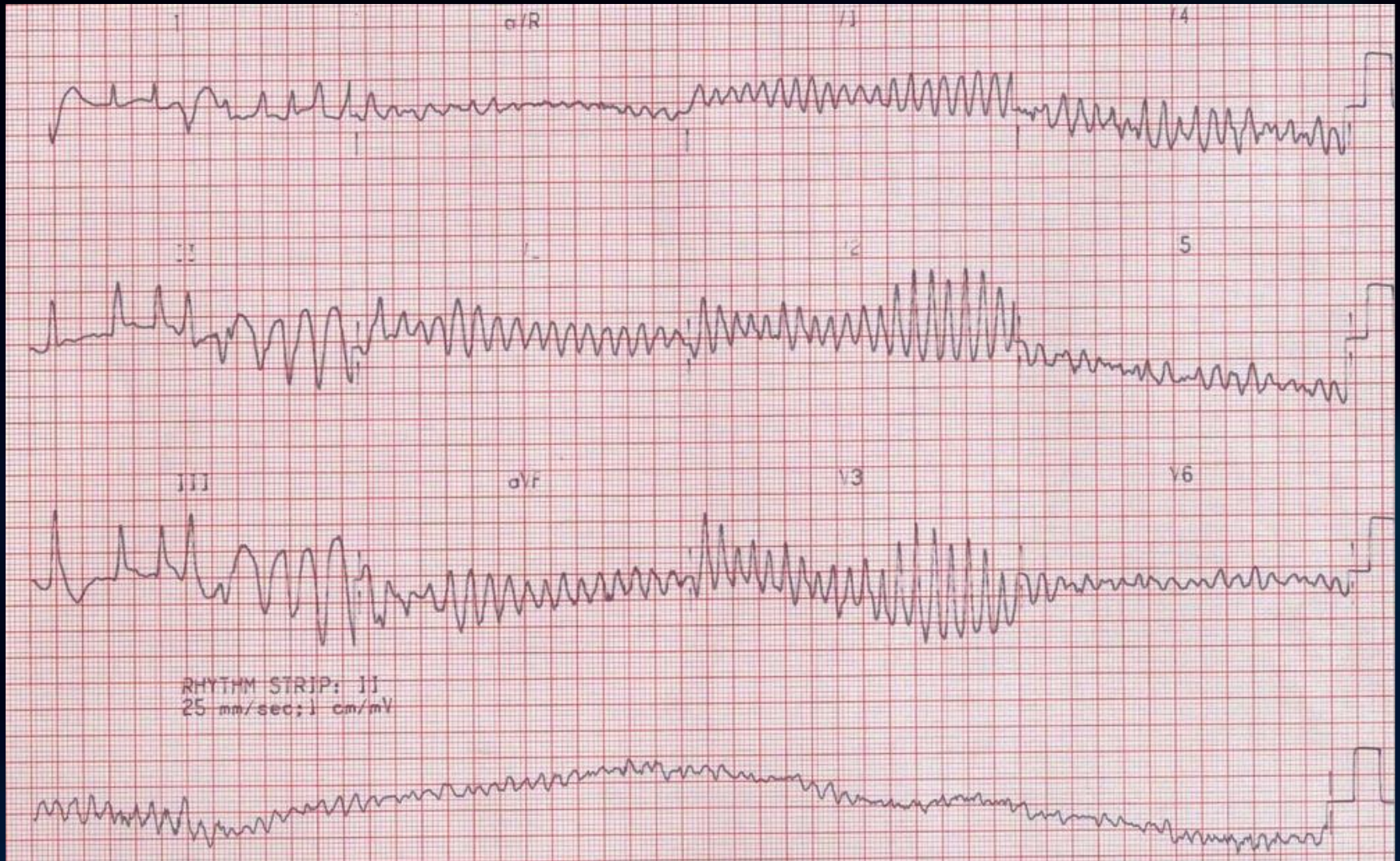
## Torsades de Pointes





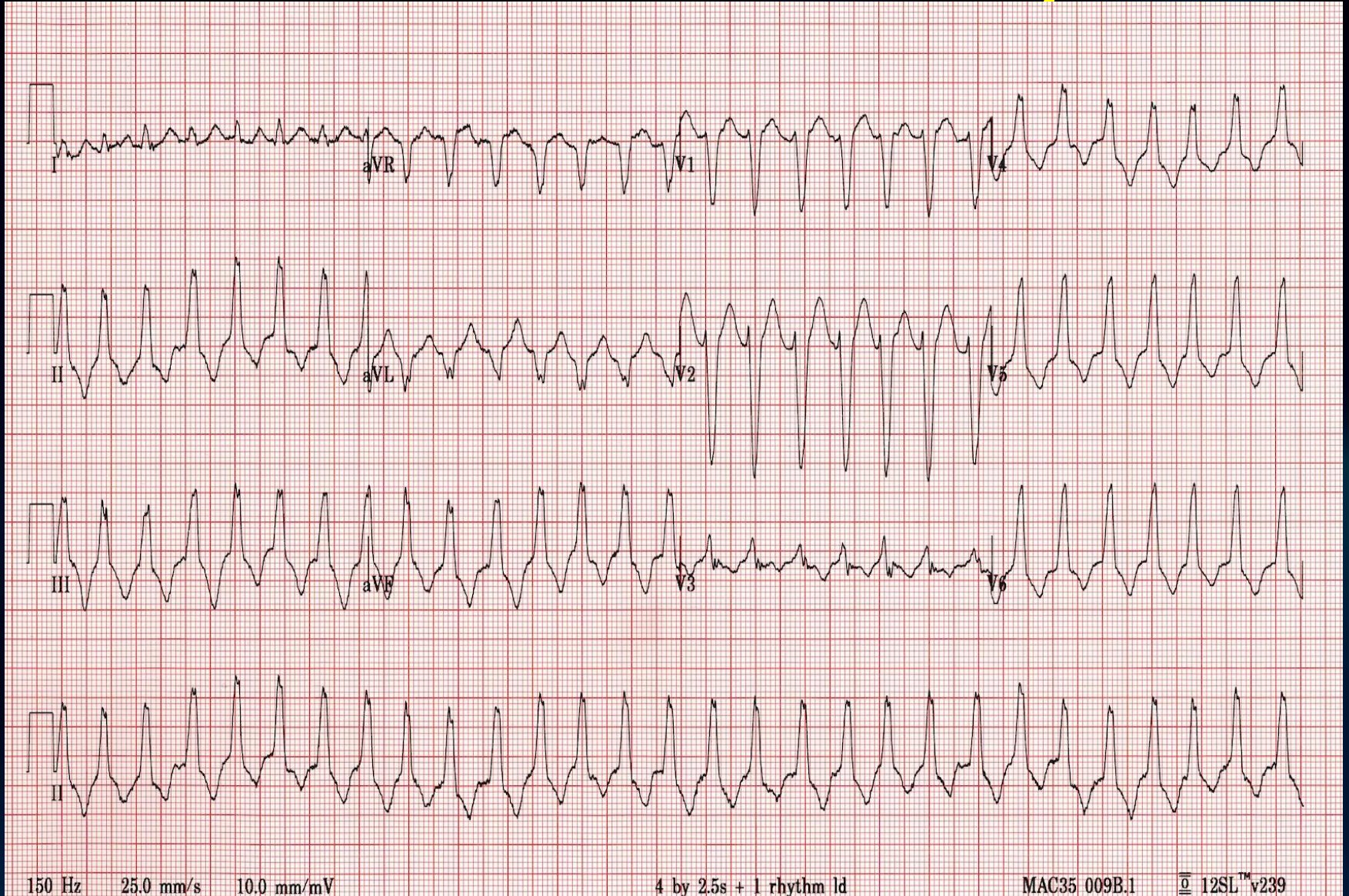
# Wide-complex tachycardia

## Ventricular fibrillation





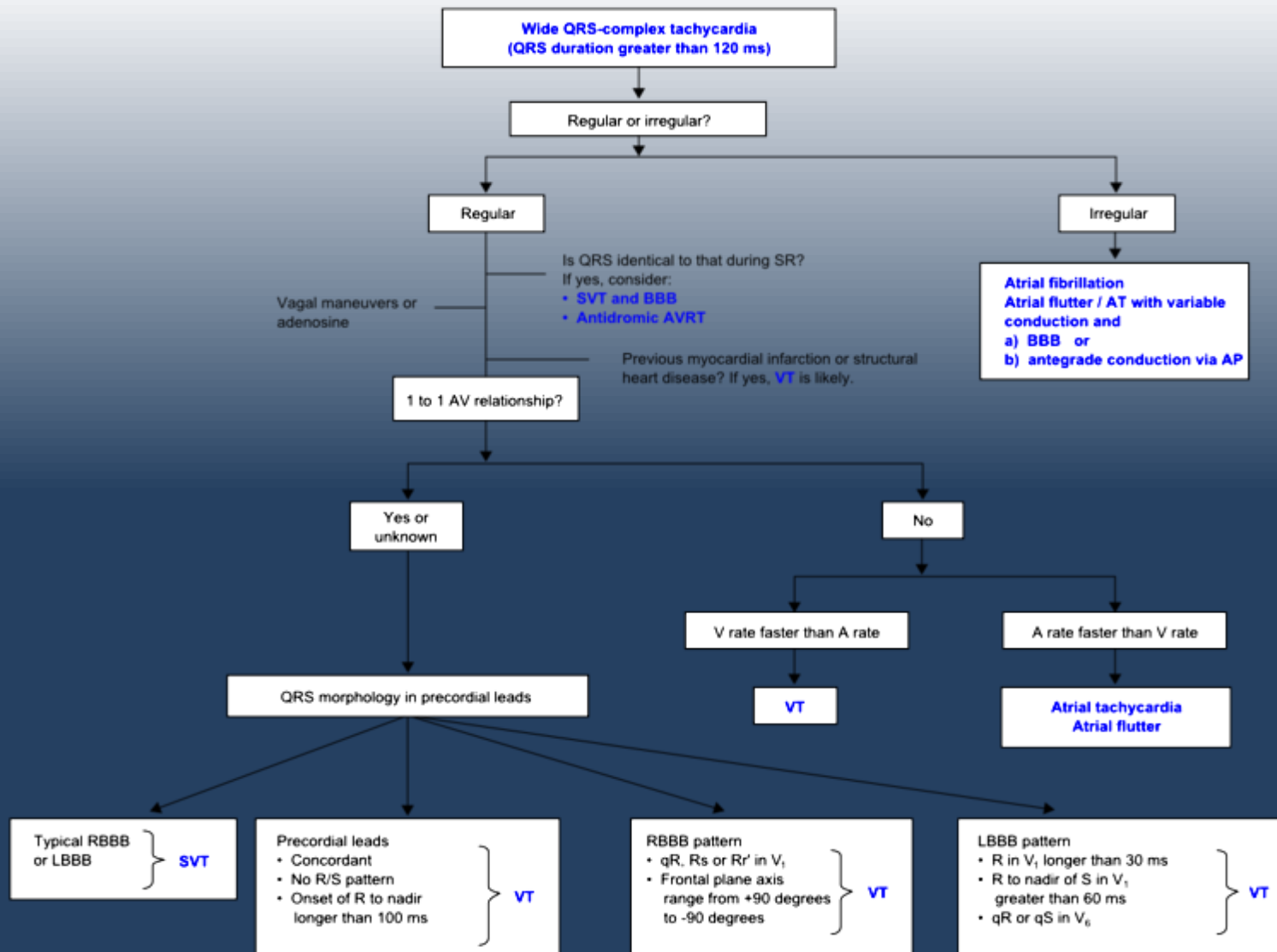
# Wide-complex tachycardia SVT with aberrancy







# Differential diagnosis SVT-VT





# IV Drugs to treat SVTs

ΦΑΡΜΑΚΟ	ΙΔΙΟΤΗΤΕΣ	ΕΝΔΕΙΞΕΙΣ	ΔΟΣΗ	ΑΕ	ΠΡΟΦΥΛΑΞΕΙΣ
<b>Adenosine</b>	Endogenous purine nucleoside; briefly depresses sinus node rate and AV node conduction; vasodilator	<ul style="list-style-type: none"> <li>Stable, narrow-complex regular tachycardias</li> <li>Unstable narrow-complex regular tachycardias while preparations are made for electrical cardioversion</li> <li>Stable, regular, monomorphic, wide complex tachycardia (therapeutic /diagnostic maneuver)</li> </ul>	6 mg IV as a rapid IV push followed by a 20 mL saline flush; repeat if required as 12 mg IV push	Hypotension, bronchospasm, Chest discomfort	Contraindicated in patients with asthma; may precipitate atrial fibrillation, which may be very rapid in patients with WPW; thus a defibrillator should be readily available; reduce dose in post-cardiac transplant patients, those taking dipyridamole or carbamazepine and when administered via a central vein
<b>Diltiazem, Verapamil</b>	Non-dihydropyridine calcium channel blockers; slow AV node conduction and increase AV node refractoriness; vasodilators/negative inotropes	<ul style="list-style-type: none"> <li>Stable, narrow-complex tachycardias if rhythm remains uncontrolled or unconverted by adenosine or vagal maneuvers or if SVT is recurrent</li> <li>Control ventricular rate in patients with AFIB or AFLUT</li> </ul>	<p><u>Diltiazem</u>: Initial dose 15 to 20 mg (0.25 mg/kg) IV over 2 minutes; additional 20 to 25 mg (0.35 mg/kg) IV in 15 minutes if needed; 5 to 15 mg/h IV maintenance infusion (titrated to AF heart rate if given for rate control)</p> <p><u>Verapamil</u>: Initial dose 2.5 to 5 mg IV given over 2 minutes; may repeat as 5 to 10 mg every 15 to 30 minutes to total dose of 20 to 30 mg</p>	Hypotension, bradycardia, precipitation of heart failure	Should only be given to patients with narrow-complex tachycardias (regular or irregular). Avoid in patients with heart failure and pre-excited AF or flutter or rhythms consistent with VT
<b>Atenolol, Esmolol, Metoprolol, Propranolol</b>	Blockers; reduce effects of circulating catecholamines; reduce heart rate, AV node conduction and blood pressure; negative inotropes	<ul style="list-style-type: none"> <li>Stable, narrow-complex tachycardias if rhythm remains uncontrolled or unconverted by adenosine or vagal maneuvers or if SVT is recurrent</li> <li>Control ventricular rate in patients with AFIB or AFLUT</li> <li>Certain forms of polymorphic VT (associated with acute ischemia, familial LQTS, catecholaminergic)</li> </ul>	<p><u>Atenolol</u> (1 specific blocker) 5 mg IV over 5 minutes; repeat 5 mg in 10 minutes if arrhythmia persists or recurs</p> <p><u>Esmolol</u> (1 specific blocker with 2- to 9-minute half-life) IV loading dose 500 mcg/kg (0.5 mg/kg) over 1 minute, followed by an infusion of 50 mcg/kg per minute (0.05 mg/kg per minute); if response is inadequate, infuse second loading bolus of 0.5 mg/kg over 1 minute and increase maintenance infusion to 100 mcg/kg (0.1 mg/kg) /min; increment; increase in this manner if required to maximum infusion rate of 300 mcg/kg 0.3 mg/kg /min</p> <p><u>Metoprolol</u> (1 specific blocker) 5 mg over 1 to 2 minutes repeated as required every 5 minutes to maximum dose of 15 mg</p> <p><u>Propranolol</u> (nonselective -blocker) 0.5 to 1 mg over 1 minute, repeated up to a total dose of 0.1 mg/kg</p>	Hypotension, bradycardia, precipitation of heart failure	Avoid in patients with asthma, COPD, decompensated heart failure and pre-excited AFIB or AFLUT



# IV Drugs to treat SVTs (con't)

ΦΑΡΜΑΚΟ	ΙΔΙΟΤΗΤΕΣ	ΕΝΔΕΙΞΕΙΣ	ΔΟΣΗ	ΑΕ	ΠΡΟΦΥΛΑΞΕΙΣ
<b>Procainamide</b>	Sodium and potassium channel blocker	<ul style="list-style-type: none"> <li>• Pre-excited AFIB</li> </ul>	20 to 50 mg/min until arrhythmia suppressed, hypotension ensues, or QRS prolonged by 50%, or total cumulative dose of 17 mg/kg; or 100 mg every 5 minutes until arrhythmia is controlled or other conditions described above are met	Bradycardia, hypotension, torsades de pointes	Avoid in patients with QTprolongation and CHF
<b>Amiodarone</b>	Multichannel blocker (sodium, potassium, calcium channel, and non competitive /- blocker)	<ul style="list-style-type: none"> <li>• Stable irregular narrow complex tachycardia (AFIB)</li> <li>• Stable regular narrow-complex tachycardia</li> <li>• To control rapid ventricular rate due to accessory pathway conduction in pre-excited atrial arrhythmias</li> </ul>	150 mg given over 10 minutes and repeated if necessary, followed by a 1 mg/min infusion for 6 hours, followed by 0.5 mg/min. Total dose over 24 hours should not exceed 2.2 g.	Bradycardia, hypotension, phlebitis	(-)
<b>Digoxin</b>	Cardiac glycoside with positive inotropic effects; slows AV node conduction –enhancing parasympathetic tone; slow onset of action	<ul style="list-style-type: none"> <li>• Stable, narrow-complex regular tachycardias if rhythm remains uncontrolled or unconverted by adenosine or vagal maneuvers or if SVT is recurrent</li> <li>• Control ventricular rate in patients with AFIB or AFLUT</li> </ul>	8 to 12 mcg/kg total loading dose, half of which is administered initially over 5 minutes, and remaining portion as 25% fractions at 4- to 8- hour intervals	Bradycardia	Slow onset of action and relative low potency renders it less useful for treatment of acute arrhythmias

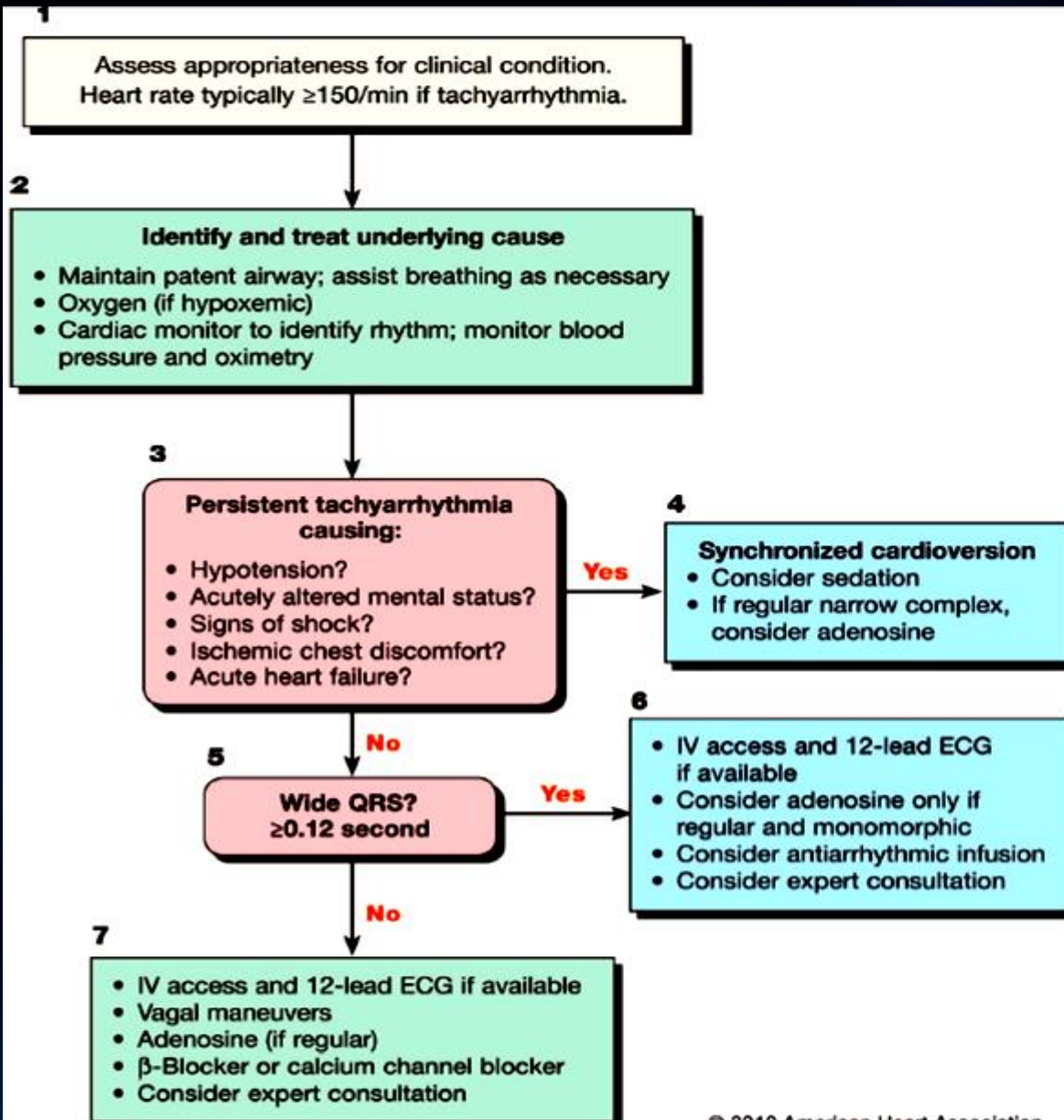


# IV Drugs to treat VTs

ΦΑΡΜΑΚΟ	ΙΔΙΟΤΗΤΕΣ	ΕΝΔΕΙΞΕΙΣ	ΔΟΣΗ	ΑΕ	ΠΡΟΦΥΛΑΞΕΙΣ
<b>Procainamide</b>	Sodium and potassium channel blocker	<ul style="list-style-type: none"> <li>Hemodynamically stable monomorphic VT</li> </ul>	20 to 50 mg/min until arrhythmia suppressed, hypotension ensues, or QRS prolonged by 50%, or total cumulative dose of 17 mg/kg; or 100 mg every 5 minutes until arrhythmia is controlled or other conditions described above are met	Bradycardia, hypotension, torsades de pointes	Avoid in patients with QTprolongation and CHF
<b>Amiodarone</b>	Multichannel blocker (sodium, potassium, calcium channel, and non competitive +/- blocker)	<ul style="list-style-type: none"> <li>Hemodynamically stable monomorphic VT</li> <li>Polymorphic VT with normal QT interval</li> </ul>	150 mg given over 10 minutes and repeated if necessary, followed by a 1 mg/min infusion for 6 hours, followed by 0.5 mg/min. Total dose over 24 hours should not exceed 2.2 g.	Bradycardia, hypotension, phlebitis	(-)
<b>Sotalol</b>	Potassium channel blocker and nonselective -blocker	<ul style="list-style-type: none"> <li>Hemodynamically stable monomorphic VT</li> </ul>	In clinical studies 1.5 mg/kg infused over 5 minutes; however, US package labeling recommends any dose of the drug should be infused slowly over a period of 5 hours	Bradycardia, hypotension, torsades de pointes	Avoid in patients with QTprolongation and CHF
<b>Lidocaine</b>	Relatively weak sodium channel blocker	<ul style="list-style-type: none"> <li>Hemodynamically stable monomorphic VT</li> </ul>	Initial dose range from 1 to 1.5 mg/kg IV; repeated if required at 0.5 to 0.75 mg/kg IV every 5 to 10 minutes up to maximum cumulative dose of 3 mg/kg; 1 to 4 mg/min (30 to 50 mcg/kg per minute) maintenance infusion	Slurred speech, altered consciousness, seizures, bradycardia	(-)
<b>Magnesium</b>	Cofactor in variety of cell processes including control of sodium and potassium transport	<ul style="list-style-type: none"> <li>Polymorphic VT associated with QT prolongation (torsades de pointes)</li> </ul>	1 to 2 g IV over 15 minutes	Hypotension, CNS toxicity, respiratory depression	Follow magnesium levels if frequent or prolonged dosing required, particularly in patients with impaired renal function



# Adult Tachycardia (with pulse); Algorithm



## Doses/Details

### Synchronized Cardioversion

Initial recommended doses:

- Narrow regular: 50-100 J
- Narrow irregular: 120-200 J biphasic or 200 J monophasic
- Wide regular: 100 J
- Wide irregular: defibrillation dose (NOT synchronized)

### Adenosine IV Dose:

First dose: 6 mg rapid IV push; follow with NS flush.  
Second dose: 12 mg if required.

### Antiarrhythmic Infusions for Stable Wide-QRS Tachycardia

#### Procainamide IV Dose:

20-50 mg/min until arrhythmia suppressed, hypotension ensues, QRS duration increases  $>50\%$ , or maximum dose 17 mg/kg given. Maintenance infusion: 1-4 mg/min. Avoid if prolonged QT or CHF.

#### Amiodarone IV Dose:

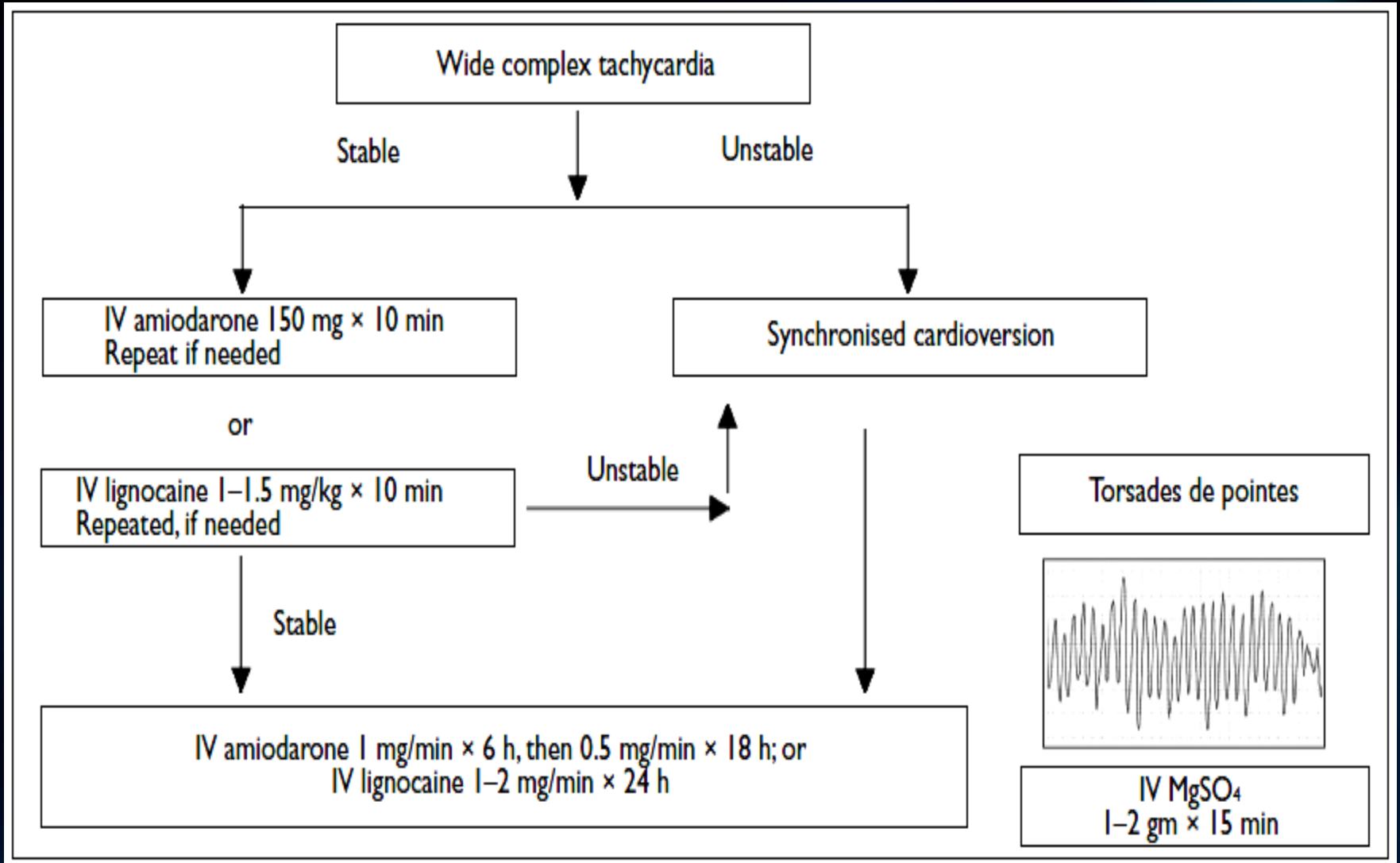
First dose: 150 mg over 10 minutes. Repeat as needed if VT recurs. Follow by maintenance infusion of 1 mg/min for first 6 hours.

#### Sotalol IV Dose:

100 mg (1.5 mg/kg) over 5 minutes. Avoid if prolonged QT.



# Wide complex tachycardia; Algorithm





# Synchronized Cardioversion

Synchronized cardioversion is shock delivery that is timed (synchronized) with the QRS complex. This synchronization avoids shock delivery during the relative refractory period of the cardiac cycle when a shock could produce VF.

## INDICATIONS:

- (1) unstable SVT
- (2) unstable atrial fibrillation
- (3) unstable atrial flutter
- (4) unstable monomorphic (regular) VT

If cardioversion is needed and it is impossible to synchronize a shock, use high-energy unsynchronized shocks (defibrillation doses).



# Synchronized Cardioversion

## Energy dose

The recommended initial biphasic energy dose for cardioversion of atrial fibrillation is 120 to 200 J (Class IIa, LOE A)

Cardioversion of atrial flutter and other SVTs generally requires less energy; an initial energy of 50 J to 100 J is often sufficient.

If the initial 50-J shock fails, the provider should increase the dose in a stepwise fashion (Class IIa, LOE A)

Cardioversion with monophasic waveforms should begin at 200 J and increase in stepwise fashion if not successful (Class IIa, LOE A)

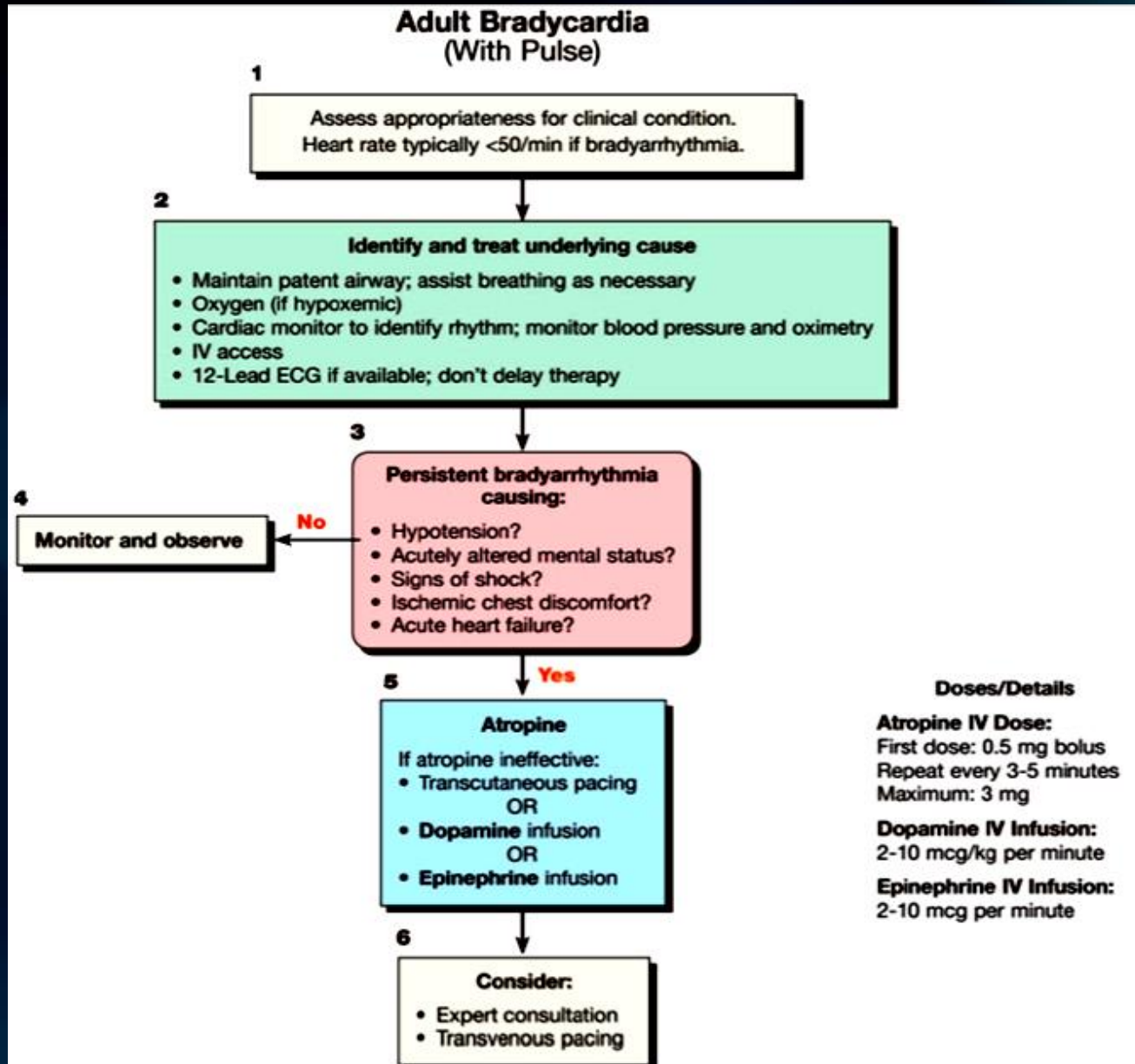
Monomorphic VT (regular form and rate) with a pulse responds well to monophasic or biphasic waveform cardioversion (synchronized) shocks at initial energies of 100 J. If there is no response to the first shock, it may be reasonable to increase the dose in a stepwise fashion.(Class IIb, LOE C)



# Management of Bradycardia



# Bradycardia algorithm





# Post-cardiac arrest care

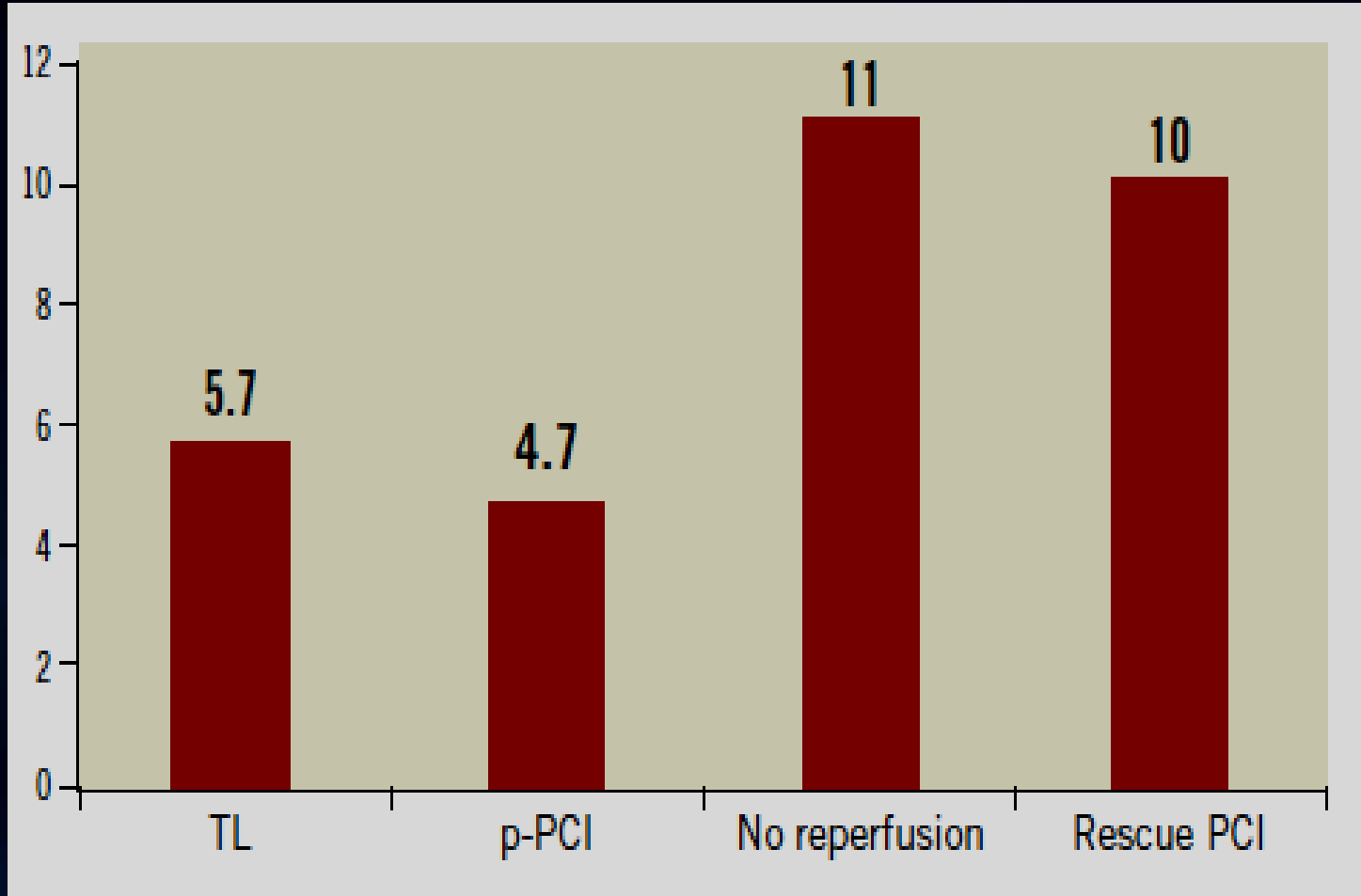
- Treatment of hypoxemia and hypotension
- Early diagnosis and treatment of ST-elevation myocardial infarction (STEMI) (Class I, LOE B)
- Therapeutic hypothermia in comatose patients (Class I, LOE B)

# Acute coronary syndromes



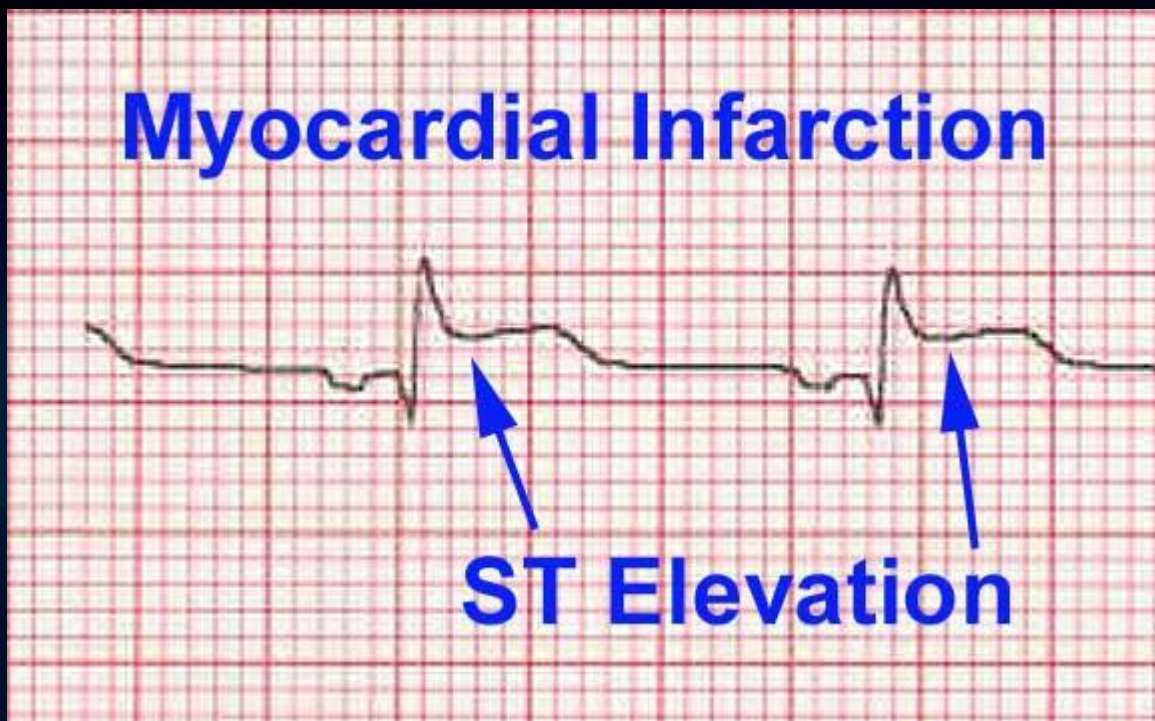
# STEMI

## In-hospital mortality in Greece



12%  
Θνητότητα  
στους 6 μήνες

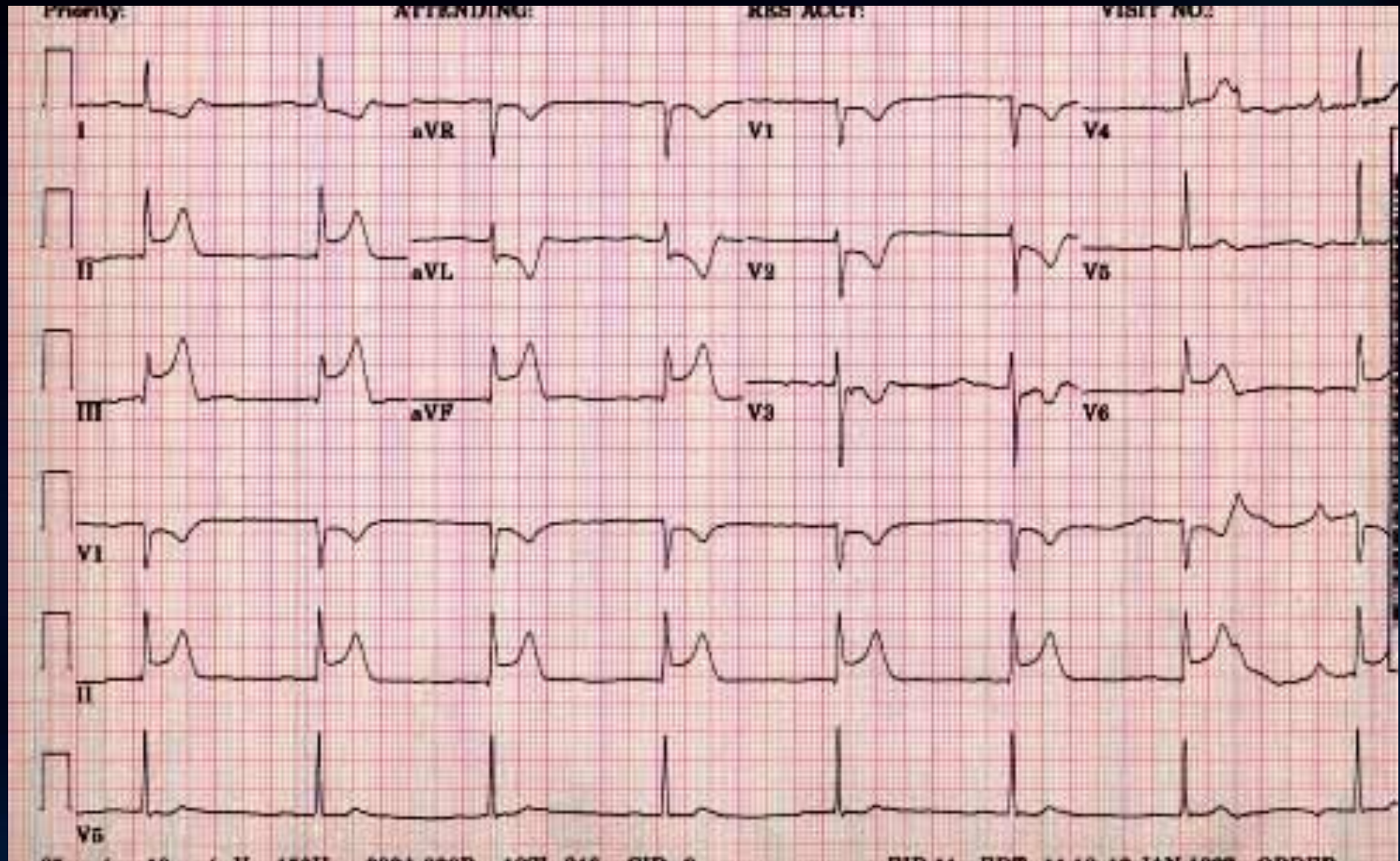
# Οξύ έμφραγμα του μυοκαρδίου (STEMI) Ηλεκτροκαρδιογράφημα



- Ανάσπαση του ST διαστήματος με το κυρτό προς τα πάνω
- Εικόνα κατόπτρου
- Νεομφανιζόμενος αποκλεισμός αριστερού σκέλους (LBBB)
- Εμφάνιση κολποκοιλιακών αποκλεισμών
- Εμφάνιση αρρυθμιών

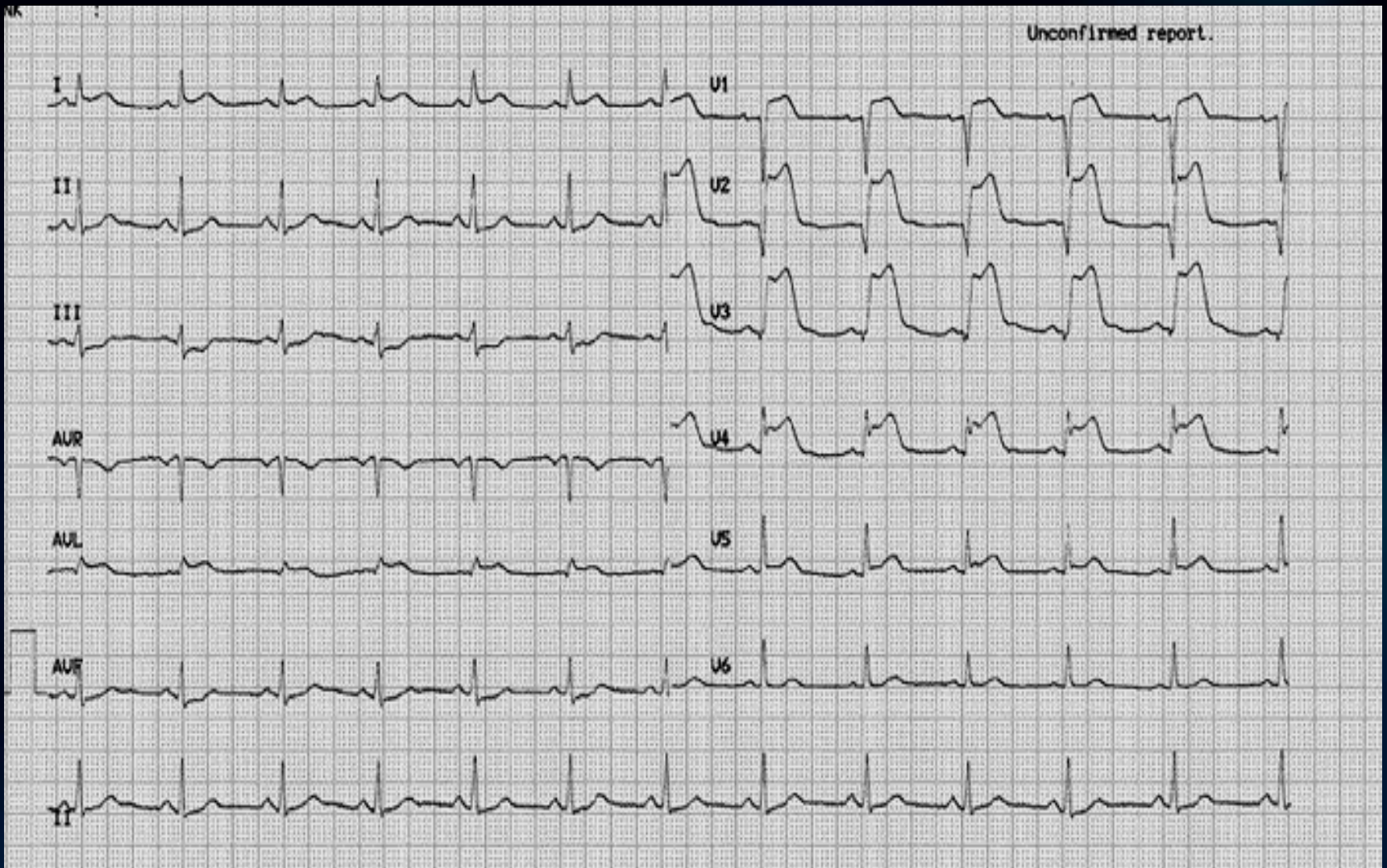


# Οξύ έμφραγμα του μυοκαρδίου κατωτέρου τοιχώματος





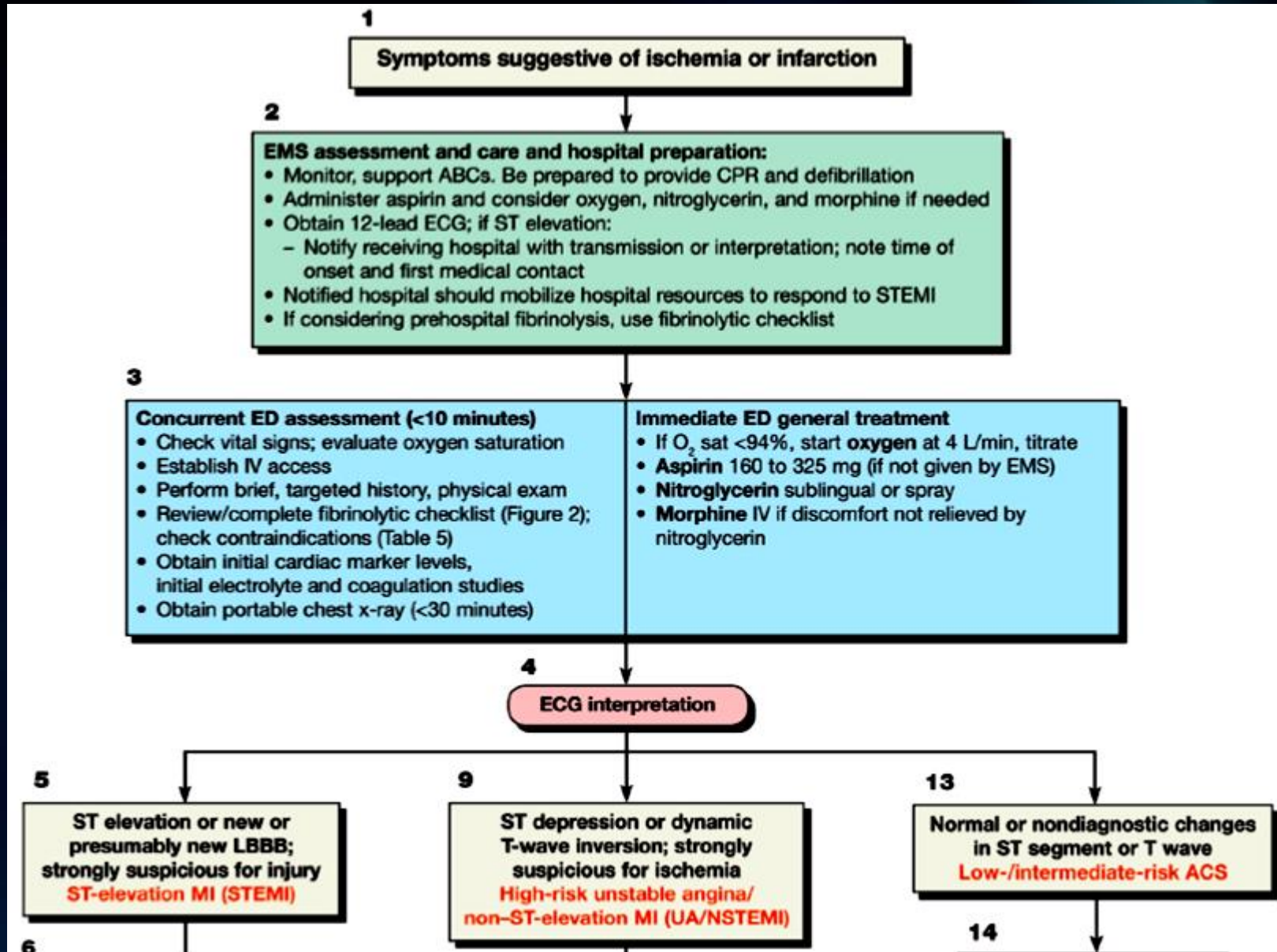
# Οξύ έμφραγμα του μυοκαρδίου προσθίου τοιχώματος





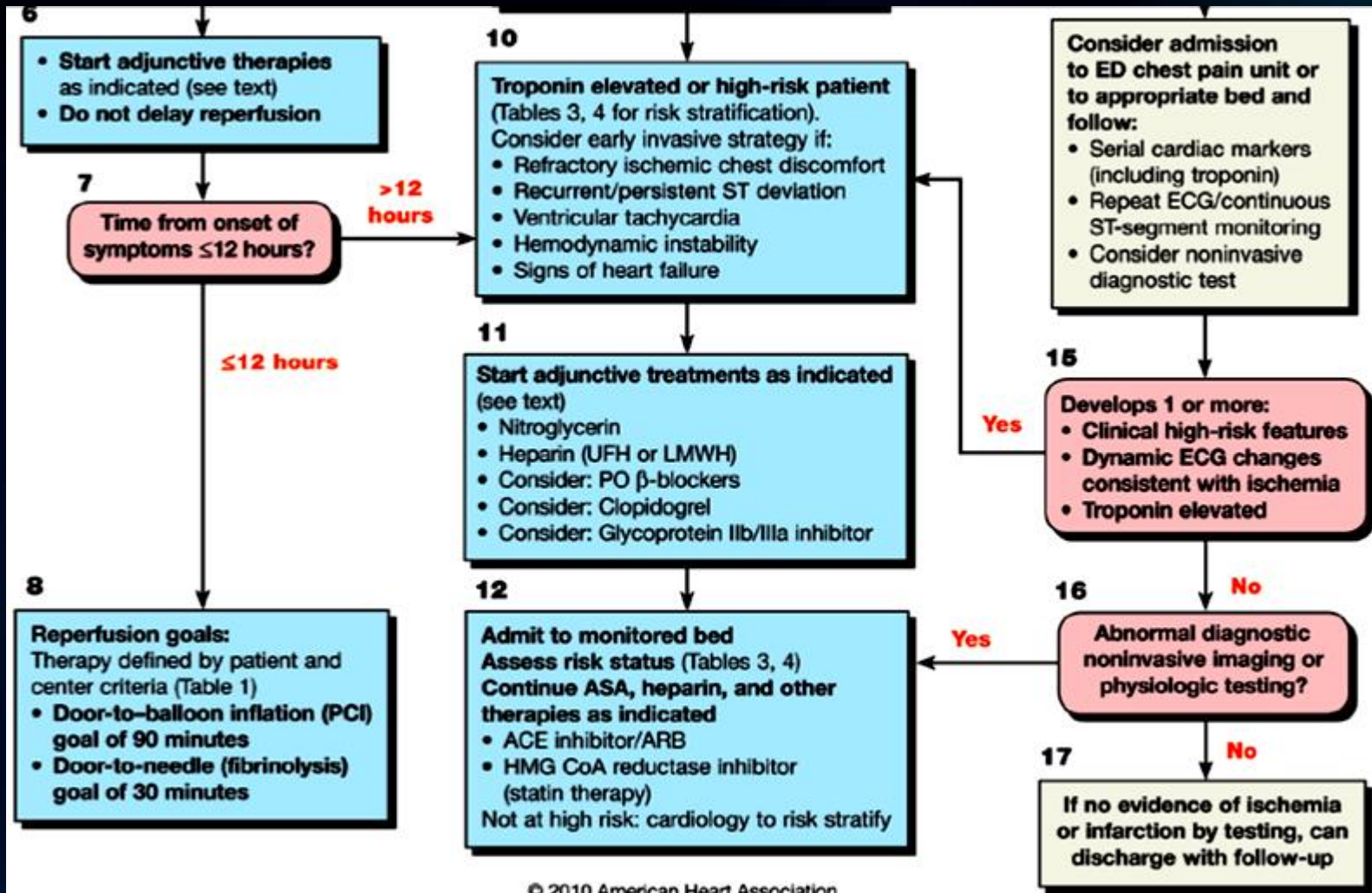


# Acute Coronary Syndromes





# Acute Coronary Syndromes (con't)



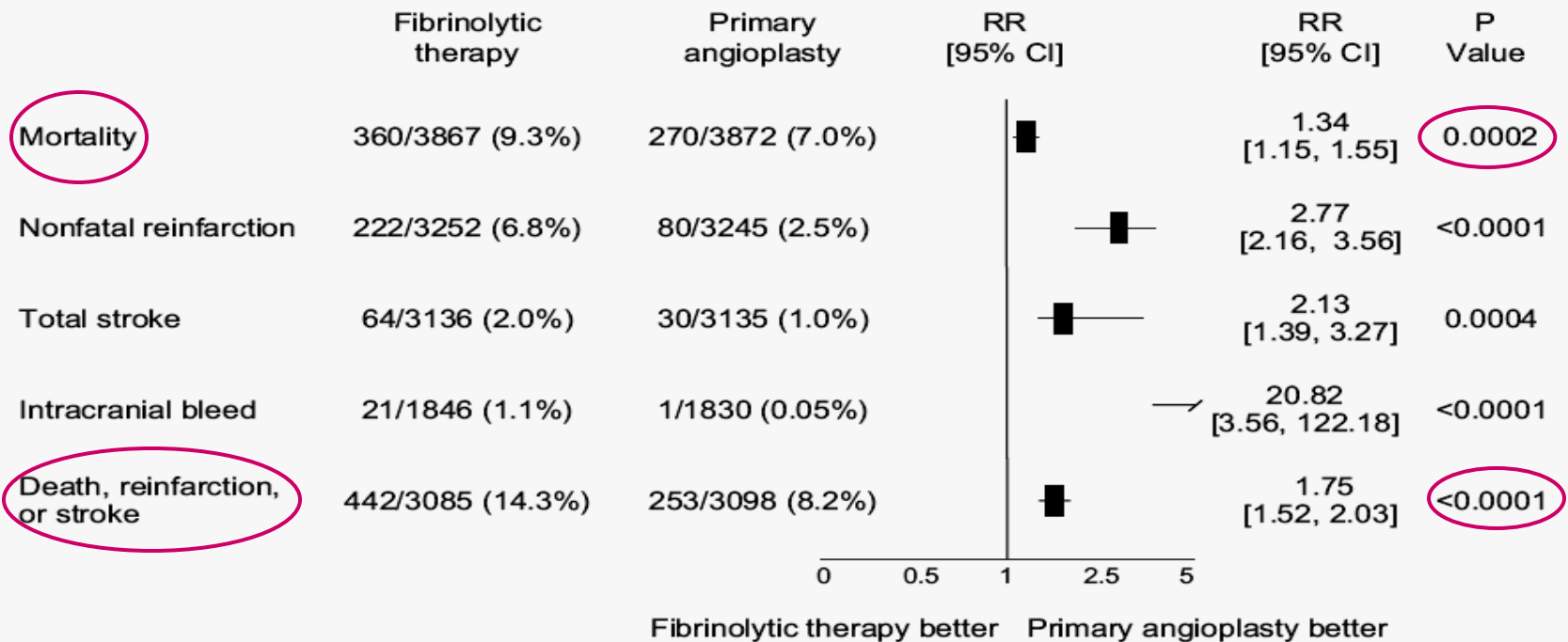
© 2010 American Heart Association



# Αντιμετώπιση στο νοσοκομείο

## Πρωτογενής αγγειοπλαστική vs Θρομβόλυση

*Meta-analysis of 23 randomized trials, n= 7739*



*Fibrinolysis only in cases that a PPCI cannot be performed within the recommended time (<2h from the first medical contact in any case or < 90 min in pts with onset of symptoms <2h)*



# Barriers related to Fibrinolysis

## Absolute Contraindications

- Any prior intracranial hemorrhage
- Known structural cerebral vascular lesion (eg. AVM)
- Known malformation
- Ischemic stroke
- Suspected aortic dissection
- Active bleeding
- Significant coagulopathy

## Relative Contraindications

- History of cerebral hemorrhage
- Severe uncontrolled hypertension (>180/100 mm Hg)†
- History of peptic ulcer disease not covered in current guidelines
- Traumatic cranial injury
- Recent (within 3 weeks) surgery
- Noncompressible hematoma
- For streptococcal meningitis: use of fibrinolytic agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

**Failed thrombolysis  
in as much as 30% of  
the cases**

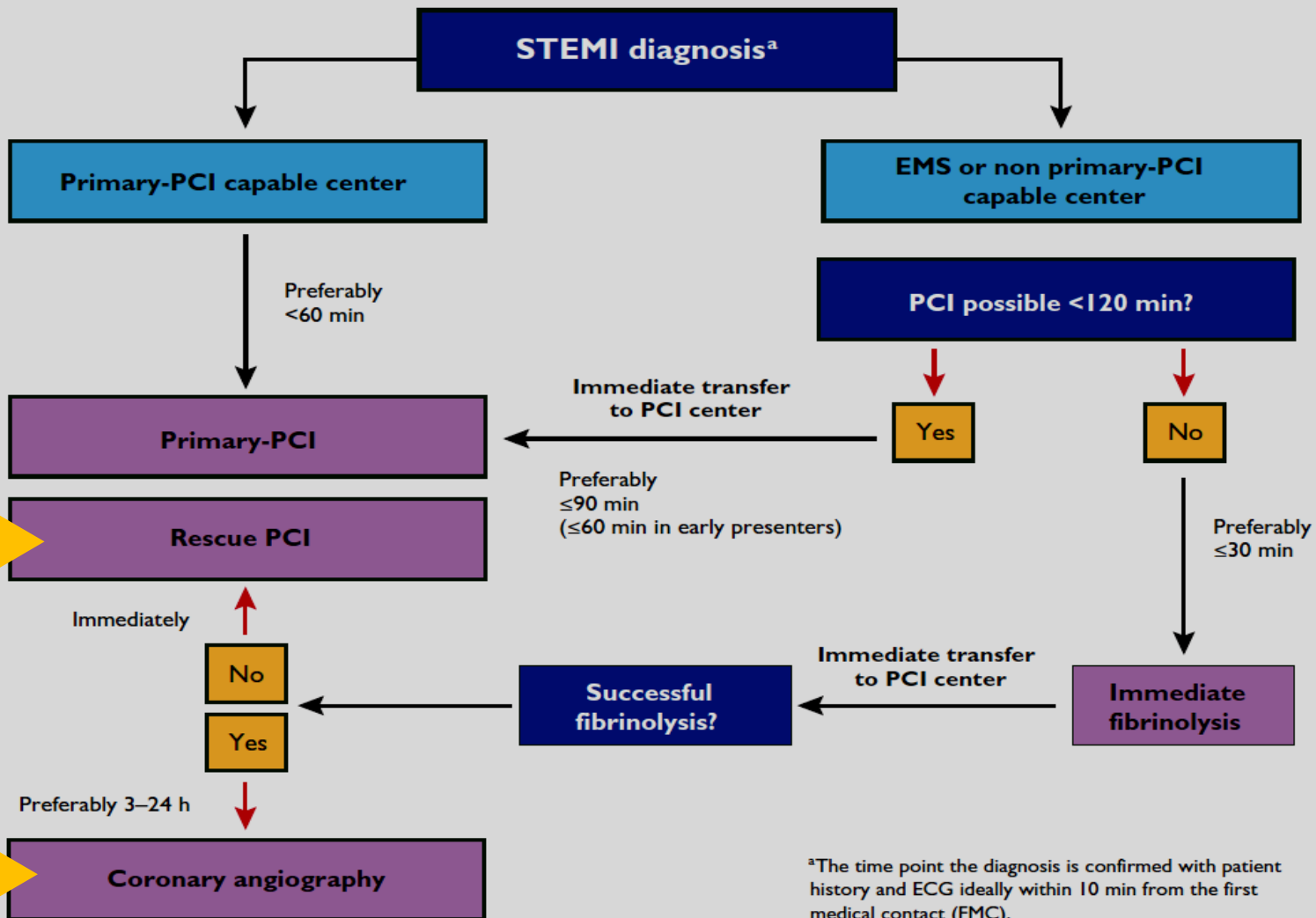
**Intracranial bleeding  
0,25-2,5%**

10 mm Hg)†  
thology not

action to these



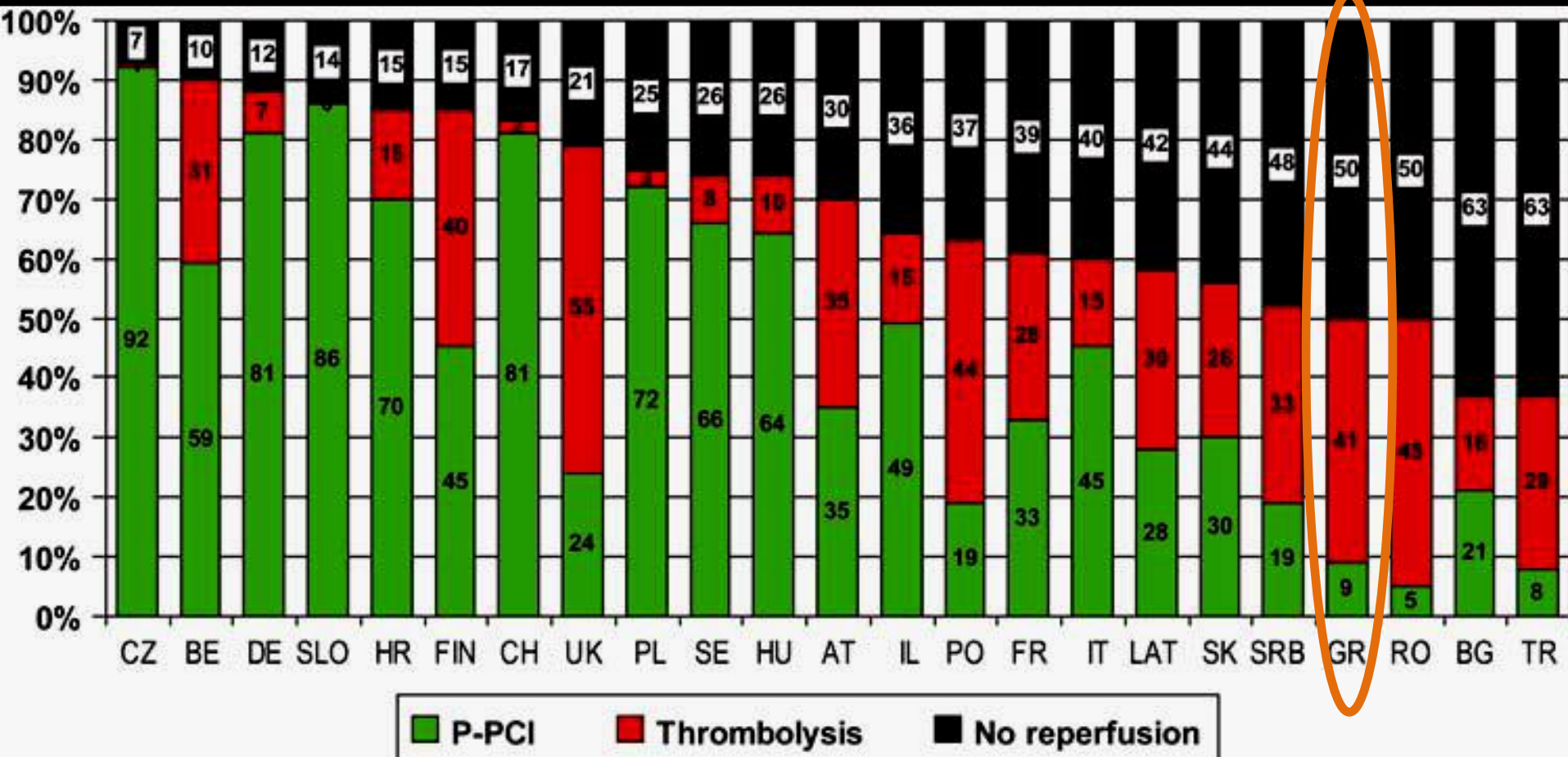
# STEMI Primary PCI





# Θεραπεία ΟΕΜ στην Ευρώπη 2004-2008

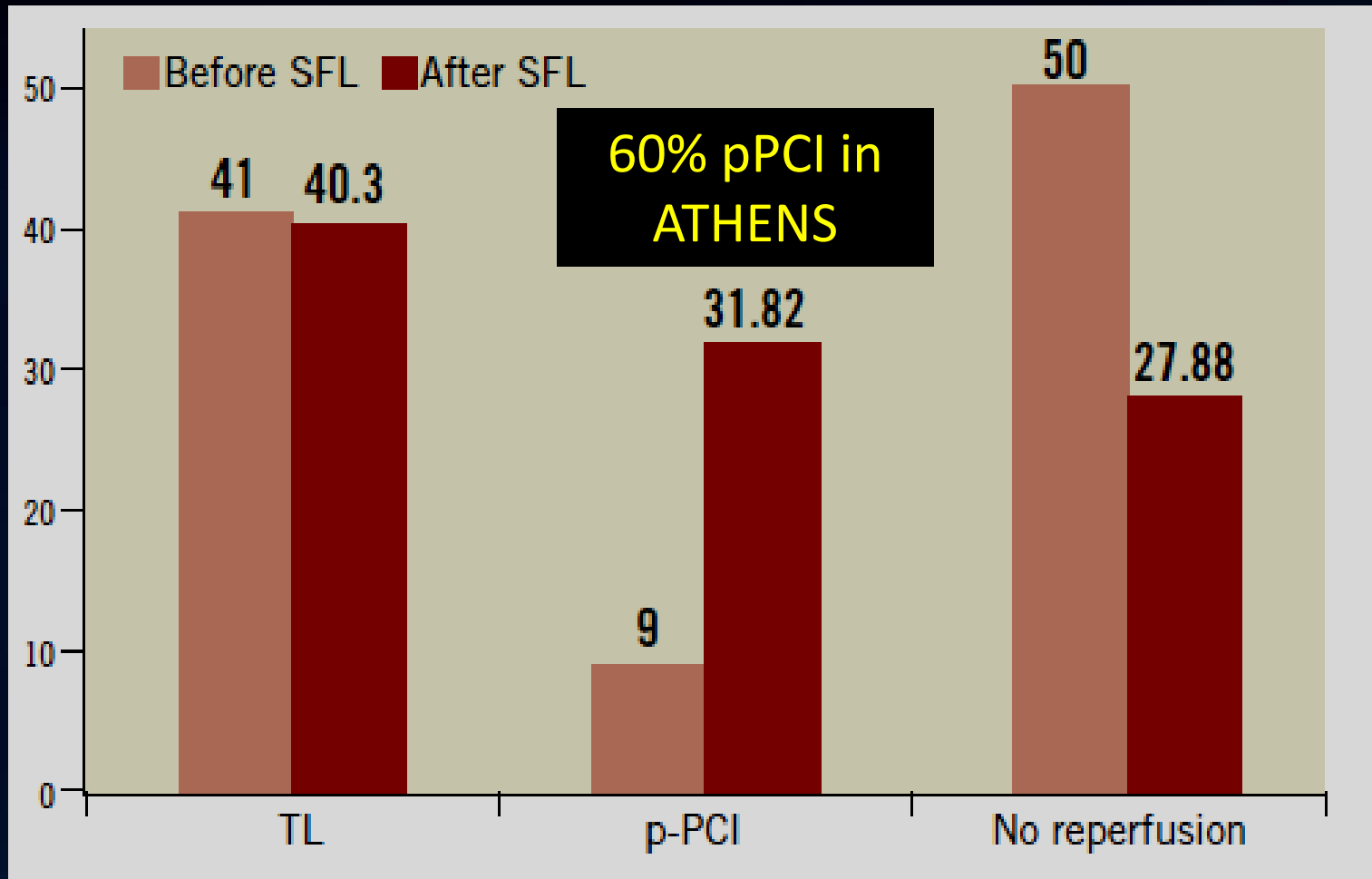
50% των ΟΕΜ στην Ελλάδα δε λάμβαναν θεραπεία επαναιμάτωσης, 41% θρομβόλυση και μόλις 9% πρωτογενή αγγειοπλαστική





# STEMI

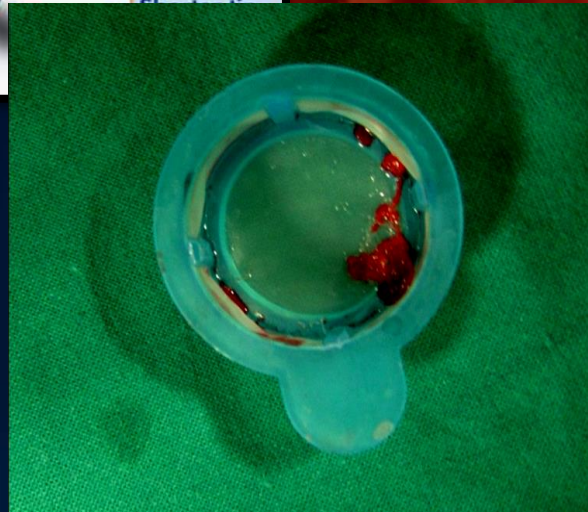
## Θεραπείες επαναιμάτωσης στην Ελλάδα





# Αντιμετώπιση στο νοσοκομείο

## Αναρρόφηση θρόμβου

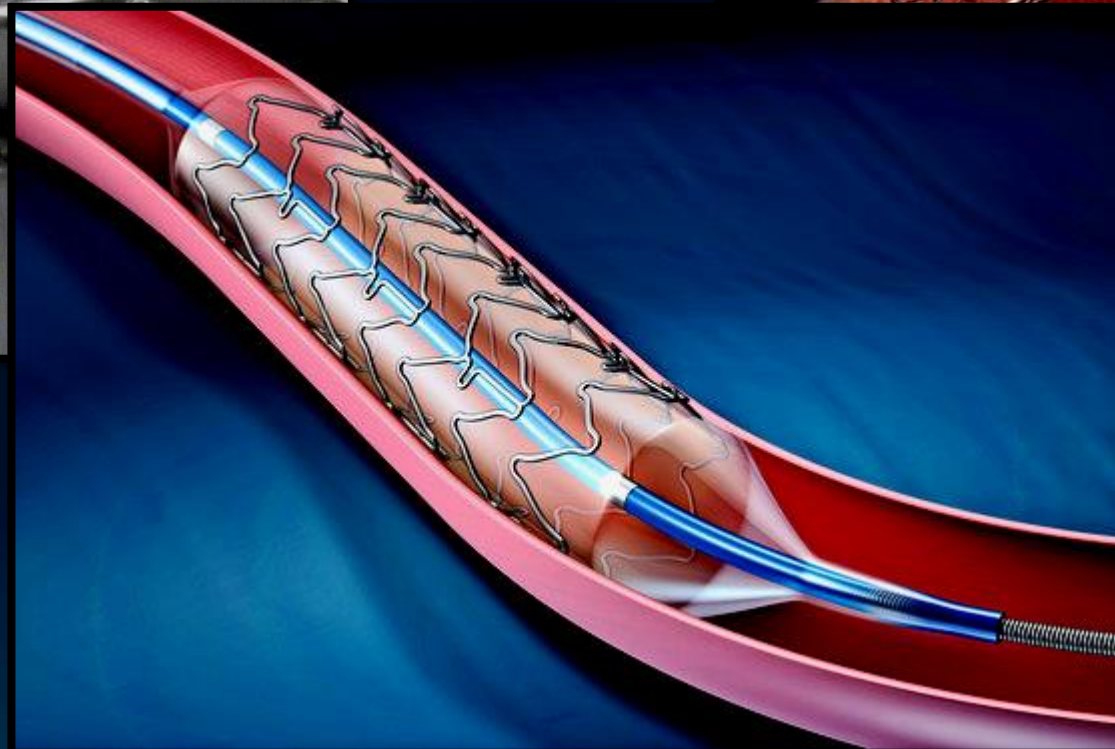
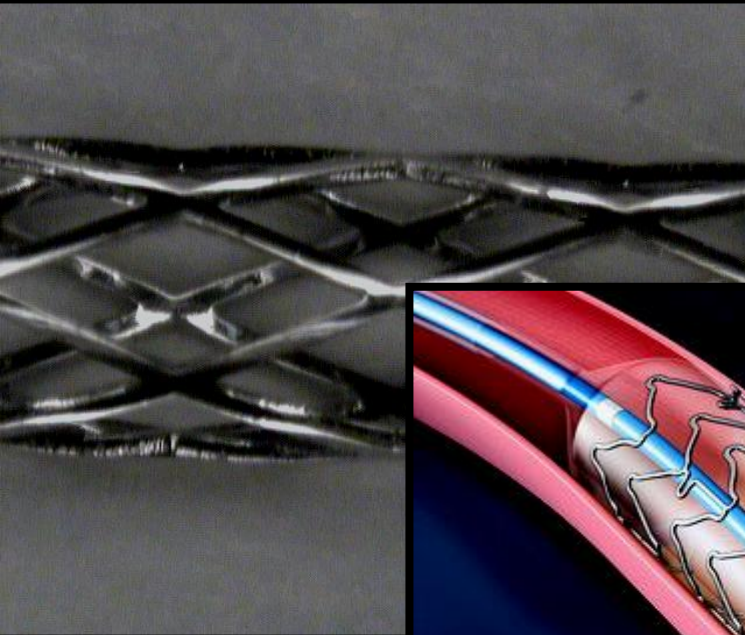






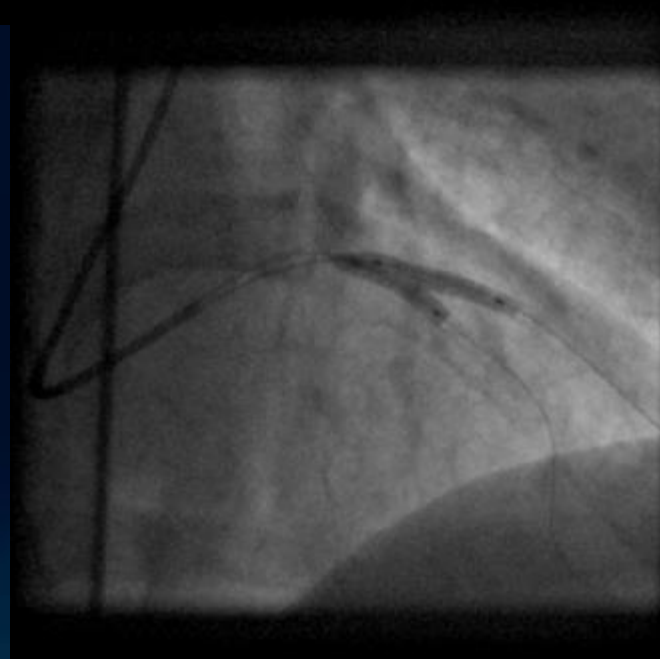
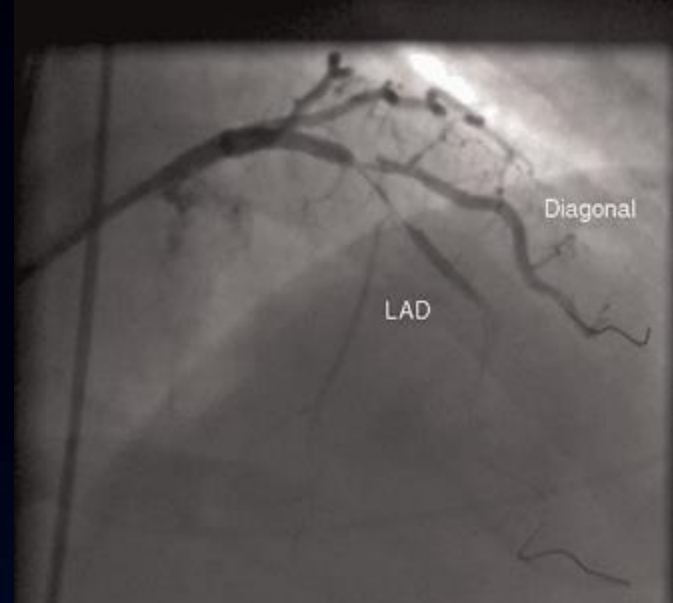
# Αντιμετώπιση στο νοσοκομείο

## Stents





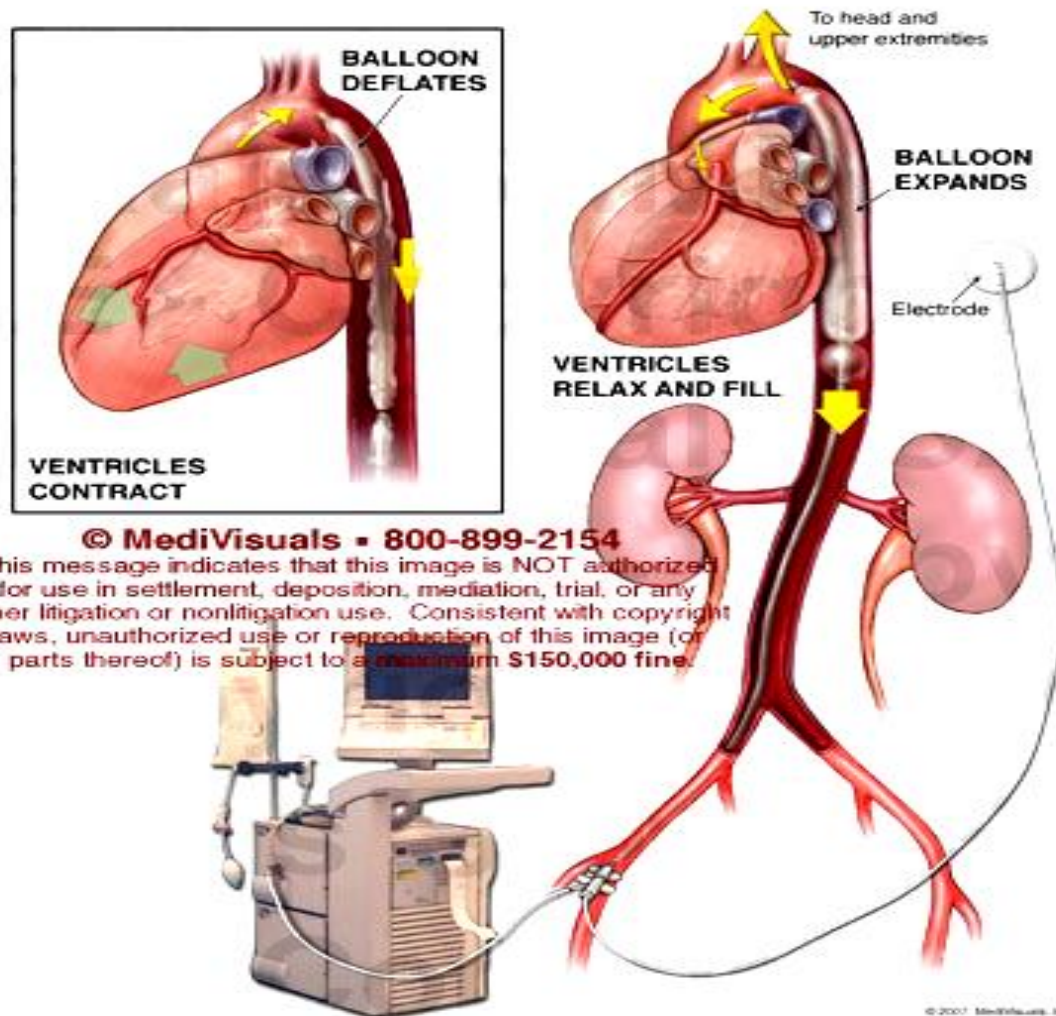
# Στεφανιογραφία





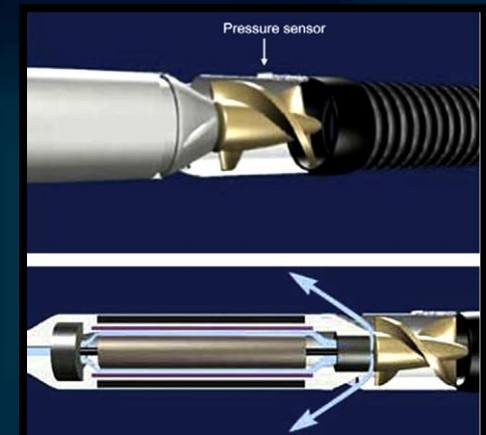
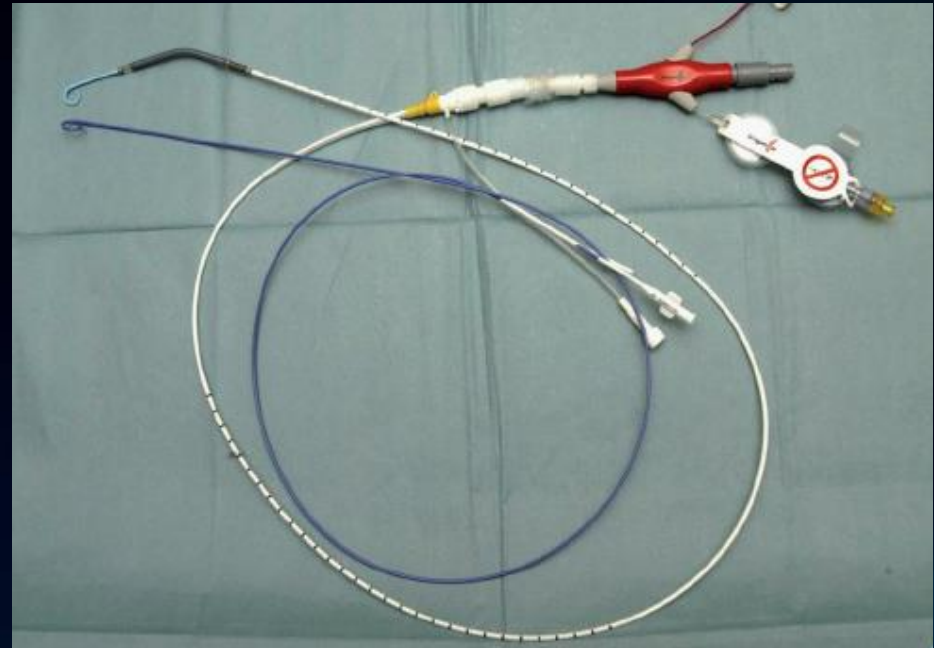
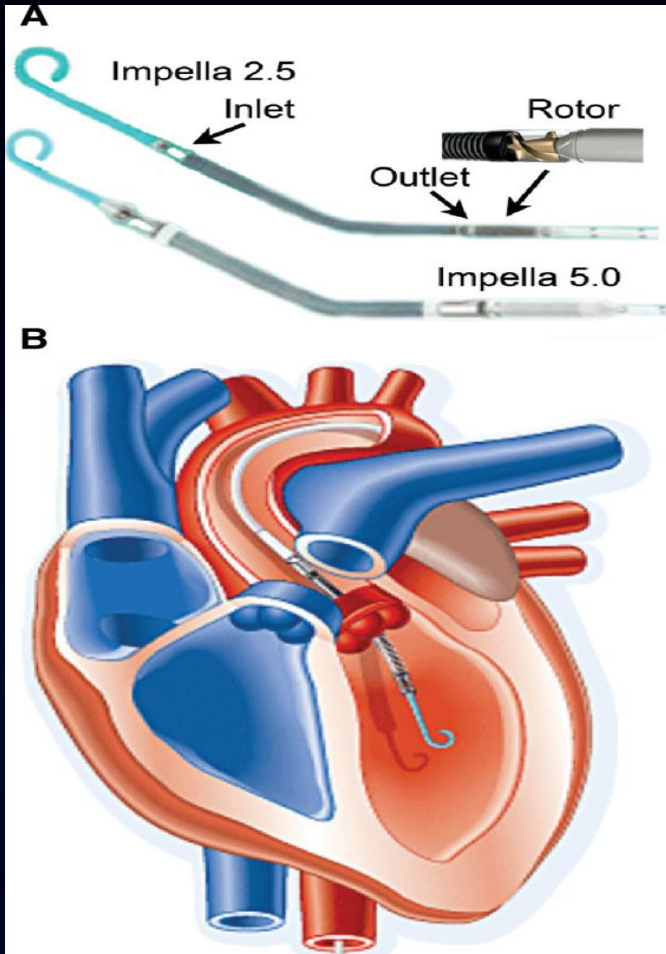
# Μηχανική υποβοήθηση Ενδαορτική αντλία αντώθησης

## Intra-Aortic Balloon Pump (Assist Device)



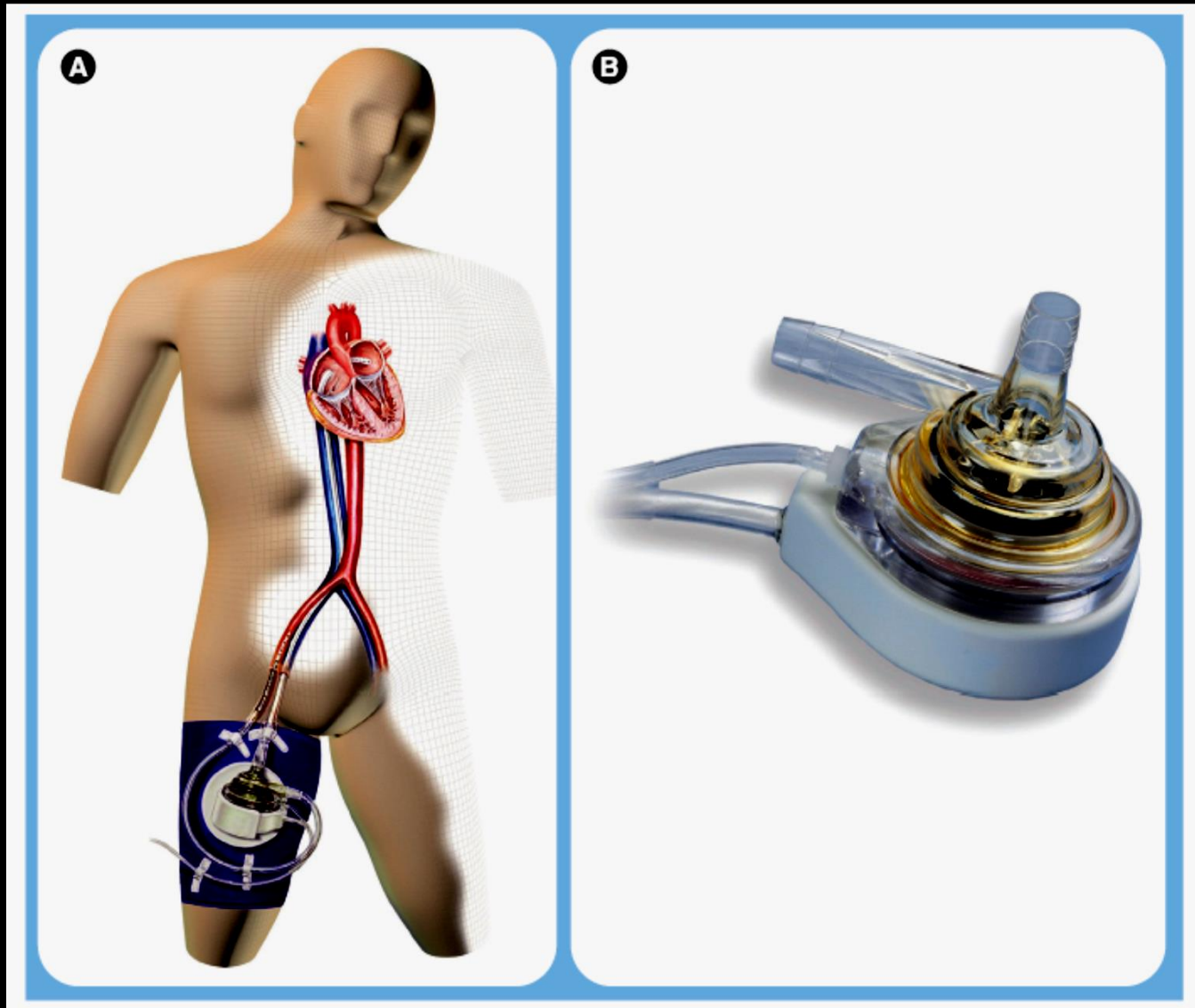


# Μηχανική υποβοήθηση Impella





# Μηχανική υποβοήθηση Tandem Heart



**ΕΥΧΑΡΙΣΤΩ ΠΟΛΥ**

**BACK UP**

**FFR vs IVUS**

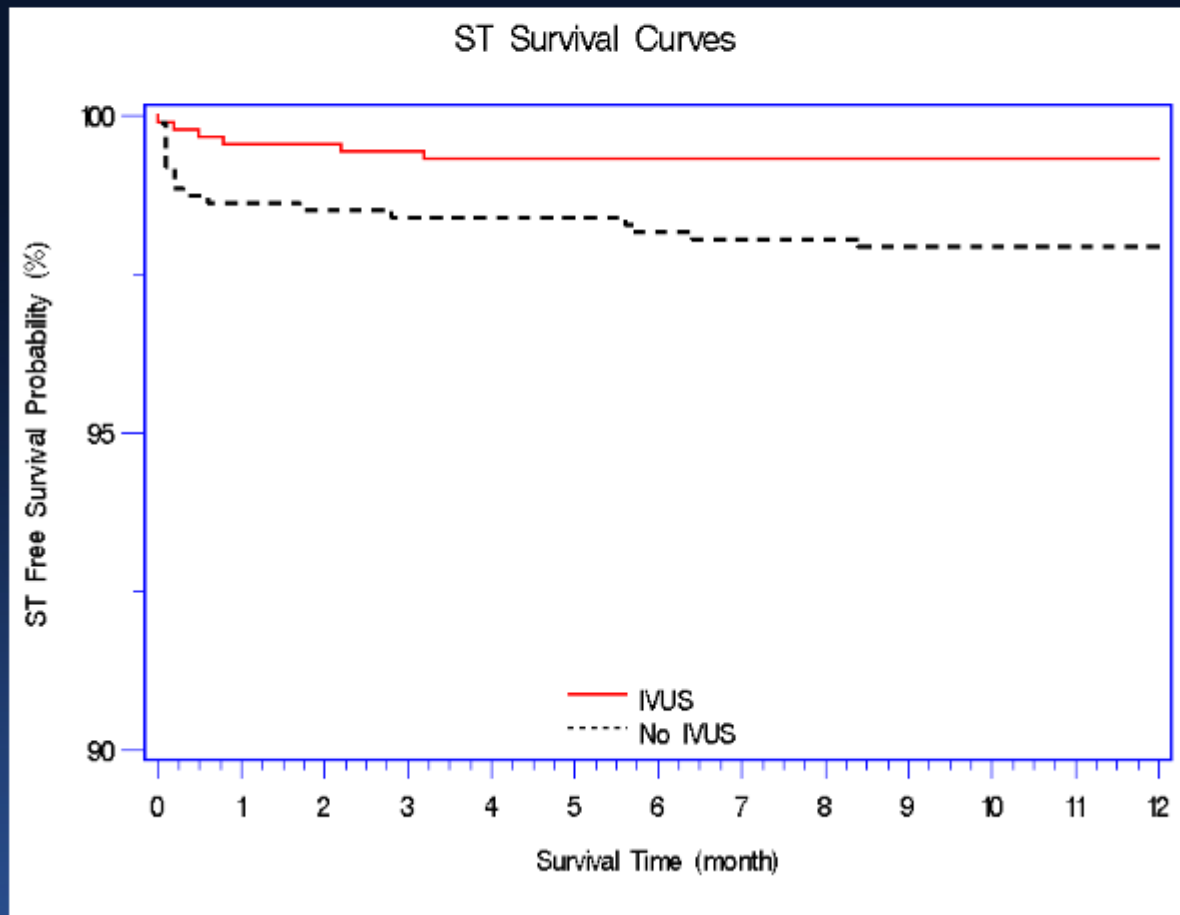
**Optimizing the PCI results**



# IVUS-guided PCI

## IVUS Guided Stent Implantation Reduced Risk of Stent Thrombosis

### *Stent Thrombosis – Event-Free Curves*



# Guidelines on revascularization; The role of FFR



EUROPEAN  
SOCIETY OF  
CARDIOLOGY®



FFR-guided PCI is recommended for detection of ischaemia-related lesion(s) when objective evidence of vessel-related ischaemia is not available.

I

A

American Heart  
Association<sup>SM</sup>  
Fighting Heart Disease  
and Stroke



## **CLASS IIa**

1. Fractional flow reserve is reasonable to assess angiographic intermediate coronary lesions (50% to 70% diameter stenosis) and can be useful for guiding revascularization decisions in patients with SIHD (89,244–247). (*Level of Evidence: A*)



# European guidelines on myocardial revascularization; The role of IVUS

IVUS-guided stent implantation may be considered for unprotected left main PCI.	IIb	C
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## The role of IVUS VH

Although the PROSPECT trial<sup>243</sup> provided new insights regarding indications for stent implantation, the role of tissue characterization for everyday practice remains to be established.

# AHA/ACC PCI guidelines;

American Heart  
Association<sup>SM</sup>  
Fighting Heart Disease  
and Stroke



## The role of IVUS



### **CLASS IIb**

1. IVUS may be reasonable for the assessment of non-left main coronary arteries with angiographically intermediate coronary stenoses (50% to 70% diameter stenosis) (248,255,256). (*Level of Evidence: B*)
2. IVUS may be considered for guidance of coronary stent implantation, particularly in cases of left main coronary artery stenting (249,254,257). (*Level of Evidence: B*)

### **CLASS IIa**

1. IVUS is reasonable for the assessment of angiographically indeterminate left main CAD (248-250). (*Level of Evidence: B*)

# 'Old classical dogmas or old classical beliefs that cannot be doubted..'



1. “dye don’t lie”

Applegate R, JACC 2010

2. “dye and decide”

3. “a coronary artery stenosis is severe if it **looks** severe even in one projection”



# FFR: basic principle

$$FFR = Q_s / Q_n \quad (1)$$

$Q_s$ : maximal flow to the myocardium in the presence of a stenosis

$Q_n$ : maximal flow to the myocardium without a stenosis

*Ohm's law*

$$R = P / Q \Rightarrow Q = P / R$$

(2)

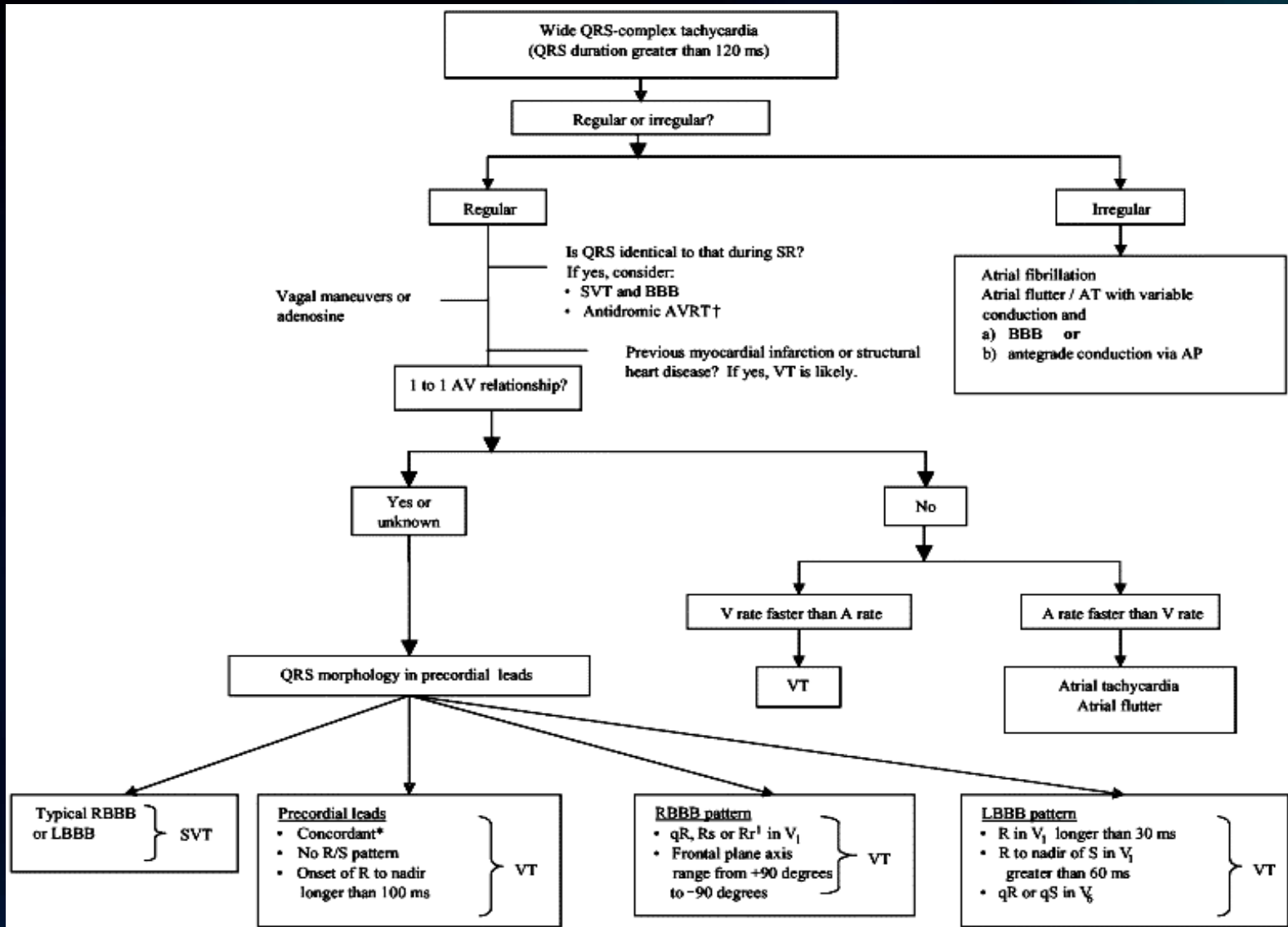
P: pressure, Q: flow, R: resistance

$$(1) \& (2) \Rightarrow FFR_{\text{myo}} = \frac{Q_s}{Q_n} = \frac{P_s / R_s}{P_n / R_n} \quad (3)$$

Under maximal hyperemia:  $R_s = R_n$

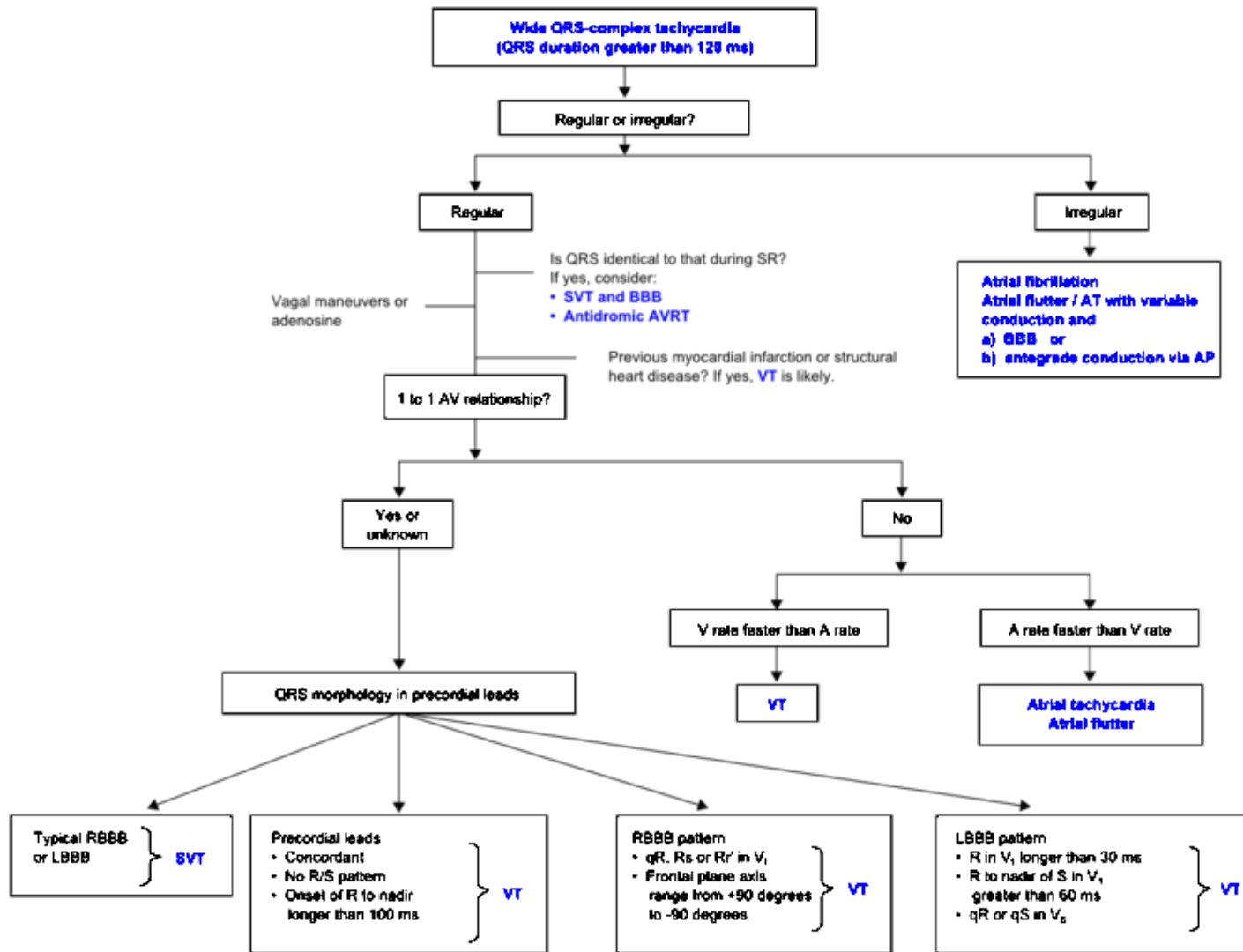


# IV Drugs to treat SVTs





# IV Drugs to treat SVTs







# Prehospital fibrinolysis check list

