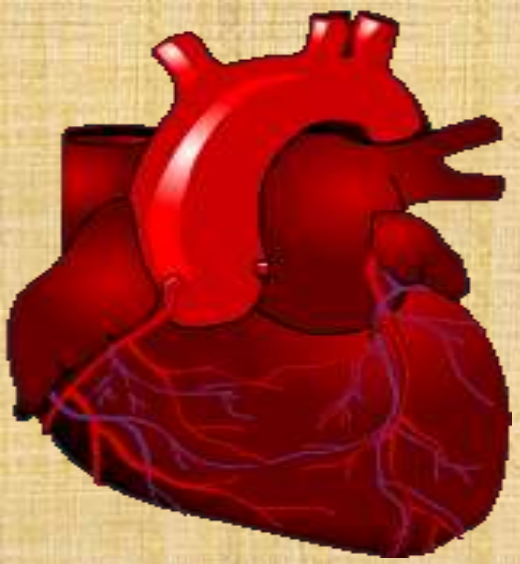
Cardiac rhythm during CPR



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استادیار دانشگاه علوم پزشکی گیلان

- Electrical mechanisms of cardiac arrest are divided into :
  - tachyarrhythmic
  - bradyarrhythmic-asystolic
- The **tachyarrhythmias** include
  - VF
  - pulseless or sustained VT

in which adequate blood flow cannot be maintained and perfusion is inadequate to meet the body's needs.

- **Bradyarrhythmic-asystolic** events include:

- Severe bradyarrhythmias
- Dissociation between spontaneous electrical activity and mechanical function (PEA)
- Asystole

- To qualify as a mechanism of cardiac arrest, severe bradyarrhythmias must be slow enough to result in an inability to adequately perfuse and maintain consciousness, which usuallyrequires a heart rate of less than 20 beats/min.
- In PEA, the electrical rate can be considerably faster, but there is no perfusion because of:

✓ inadequate or absent mechanical activity

✓ mechanical obstruction to blood flow as in massive pulmonary embolism.

- It is likely that many victims found to be asystolic at contact were initially in VF or VT.
- After a variable time, fibrillation may cease and asystole or PEA emerges. In contrast to earlier data, the most common initial recording documented in recent years is asystole or PEA, which can continue as such or very rarely transform into VF.

- Asystolic arrest:
- The basic electrophysiologic mechanism in this form of arrest is failure of normal subordinate automatic activity to assume the pacemaking function of the heart in the absence of normal function of the sinus node, AV junction, or both.

- Asystolic arrest is more common in severely diseased hearts and in patients with a number of endstage disorders, cardiac and noncardiac.

- These mechanisms may result, in part, from diffuse involvement of subendocardial Purkinje fibers in advanced heart disease.

- Pulseless Electrical Activity PEA, formerly called electromechanical dissociation, is separated into :

1. **primary**
2. **secondary forms**

- No one unifying definition for PEA, chanistically or clinically, is recognized. The common denominator in both is the presence of organized cardiac electrical activity in the absence of effective mechanical function.

The absence of rapid spontaneous return of circulation is important in that it excludes transient losses of cerebral blood flow, such as the various patterns of vasovagal reflex syncope, which have different clinical implications than the meaning attributed to true PEA..

- The secondary form of PEA includes causes that result from an abrupt cessation of cardiac venous return, such as:
  - ✓ massive pulmonary embolism,
  - ✓ acute malfunction of prosthetic valves,
  - ✓ cardiac tamponade
  - ✓ hemopericardium.
- The **primary** form is the more familiar; in this form none of these obvious mechanical factors is present, but ventricular muscle fails to produce an effective contraction despite continued electrical activity (i.e., failure of electromechanical coupling). It usually occurs as an end-stage event in advanced heart disease, but it can occur in patients with acute ischemic events or, more commonly, after electrical resuscitation from prolonged cardiac arrest

- . Although it is not thoroughly understood, it appears that diffuse disease, metabolic abnormalities, or global ischemia provides the pathophysiologic substrate. The proximate mechanism for failure of electromechanical coupling may be abnormal intracellular Ca2+ metabolism, intracellular acidosis, or perhaps depletion of ATP.

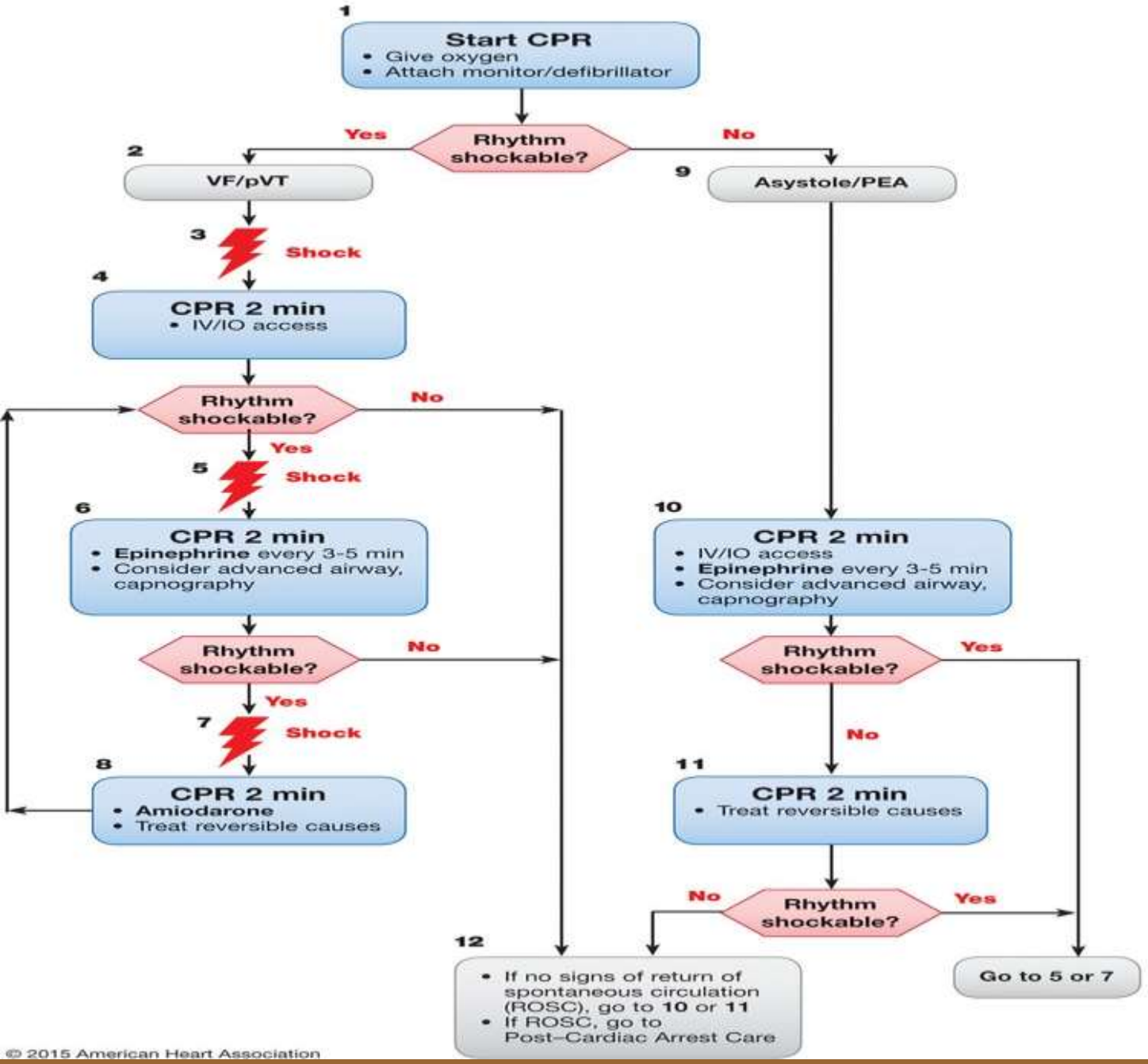
Importance of Electrical Mechanisms

- Some studies have now suggested that less than 50% of victims have shockable rhythms at initial contact. This fact is associated with a reduction in cumulative survival probability with community-based interventions.
- The electrical mechanism of out-of-hospital cardiac arrest, as defined by the initial rhythm recorded by emergency rescue personnel, has a powerful impact on outcome. The subgroup of patients who are in sustained VT at the time of first contact, although small, has the best outcome. Eighty-eight percent of patients in cardiac arrest related to VT were successfully resuscitated and admitted to the hospital alive, and 67% were ultimately discharged alive. However, this relatively low-risk group represents only 7% to 10% of all cardiac arrests. Because of the inherent time lag between collapse and initial recordings, it is likely that many more cardiac arrests begin as rapid sustained VT and degenerate into VF before the arrival of rescue personnel.

- Patients with a bradyarrhythmia or with asystole or PEA at initial contact have the worst prognosis; only 9% of such patients in the Miami study were admitted to the hospital alive, and none were discharged.
- In a later experience, some improvement in outcome was noted, although the improvement was limited to patients in whom the initial bradyarrhythmia recorded was an idioventricular rhythm that responded promptly to chronotropic agents in the field. In a large prospective observational in-hospital study of cardiac arrests in children and adults, children had a higher probability of asystole or PEA as the initial documented rhythm but had a better overall survival rate because they had better outcomes of interventions for these rhythms than adults did.148 Overall survival after PEA appears to be better in recent years,149 but it is not clear whether this applies to asystole.

- Bradyarrhythmias also have adverse prognostic implications after defibrillation from VF in the field. Patients with a heart rate lower than 60 beats/min after defibrillation, regardless of the specific bradyarrhythmic mechanism, had a poor prognosis, with 95% of such patients dying before hospitalization or in the hospital. The outcome in the group of patients in whom VF is the initial rhythm recorded is intermediate between the outcomes associated with sustained VT and with bradyarrhythmia and asystole. Of such patients, 40% were resuscitated successfully and admitted to the hospital alive, and 23% were ultimately discharged alive.

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

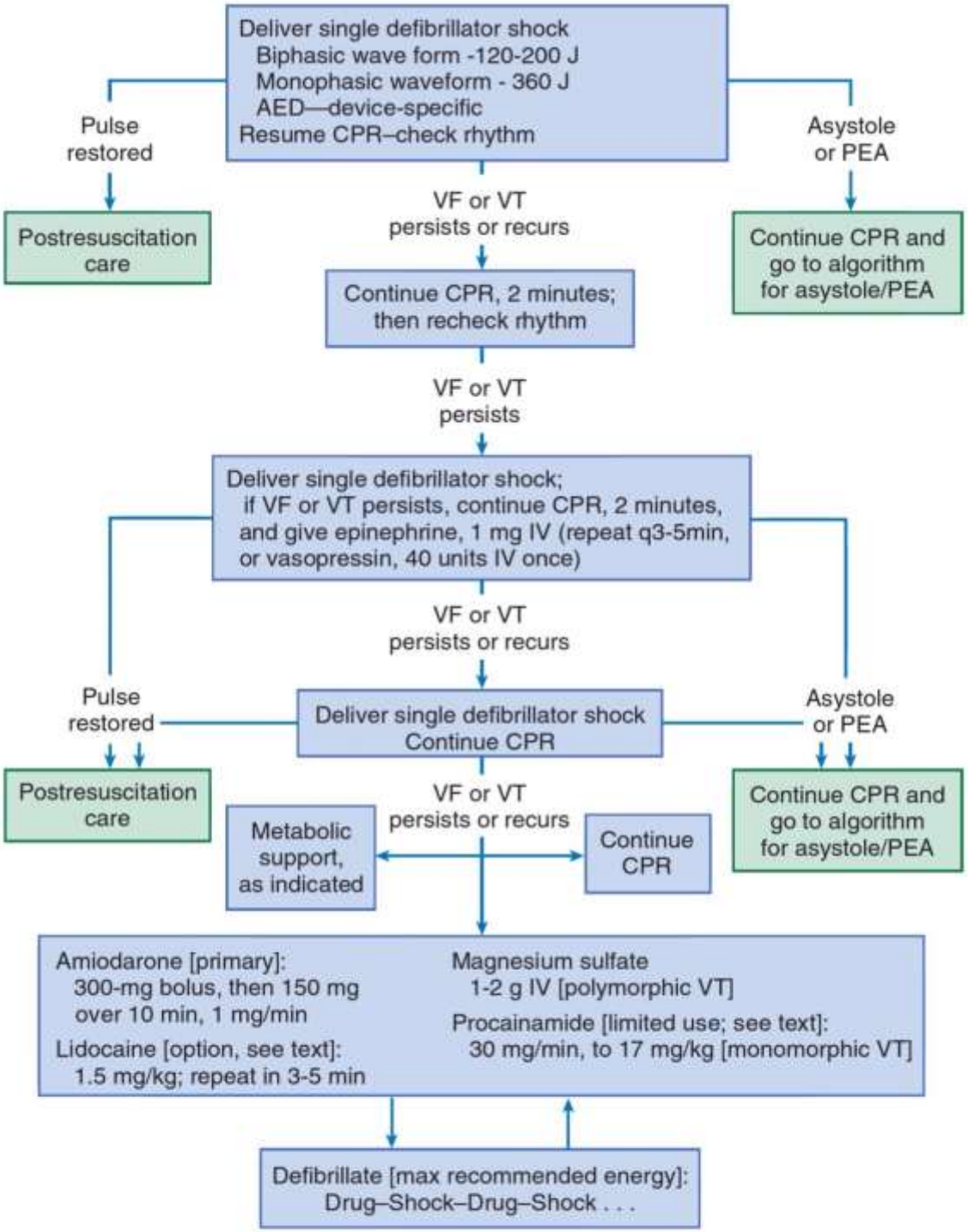


FIGURE 39-17 Advanced life support for VF and pulseless VT. If initial defibrillation fails, the patient should be intubated and intravenous access established immediately while CPR is continued. Epinephrine, 1 mg intravenously, should be administered and may be repeated several times with additional defibrillation attempts at 360 J. If conversion is still unsuccessful, epinephrine may be administered again, although it is unlikely that higher doses will provide any further benefit. Sodium bicarbonate should be administered at this time only if the patient is known to be hyperkalemic, and intravenous antiarrhythmic drugs should be tried (see text). Additional attempts to defibrillate should follow the administration of each drug attempted. Concomitant with all steps, continuation of CPR is paramount. (Modified from 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science. Circulation 122[Suppl 3]:S640, 2010.)

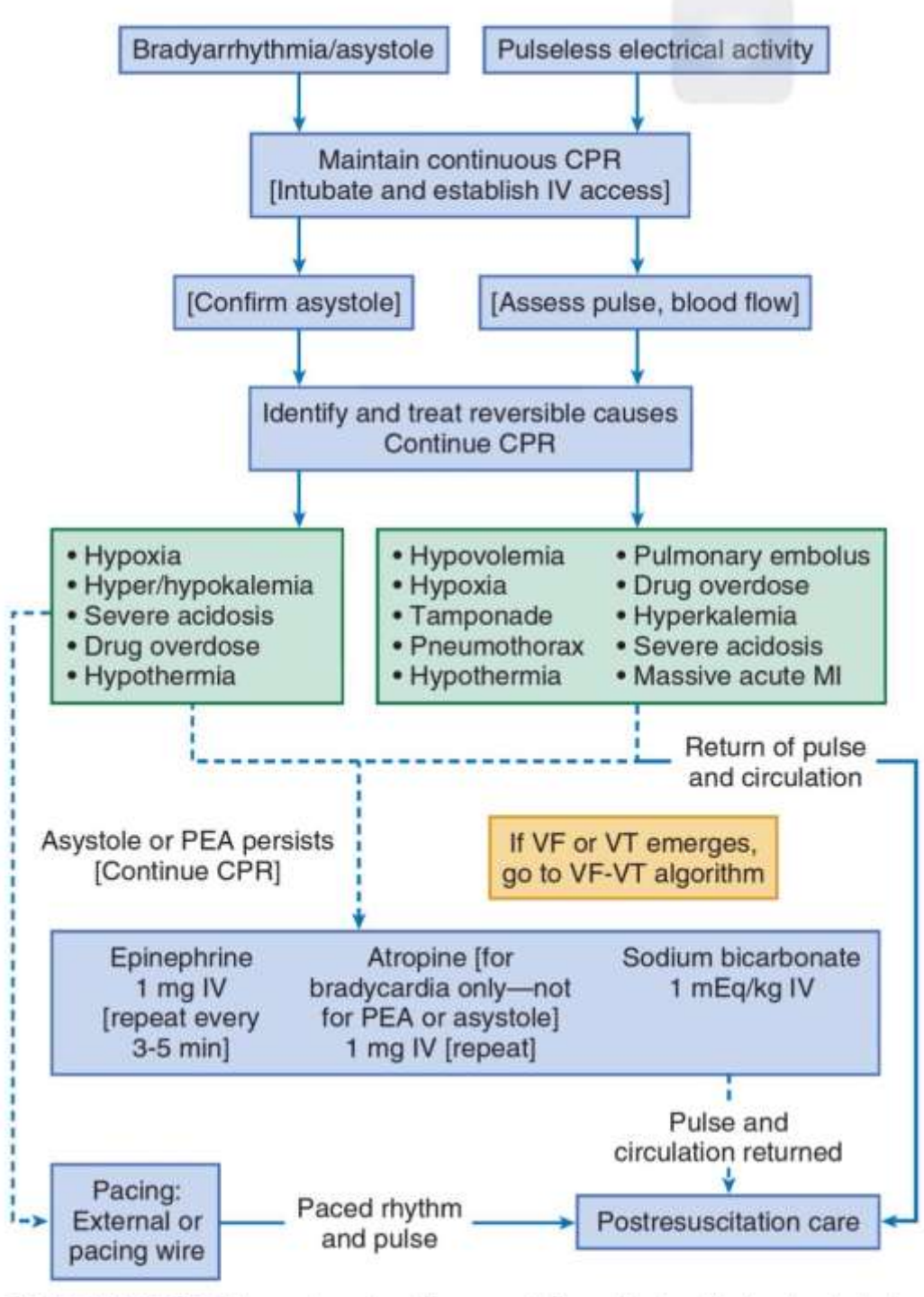


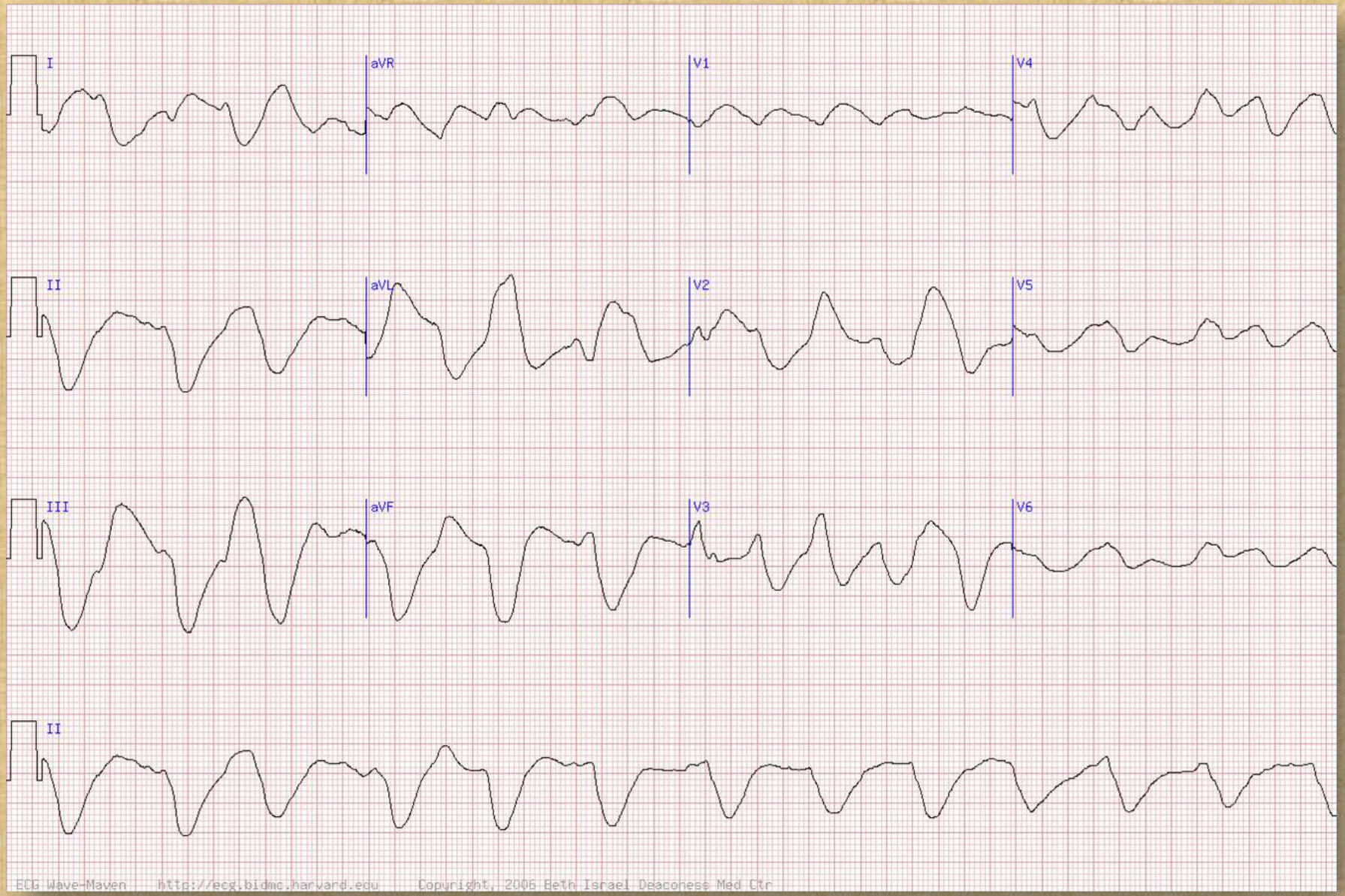
FIGURE 39-18 Advanced cardiac life support for patients with bradyarrhythmicasystolic arrest and PEA. A patient in any of these states should have CPR continued and be intubated, with intravenous access established, before pharmacologic treatment. The initial activity is to confirm persisting asystole or attempt to assess blood flow in patients thought to have PEA. An immediate attempt should be made to identify and treat reversible or treatable causes of these forms of cardiac arrest. Epinephrine is generally administered first, and atropine or bicarbonate, or both, may be administered subsequently. An attempt to pace the heart with an external device or an intracardiac pacing catheter is advisable although not usually successful, except for certain reversible bradyarrhythmias. MI = myocardial infarction. (From 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science. Circulation 122[Suppl 3]:S640, 2010.)

ACLS: De-emphasis of Devices, Drugs and other Distracters

- Focus on high-quality CPR and defibrillation
- Atropine no longer recommended for routine use in management of PEA/asystole.
- Chronotropic drug infusions now recommended as alternative to pacing in symptomatic and unstable bradycardia.
- Adenosine recommended as safe and potentially effective for treatment and diagnosis in initial management of undifferentiated regular monomorphic wide-complex tachycardia.

ACLS: Medications

- Atropine: deleted from pulseless arrest algorithm
- Epinephrine: dose, interval unchanged
- Vasopressin: deleted
- Amiodarone: dose, indications unchanged
- Lidocaine: dose, indications unchanged
- Sodium Bicarbonate: Routine use not recommended (Class III, LOE B).
- Calcium: Routine administration for treatment of cardiac arrest not recommended (Class III, LOE B).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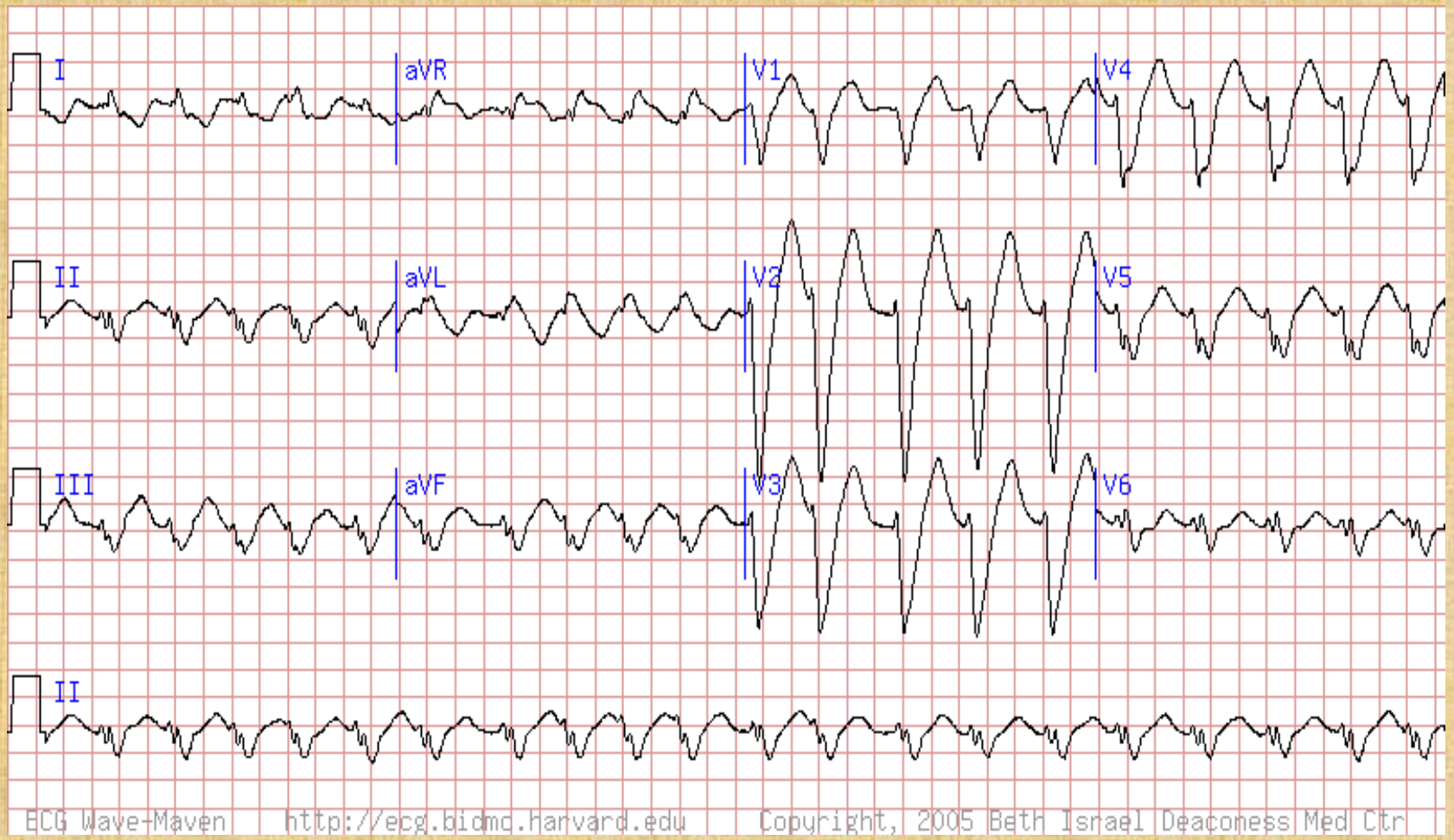
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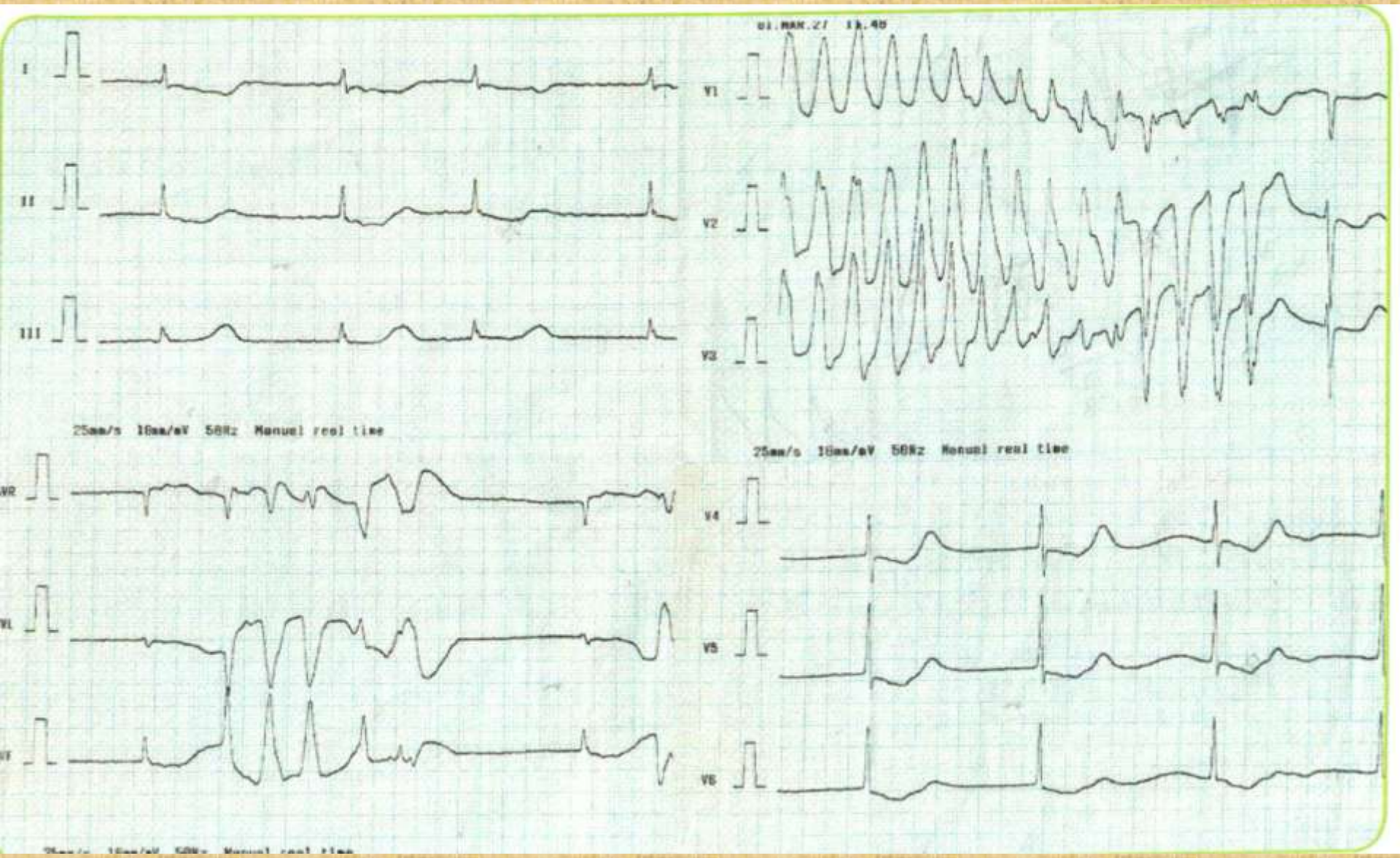
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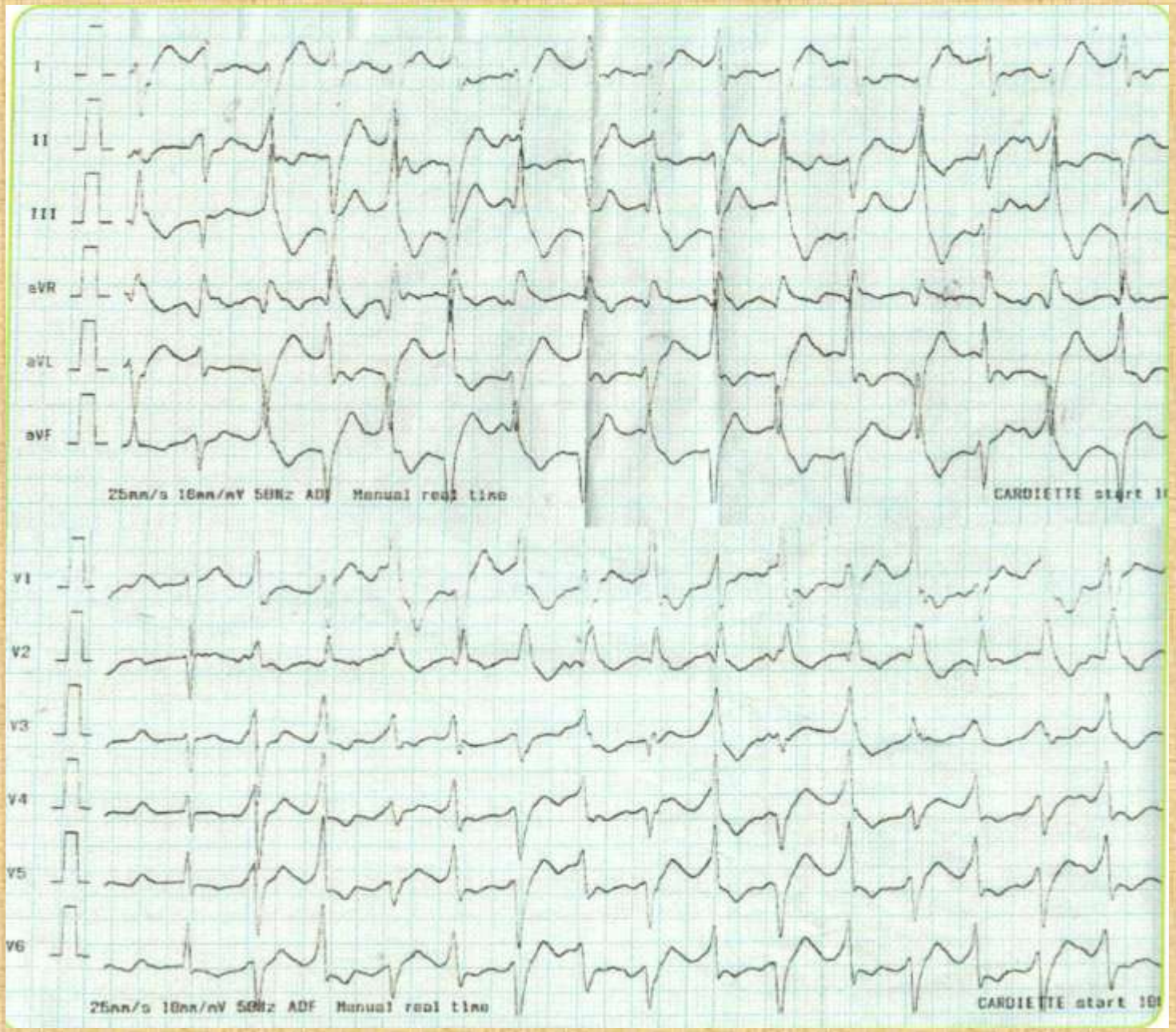
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Thanks For Your Attention