Acute Cardiology
Cardiac Emergencies

-
- Cardiologist
- Faculty members of
  sciences

Acute Cardiology:
Cardiac Emergencies

- Definition:
-
-

Clinical endpoints:

Physiopathology:
A- Low cardiac output and systemic h
B- Severe myocardial ischemia a

Acute Cardiology: Symptoms

Diagnosis of cardiac emergencies:

Main symptoms:

6- Sy[illegible]esyncope
7- Sudden death

Acute Cardiology
Physical examination

CLİNİCAL KEY POİNTS OF PHYSİCAL EXAMS:

- History.

1. Blood pressure:
2. Peripheral pulses: ic, arryrthmic.
3. Signs of systemic hypoperfusion: Consciousness, ski
   , urinary outp
4. General posture of the patient: In
   supine position, pale, swea
5. Killip class. (I-IV).

Chest Pain

Classification of myocardial ischemi(

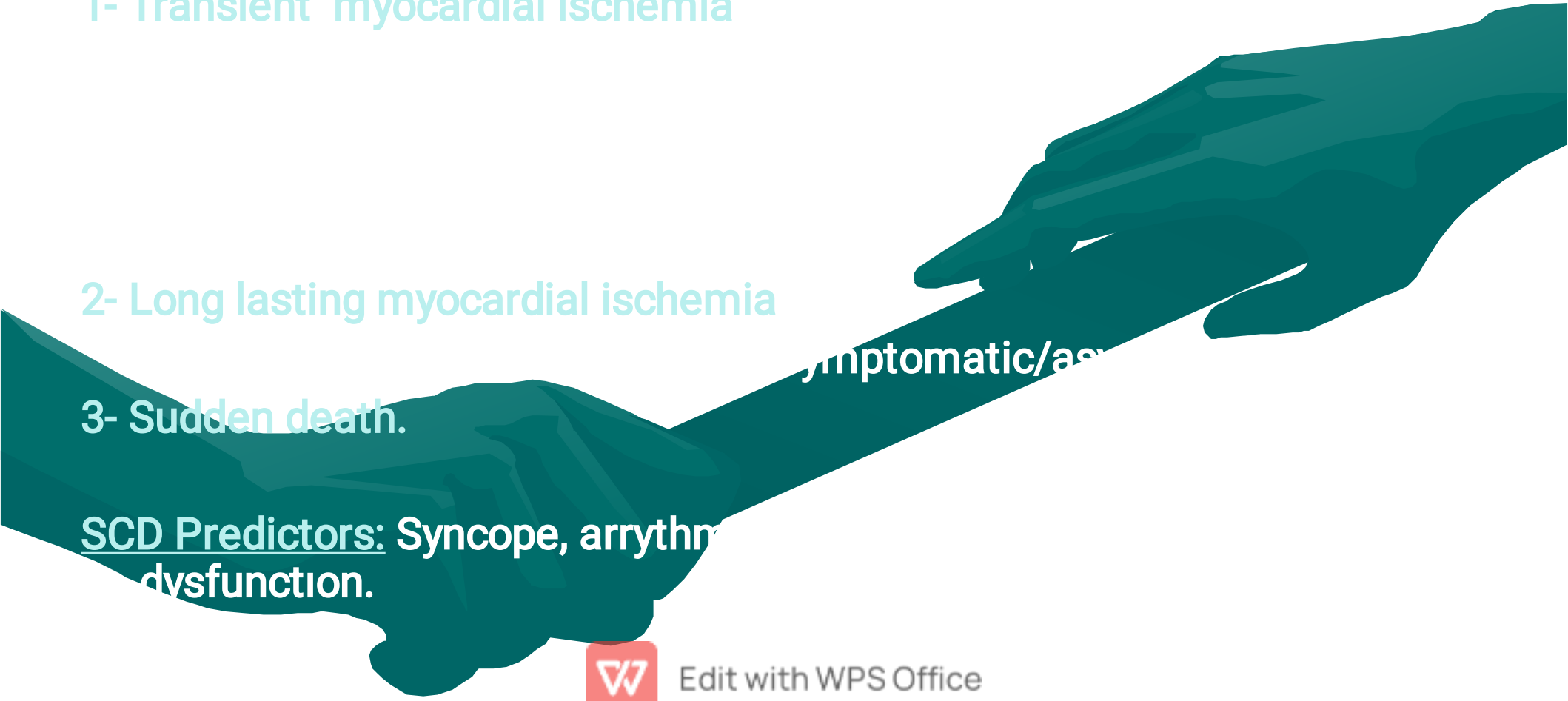
1- Transient myocardial ischemia

2- Long lasting myocardial ischemia

ymptomatic/as

3- Sudden death.

SCD Predictors: Syncope, arrythm
dysfunction.



Main Causes of Chest Discomfort and Pain:

A- Cardiac:

-
-
-
-
-
-

B- Anginal Equivalents:

-
-
- Shoulder or arm discomfort, particularly
  forearm and hand.
- Epigastric discomfort.
- Back (interscapular) discom

C- Noncardiac Causes of Chest Pain:

-
-
-
-
-
-
-
-
- Pul[illegible]ypertension.

Characterization of chest pain Probability of MI, ischemia:

TABLE 50-2 Value of Elements of the Chest Pain History for the Diagnosis of Acute Coronary Syndrome

PAIN DESCRIPTOR	POSITIVE LIKELIHOOD RATIO (95% CI)
Increased Likelihood of AMI	
Radiation to the right arm or shoulder	4.7 (1.9-12.0)
Radiation to both arms or shoulders	4.1 (2.5-6.5)
Associated with exertion	2.4 (1.5-3.8)
Radiation to the left arm	2.3 (1.7-3.1)
Associated with diaphoresis	2.0 (1.9-2.2)
Associated with nausea or vomiting	1.9 (1.7-2.3)
Worse than previous angina or similar to previous MI	1.8 (1.6-2.0)
Described as pressure	1.3 (1.2-1.5)
Decreased Likelihood of AMI	
Described as pleuritic	0.2 (0.1-0.3)
Described as positional	0.3 (0.2-0.5)
Described as sharp	0.3 (0.2-0.5)
Reproducible with palpation	0.3 (0.2-0.4)
Inframammary location	0.8 (0.7-0.9)
Not associated with exertion	0.8 (0.6-0.9)

AMI = acute myocardial infarction; CI = confidence interval.

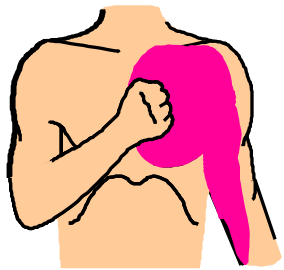
Modified from Swap CJ, Nagurney JT: Value and limitations of chest pain history in the evaluation of patients with suspected acute coronary syndromes. JAMA 294:2623, 2005.

Diagnosis: “Non-Anginal Pain”:

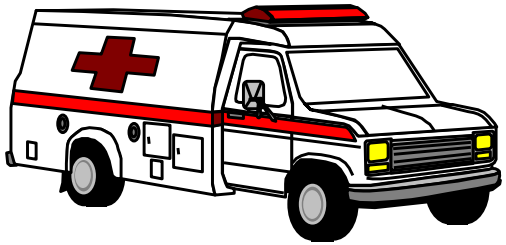
- Major characteristics of “Non-Anginal Pain”:

1.
2. bdominal
   mandibular region.
3. ip.
4. lpation of pecteral
   s.
5. sts over
6. Instantenous pain.

Options for Transport of Patients With STEMI and Initial Reperfusion Treatment



Call 9-1-1
Call fast



Onset of symptoms of STEMI

9-1-1 EMS Dispatch

EMS on-scene
- Encourage 12-lead ECGs.
- Consider prehospital fibrinolytic if capable and EMS-to-needle within 30 min.

EMS Triage Plan

Hospital fibrinolysis: Door-to-Needle within 30 min.

Not PCI capable

Inter-Hospital Transfer

PCI capable

GOALS

5 min. | 8 min. | EMS Transport

Patient | EMS

Prehospital fibrinolysis
EMS-to-needle
within 30 min.

EMS transport
EMS-to-balloon within 90 min.
Patient self-transport
Hospital door-to-balloon
within 90 min.

Dispatch
1 min.

Golden Hour = first 60 min.

Total ischemic time: within 120 min.

http://content.onlinejacc.org/

cgi/content/full/j.jacc.2007.10.001

Acute and new onset MI (AMİ):

Key of the definition: to be present of myocardial necrosis evidences on the persistan myocardial iashemia milieu.

Diagnosis: At least one of the criteria below:

1. Elevated Biomarkers.

- - Troponin: Typical elevation and then delayed typical fall.
- - CK/CK-MB: Rapid elevation and fall.

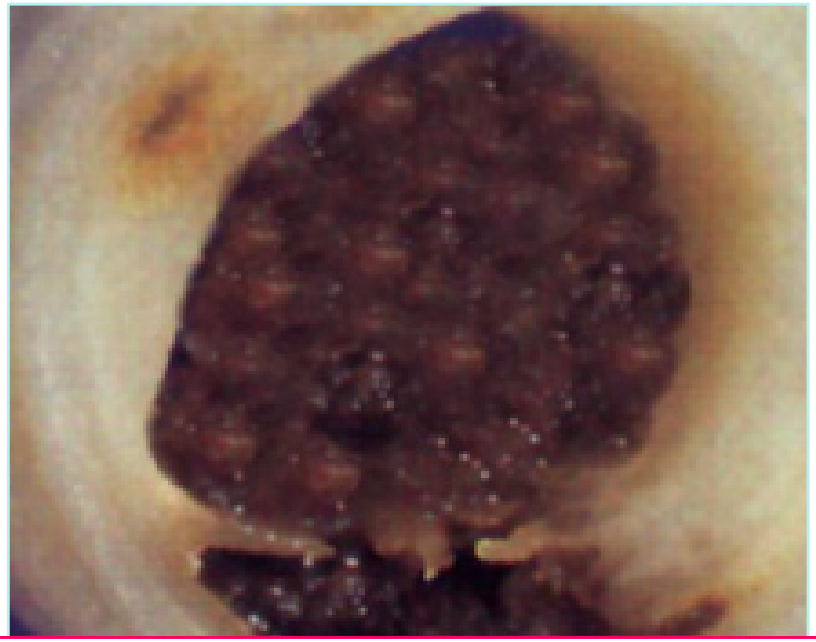
2. Biomarker elevation in the prescence of two of the criteria below:

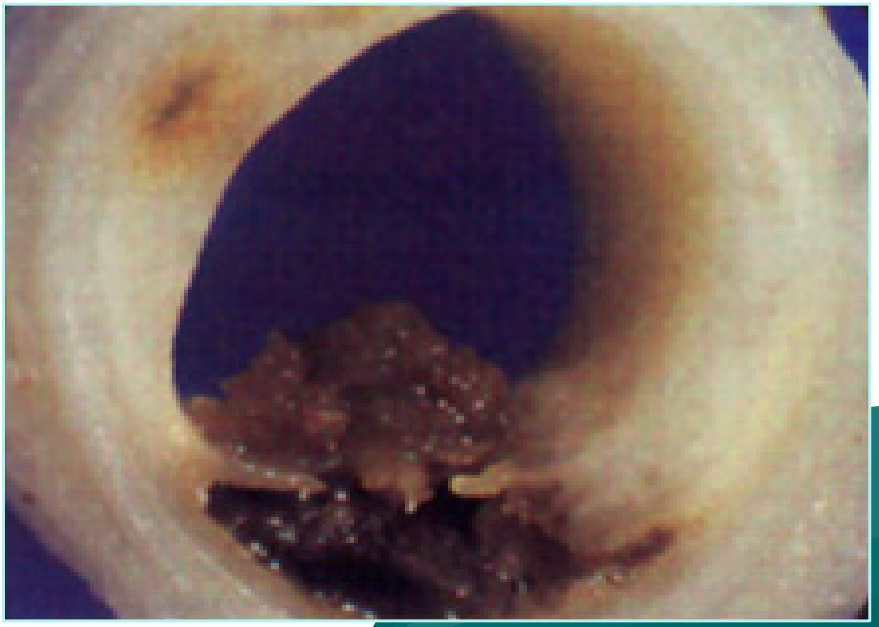
a- ECG and ischemic symptoms.

b- Old Mİ: Q waves on ECG.

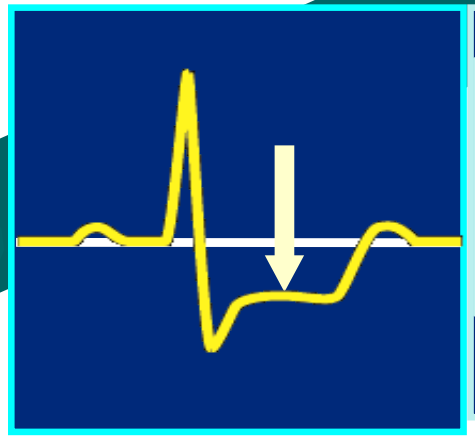
c- ECG findings of acute ischemia: ST- T wave changes. (ST elevation, depression, new LBBB).

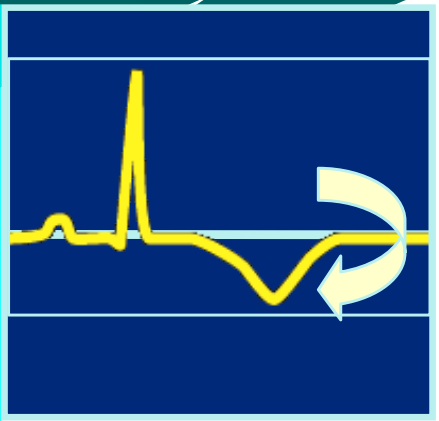
d-





Patology: Total oclusive/Nonocclusive thrombus





necrosi

Normal

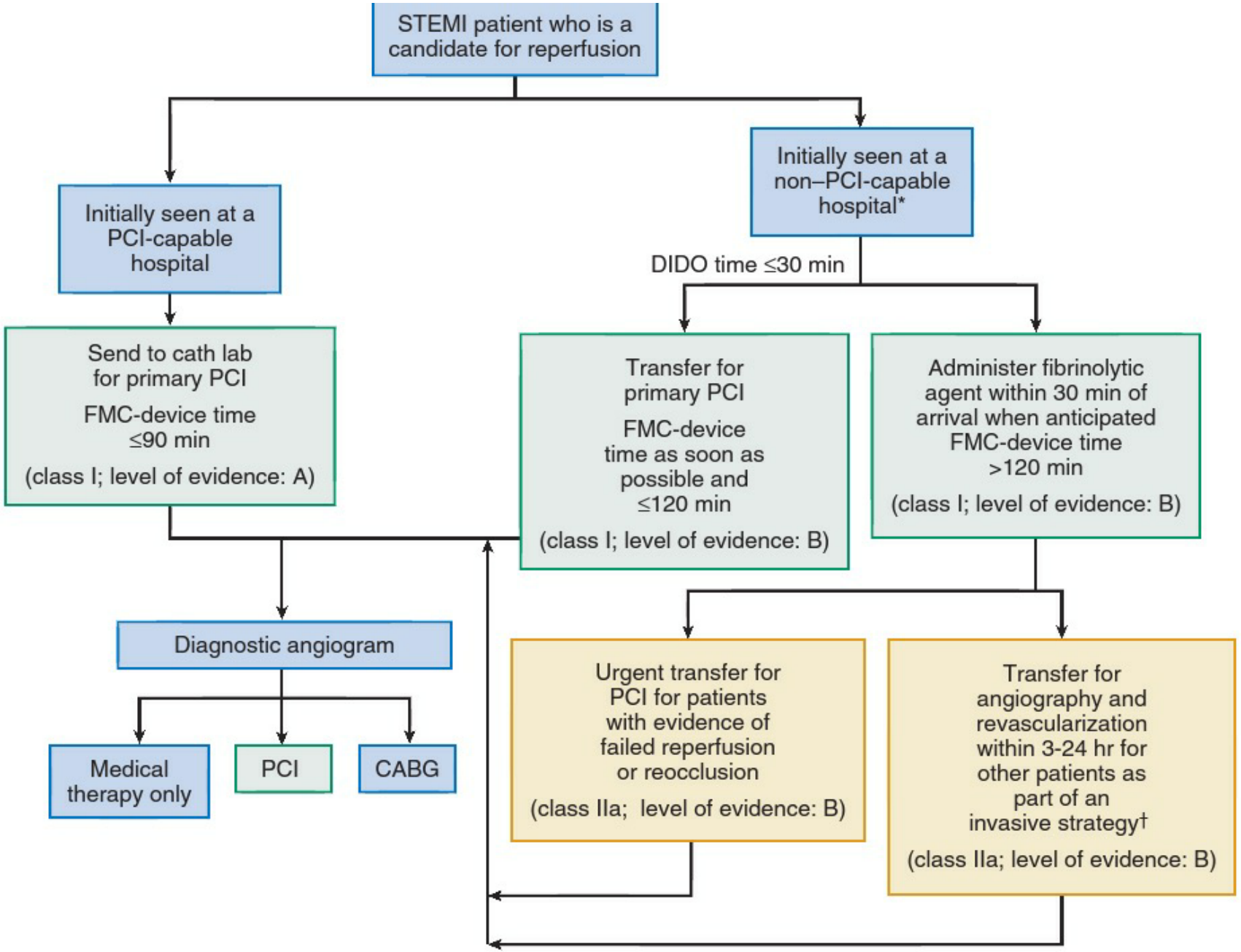


FIGURE 52G-1 Reperfusion therapy for patients with STEMI. The *bold arrows* and *boxes* are the preferred strategies. Performance of PCI is dictated by an anatomical appropriate culprit stenosis. *Patients with cardiogenic shock or severe heart failure initially seen at a non–PCI-capable hospital should be transferred for cardiac cathete ization and revascularization as soon as possible, irrespective of the delay in time after the onset of MI (class I; Level of evidence: B). †Angiography and revascularizatio should not be performed within the first 2 to 3 hours after the administration of fibrinolytic therapy. DIDO = door in–door out. *(Modified from O'Gara PT, Kushner FG Ascheim DD, et al: 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: A report of the American College of Cardiology Foundation American Heart Association Task Force on Practice Guidelines. Circulation 127:e362, 2013.)*

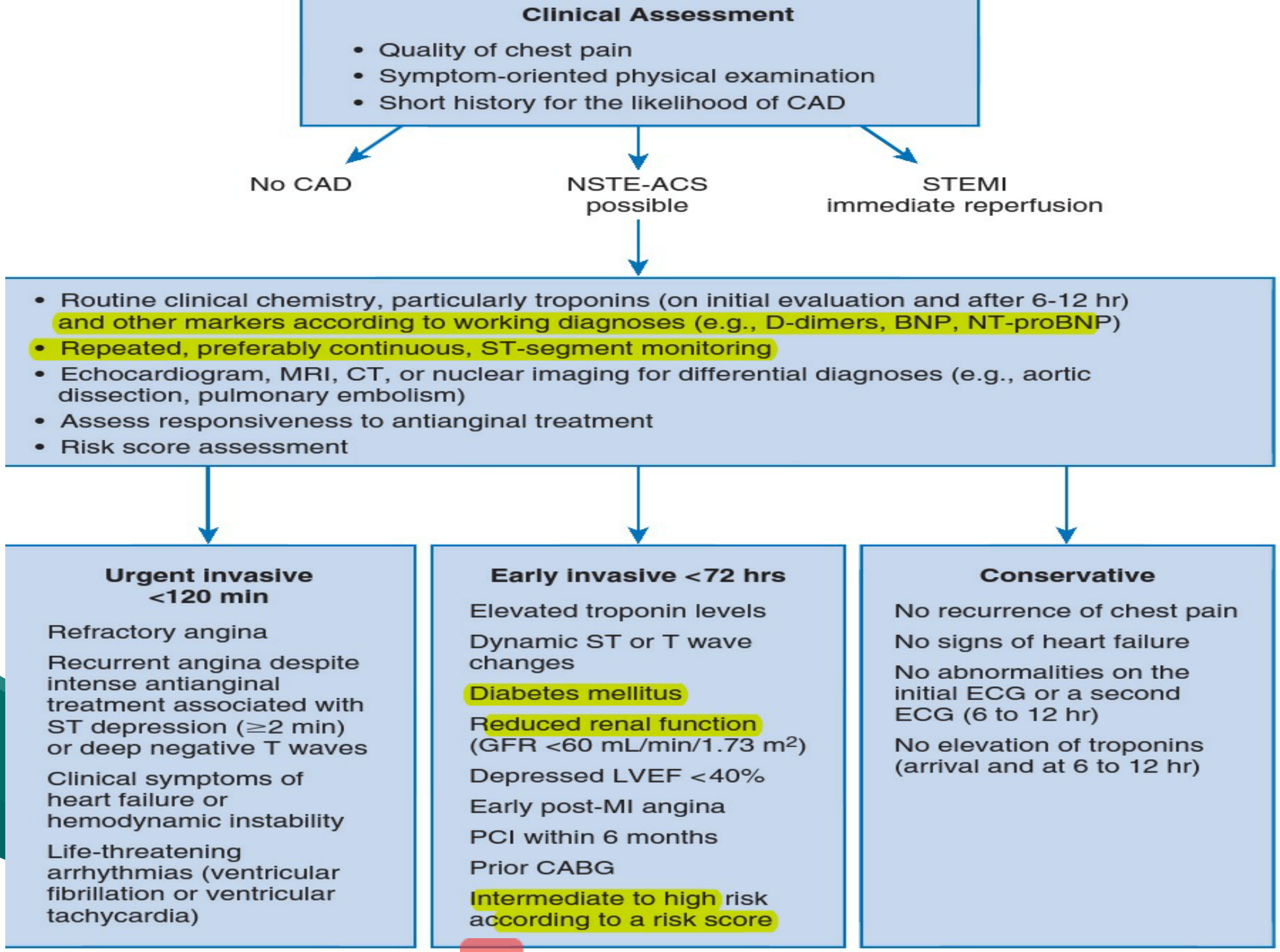


FIGURE 53-8 Decision-making algorithm for the management of NSTE-ACS. CABG = coronary artery bypass grafting; GFR = glomerular filtration rate; LVEF = left ventricular ejection fraction. *(From Bassand JP, Hamm CW: Diagnosis and treatment of non–ST-segment elevation acute coronary syndromes: European Society of Cardiology guidelines. Eur Heart J 32:369, 2011.)*

Acute Dyspnea:

- (A) Cardiac dyspnea:
- (B) Pulmonary causes:
- 2- Pneumonia
- 3- Pulmonary embolus, fat embolism
- 4- Acute Respiratory Distress

Heart Failure

Diagnosis :

- A-
- B-
- C-

Ancillary Markers:

Management of Heart Failure

STEP- 1: Diagnosis:

STEP- 2: Clinical Profile:

STEP- 3: Ethiology. -Advan uation.

STEP- 4: Precipitating Factors: Anemia, İn
ischemia, HTA crisis

Tiroid disorder, drugs (NSAİ, Ste

STEP- 5,6: Evaluation of prognosis.

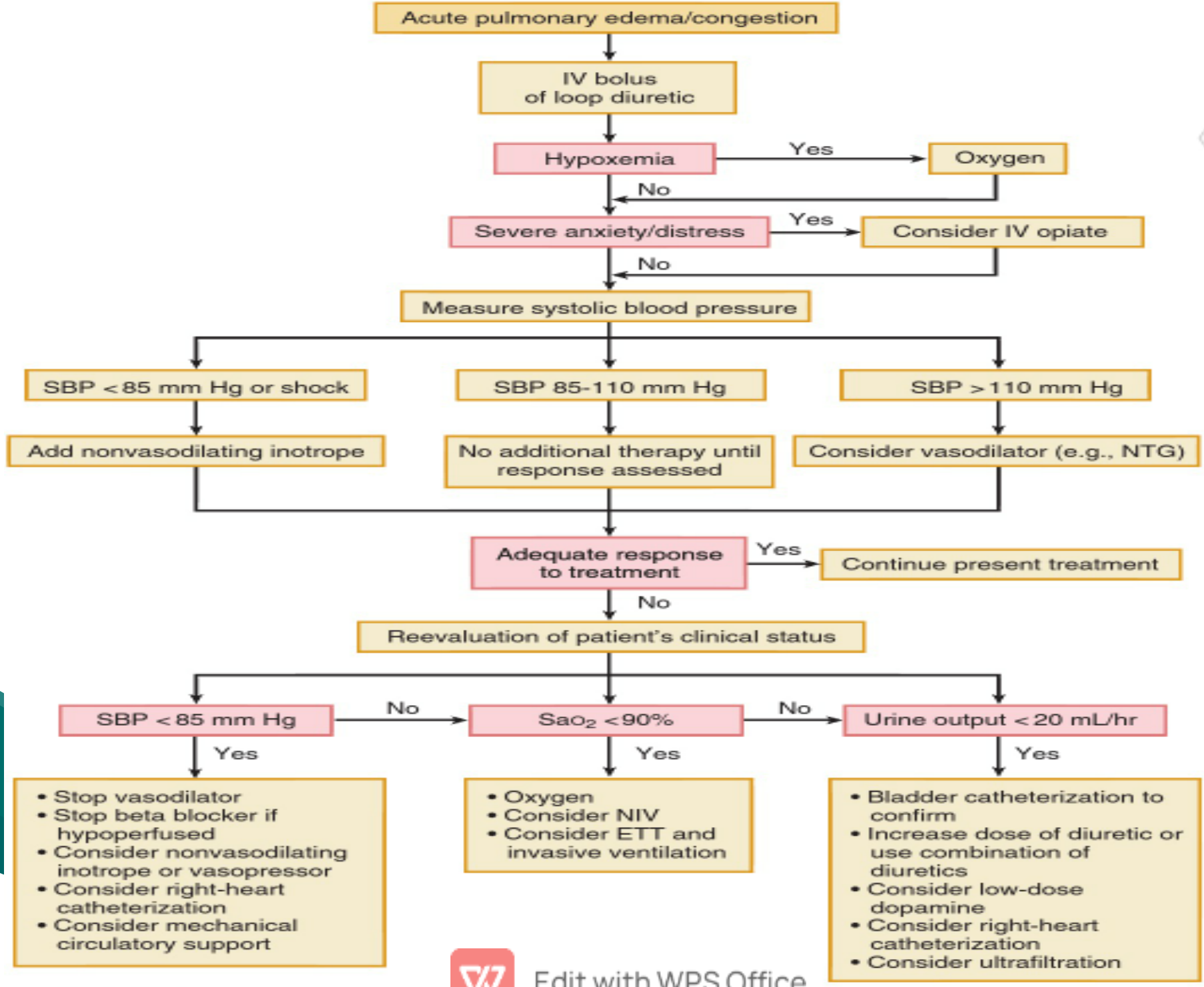


FIGURE 24-7 Algorithm for management of patients admitted with AHF and pulmonary edema/congestion. ETT = endotracheal tube; IV = intravenous; NTG = nitroglycerin. *(Modified from McMurray JJ, Adamopoulos S, Anker SD, et al: ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collabora-*

Sudden Cardiac Death: SCD

Definition:

(1)

(2) ...e initiation of the ...th is not known.

Key for definition:

(a) ...nature ...ent.

(b) Sudden and unexpected.

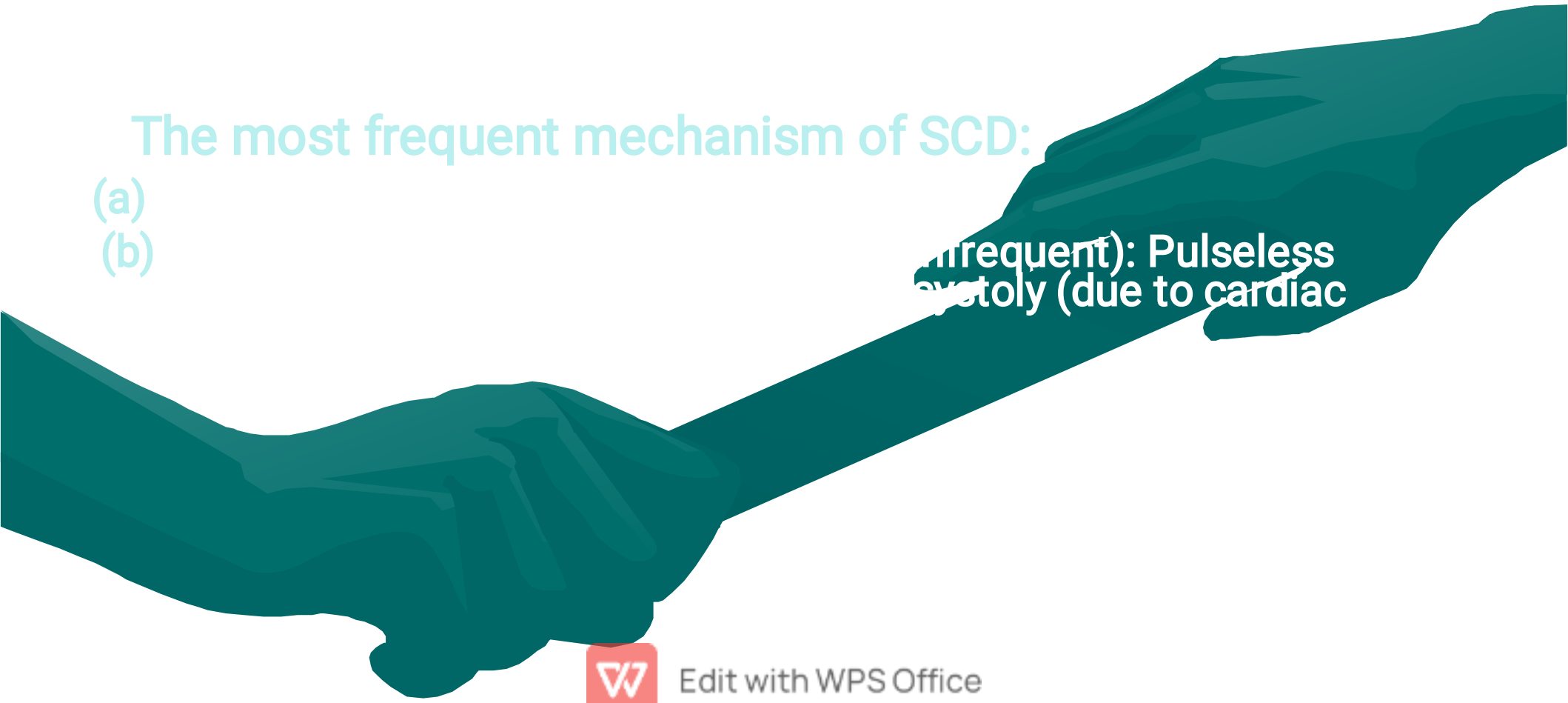
CARDIAC ARREST

Definition:

The most frequent mechanism of SCD:

(a)

(b)



Cardiovascular Causes of SCD:

1-

2-

3-

4-

5-

Electrical Causes of SCD:

(a)
(b)
(c)
(d)
(e)

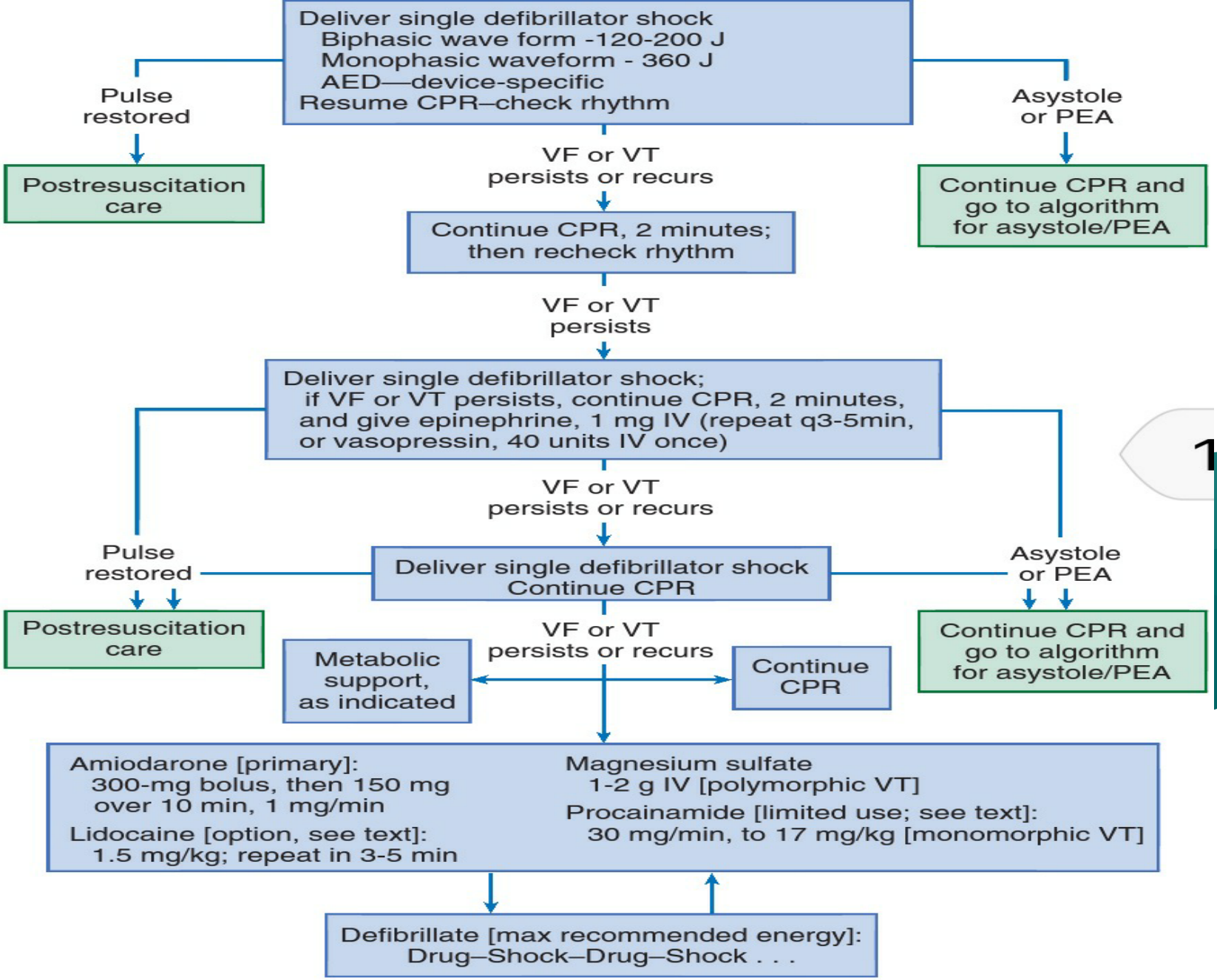


FIGURE 39-17 Advanced life support for VF and pulseless VT. If initial defibrillation fails, the patient should be intubated and intravenous access established immediately while CPR is continued. Epinephrine, 1 mg intravenously, should be administered and may be repeated several times with additional 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.)*

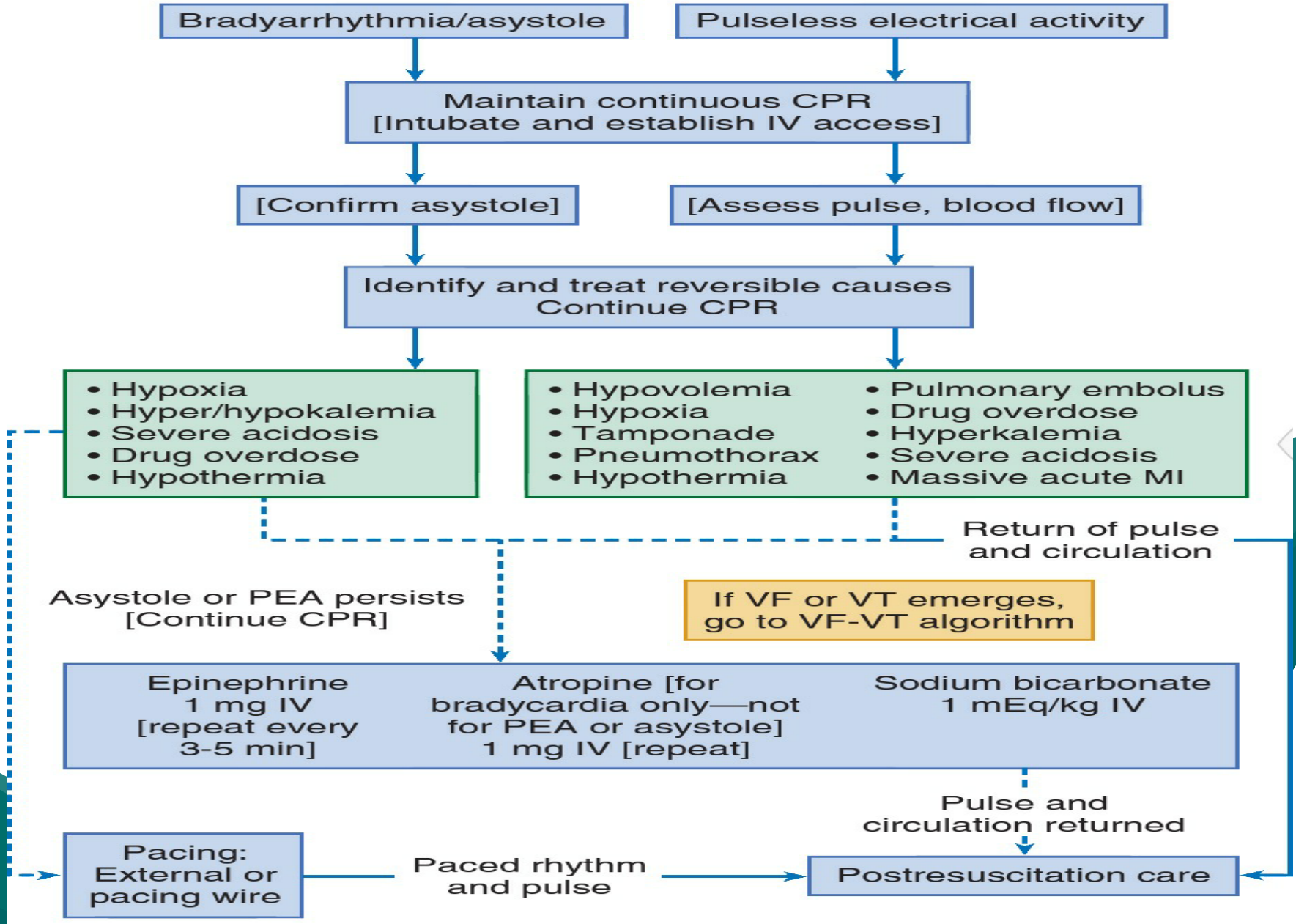


FIGURE 39-18 Advanced cardiac life support for patients with bradyarrhythmic-asystolic arrest and PEA. A patient in any of these states should have CPR continued and be intubated, with intravenous access established, before pharmacologic treatment. The initial activity is to confirm persisting asystole 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.)*

Neurocardiogenic syncope

Definition:

ABC of syncope:

A-

onus that is associated
neously.

B-

bruptly, then s

C- Mechanism: Sudden / short interval of

Neurocardiogenic Syncope: Cardiac Causes.

- Anatomic causes:
- Aortic stenosis
- Hypertrophic CMP
- Miocardial ischemia /AMI
- Aortic dissection

- Atrial mixoma

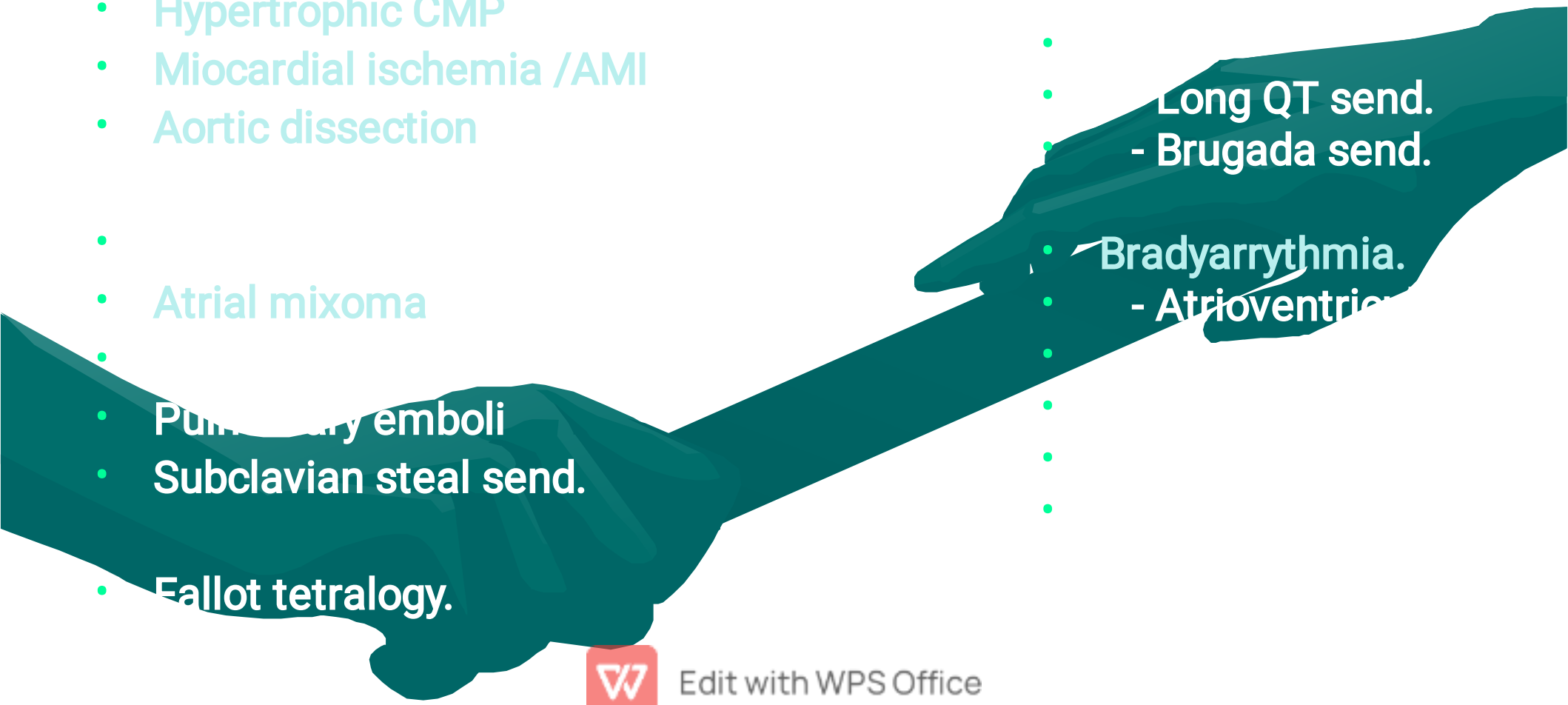
- Pul[illegible]ary emboli
- Subclavian steal send.

- Fallot tetralogy.

- Arrythmic causes:
- Tachyarrythmia

- Long QT send.
- - Brugada send.

- Bradyarrythmia.
- - Atrioventric[illegible]



Neurocardiogenic syncope:
Absolute diagnostic criteria.

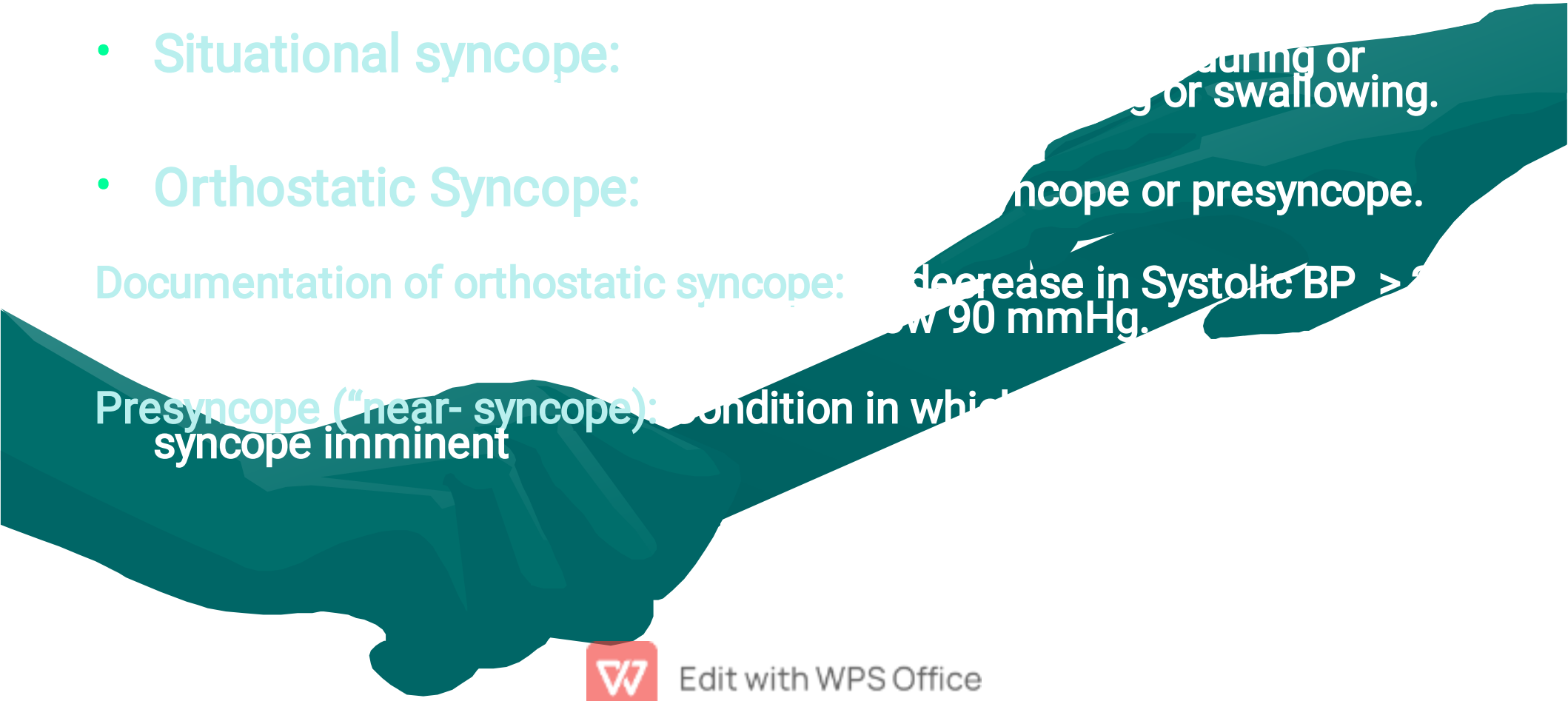
- Vasovagal Syncope.

- Situational syncope:

- Orthostatic Syncope:

Documentation of orthostatic syncope:

Presyncope (“near- syncope):
syncope imminent



Cardiac ischemia related syncope

Arrhytmia syncope was diagnosed by ECG when there is:

Cardiogenic Shock

- Definition:
- (1)
- (2)

- Characteristics:
-
-
-

- Clinical findings and diagnosis:
- (a) Pump failure, and clinical signs a
- (b) SBP: <90mmHg, urine outp
- (c) Exclusion of other cause
- (d) Clinical shock. (loss of

HYPERTENSIVE CRISIS

- Hypertensive Emergency - Hypertensive Crisis:
-
- Basic Principle:
- (a)
-
- Principle of management:
- Arterial BP must fall within

TABLE 44-13 Recommended Treatment of Hypertensive Emergencies by End-Organ Involved

TYPE OF EMERGENCY	TIMELINE, TARGET BLOOD PRESSURE	FIRST-LINE THERAPY	ALTERNATIVE THERAPY
Hypertensive crisis with retinopathy, microangiopathy, or acute renal insufficiency	Several hours, MAP –20% to –25%	Labetalol	Nitroprusside Nicardipine Urapidil
Hypertensive encephalopathy	Immediate, MAP –20% to –25%	Labetalol	Nicardipine Nitroprusside
Acute aortic dissection	Immediate, SBP < 110 mm Hg	Nitroprusside + metoprolol	Labetalol
Acute pulmonary edema	Immediate, MAP 60 to 100 mm Hg	Nitroprusside with loop diuretic	Nitroglycerin Urapidil with loop diuretic
Acute coronary syndrome	Immediate, MAP 60 to 100 mm Hg	Nitroglycerin	Labetalol
Acute ischemic stroke and BP >220/120 mm Hg	1 hour, MAP –15%	Labetalol	Nicardipine Nitroprusside
Cerebral hemorrhage and SBP >180 mm Hg or MAP >130 mm Hg	1 hour, SBP < 180 mm Hg and MAP <130 mm Hg	Labetalol	Nicardipine Nitroprusside
Acute ischemic stroke with indication for thrombolytic therapy and BP >185/110 mm Hg	1 hour, MAP less than –15%	Labetalol	Nicardipine Nitroprusside
Cocaine/XTC intoxication	Several hours, SBP < 140 mm Hg	Phentolamine (after benzodiazepines)	Nitroprusside
Pheochromocytoma crisis	Immediate	Phentolamine	Nitroprusside Urapidil
Perioperative hypertension during or after CABG	Immediate	Nicardipine	Urapidil Nitroglycerin
During or after craniotomy	Immediate	Nicardipine	Labetalol
Severe preeclampsia/eclampsia	Immediate, BP < 160/105 mm Hg	Labetalol (plus $MgSO_4$ and oral antihypertensives)	Ketanserin Nicardipine

CABG = coronary artery bypass graft; MAP = mean arterial pressure; $MgSO_4$ = magnesium sulfate; XTC = Ecstasy.

Modified from van den Born BJ, Beutler JJ, Gaillard CA, et al: Dutch guideline for the management of hypertensive crisis—2010 revision. Neth J Med 69:248, 2011.

AORTIC DISSECTION

Signs of clinical diagnosis:

a-

b-

c-

Complications:

Ischemic neuropathy at lower extremities: Si

Gold standart of diagnosis: TEE, C
artography .

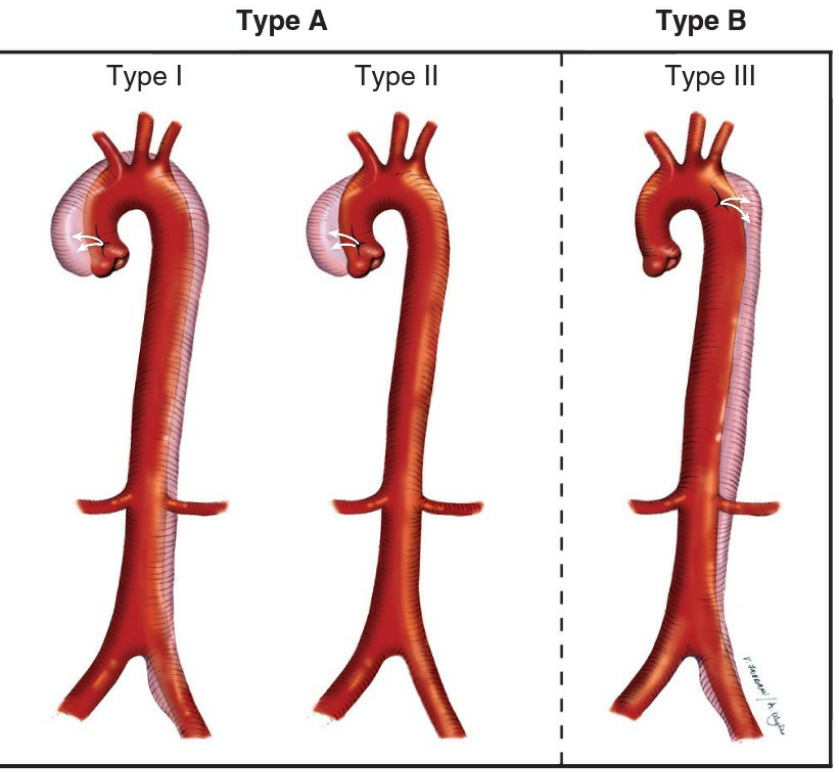


FIGURE 57-11 Classification schemes of acute aortic dissection.

TABLE 57-4 Classification Schemes of Acute Aortic Dissection

DeBakey Classification	
Type I	Originates in the ascending aorta and extends at least to the aortic arch and often to the descending aorta (and beyond)
Type II	Originates in the ascending aorta and confined to this segment
Type III	Originates in the descending aorta, usually just distal to the left subclavian artery, and extends distally
Stanford Classification	
Type A	Dissections involving the ascending aorta (with or without extension into the descending aorta)
Type B	Dissections not involving the ascending aorta

TABLE 57-6 Organ System Complications of Acute Aortic Dissection

Cardiovascular	Cardiac arrest Syncope Aortic regurgitation Congestive heart failure Coronary ischemia Myocardial infarction Cardiac tamponade Pericarditis
Pulmonary	Pleural effusion Hemothorax Hemoptysis (from an aortotracheal or bronchial fistula)
Renal	Acute renal failure Renovascular hypertension Renal ischemia or infarction
Neurologic	Stroke Transient ischemic attack Paraparesis or paraplegia Encephalopathy Coma Spinal cord syndrome Ischemic neuropathy
Gastrointestinal	Mesenteric ischemia or infarction Pancreatitis Hemorrhage (from an aortoenteric fistula
Peripheral vascular	Upper or lower extremity ischemia
Systemic	Fever

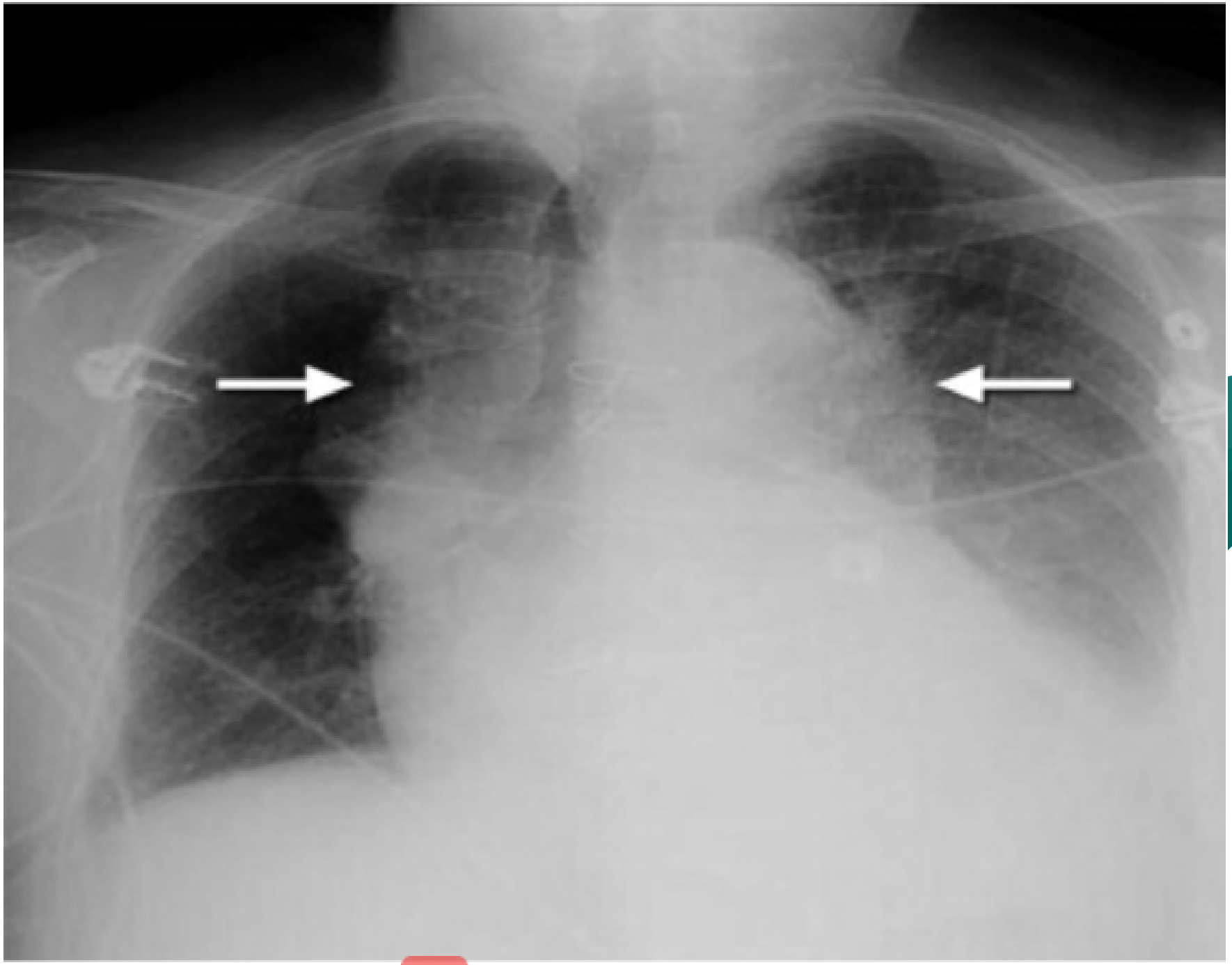


FIGURE e57-7

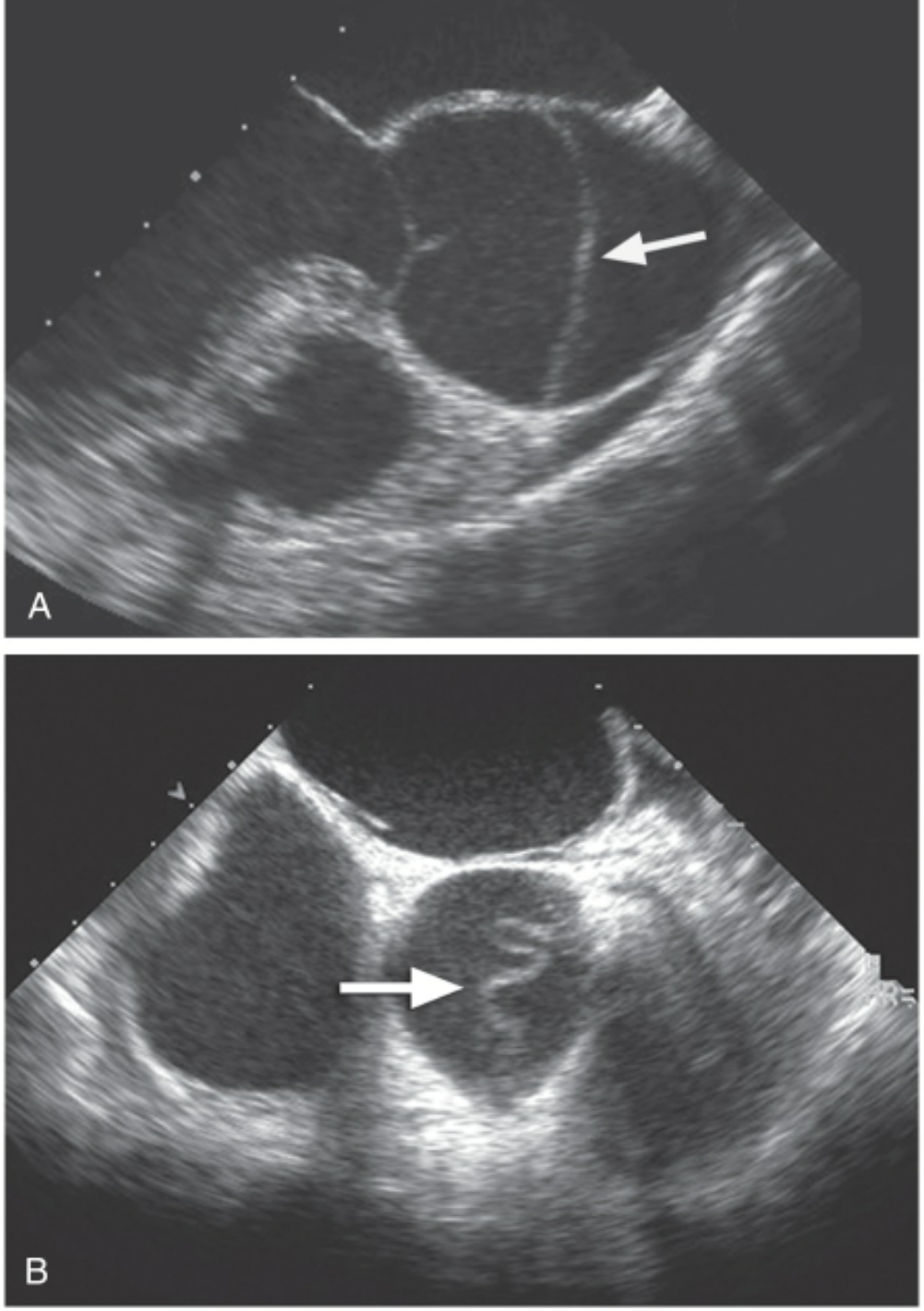


FIGURE 57-15 TEE showing acute aortic dissection. **A,** Acute type A dissection in a patient with MFS. The dissection flap *(arrow)* is present in the dilated aortic root. **B,** Serpiginous intimal flap *(arrow)* immediately distal to the aortic valve in a patient with a type A aortic dissection.

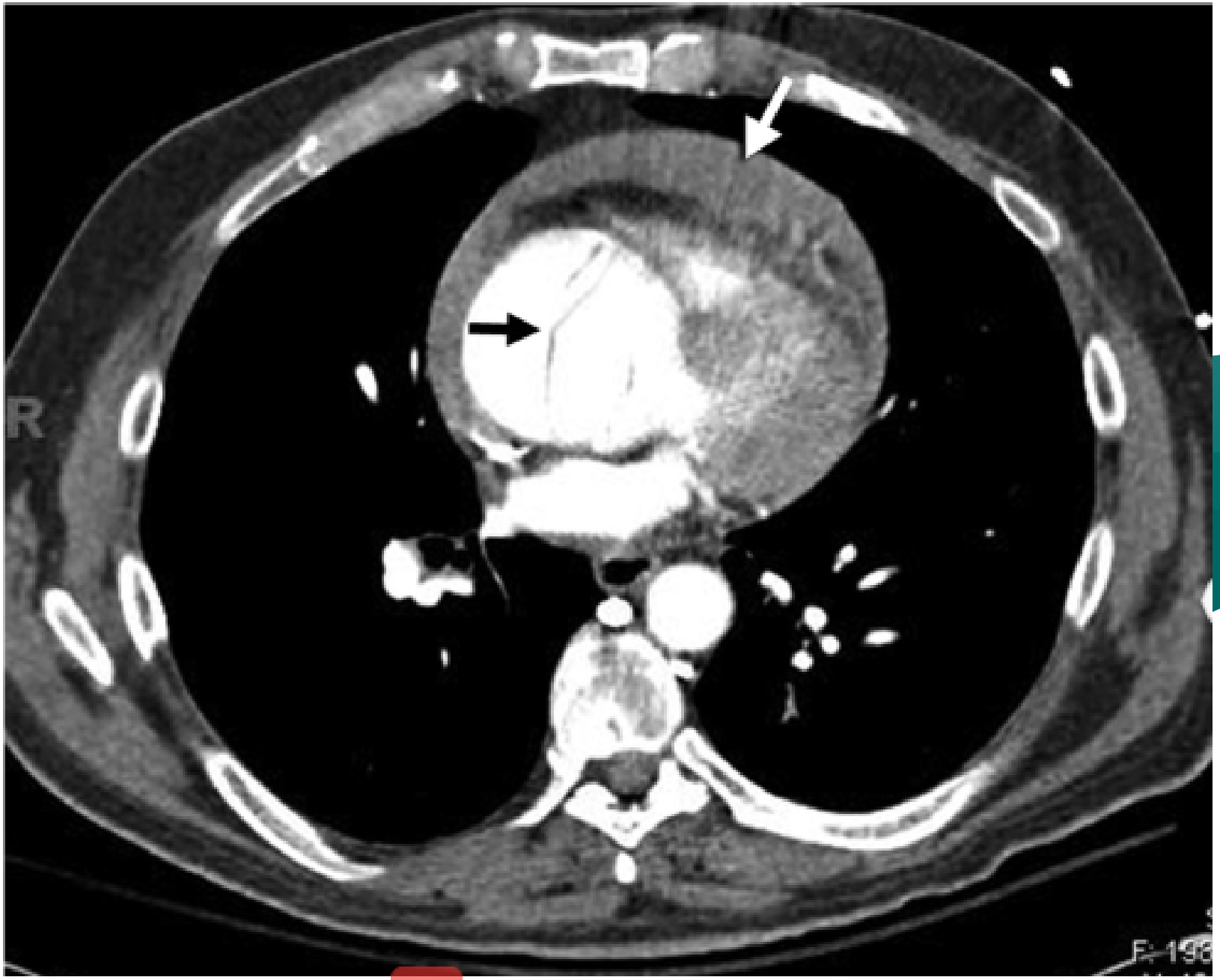
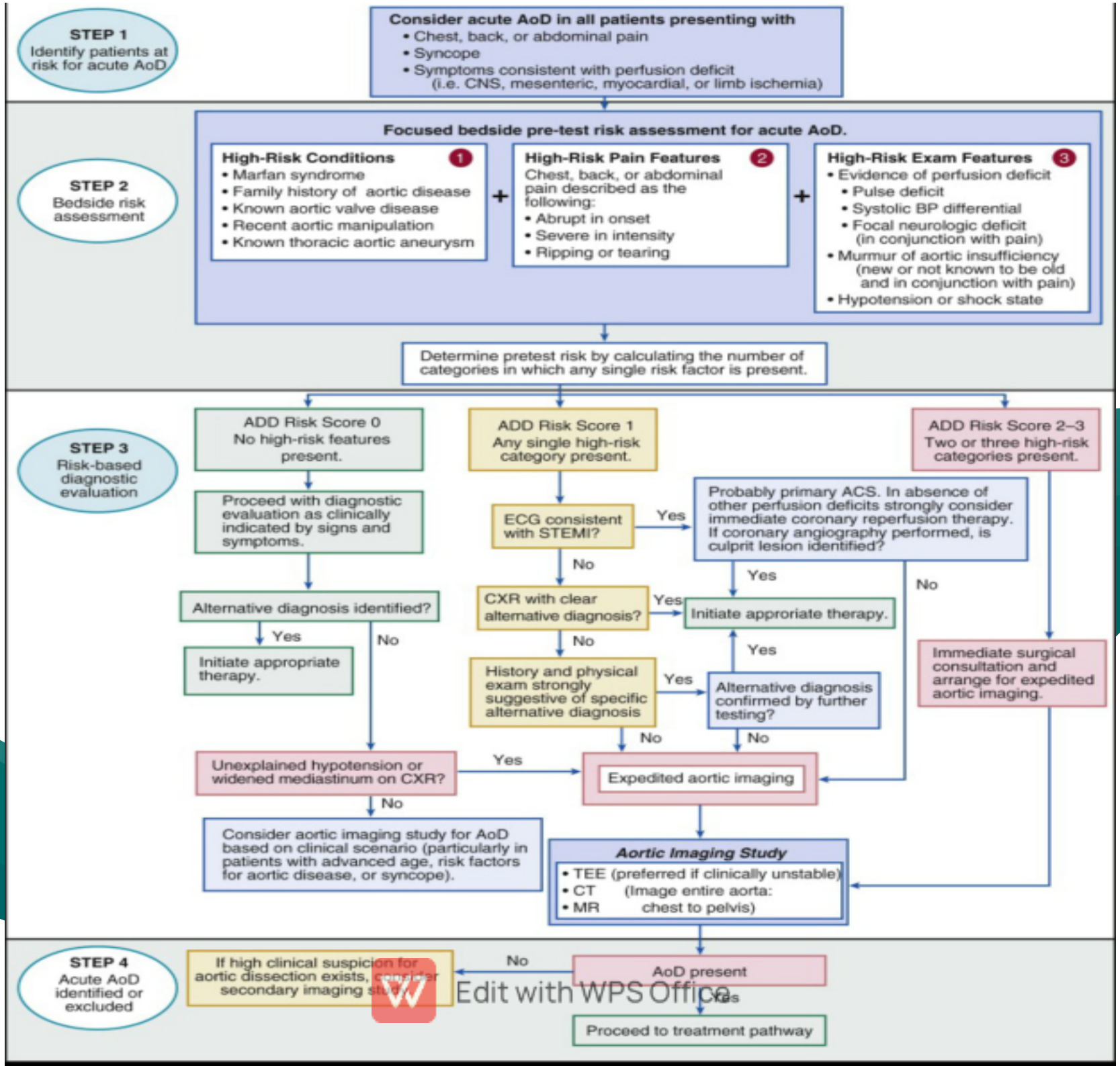
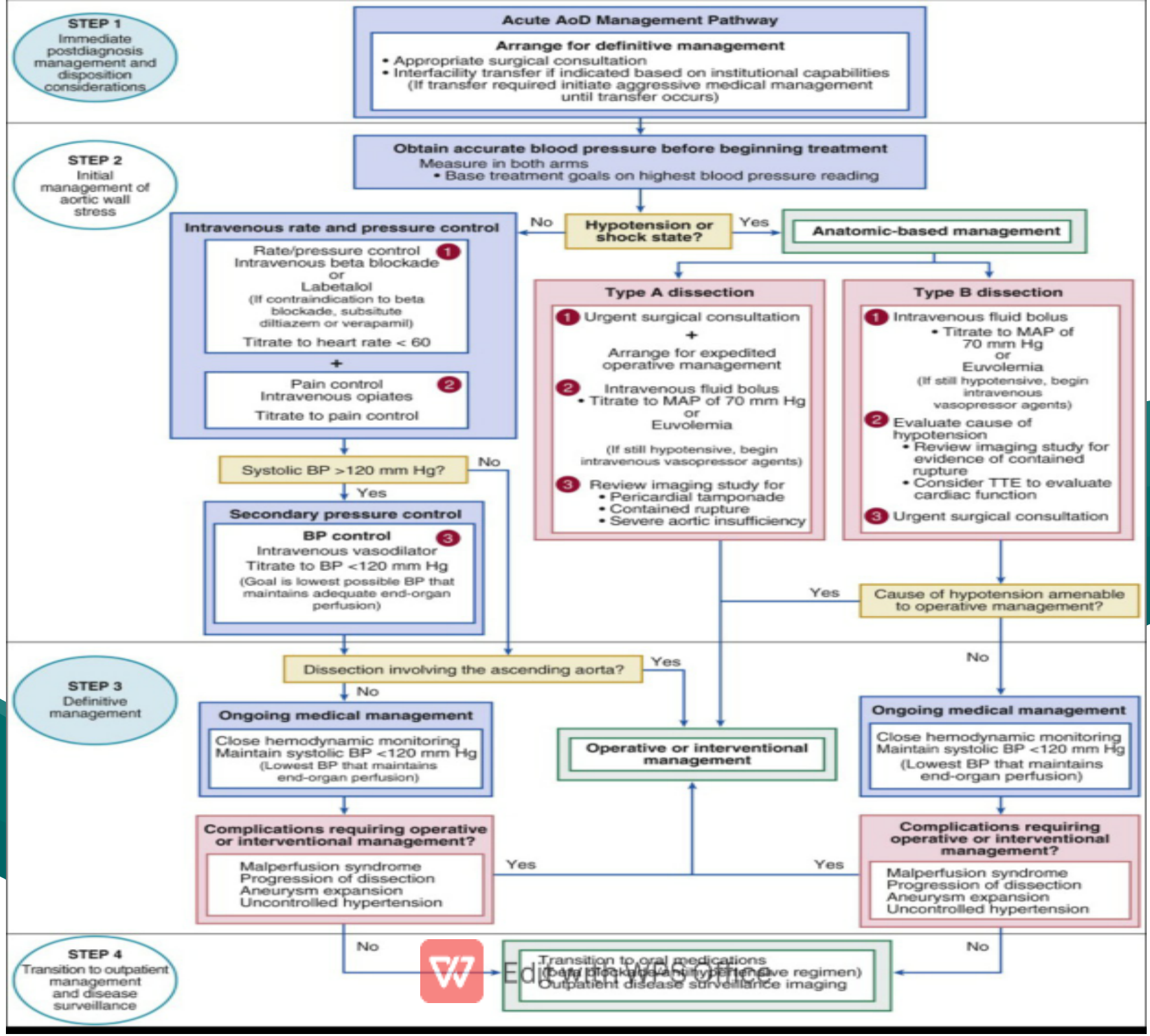


FIGURE e57-14 Contrast-enhanced CT scan of acute type A aortic dissection *(black arrow)* with associated hemopericardium *(white arrow)*.

TABLE e57-3 Diagnostic Information from Imaging of Acute Aortic Dissection

1. Establish the presence of aortic dissection or variant (aortic IMH, PAU)
2. Location of the dissection (type A, type B)
3. Anatomic features
    a. Extent of dissection
    b. Sites of entry and reentry
    c. False lumen patency, partial thrombosis, thrombosis
4. Complications of dissection
    a. Type A
        i. Aortic regurgitation
        ii. Coronary artery involvement
        iii. Pericardial effusion/hemopericardium
    b. Aortic rupture or leakage
    c. Branch vessel involvement
    d. Malperfusion




STEP 1
Immediate postdiagnosis management and disposition considerations
Acute AoD Management Pathway
Arrange for definitive management
• Appropriate surgical consultation
• Interfacility transfer if indicated based on institutional capabilities
(If transfer required initiate aggressive medical management until transfer occurs)
STEP 2
Initial management of aortic wall stress
Obtain accurate blood pressure before beginning treatment
Measure in both arms
• Base treatment goals on highest blood pressure reading
Intravenous rate and pressure control
Rate/pressure control
Intravenous beta blockade
or
Labetalol
(If contraindication to beta blockade, subsitute diltiazem or verapamil)
Titrate to heart rate < 60
+
Pain control
Intravenous opiates
Titrate to pain control
No
Hypotension or shock state?
Yes
Anatomic-based management
Type A dissection
1 Urgent surgical consultation
+
Arrange for expedited operative management
2 Intravenous fluid bolus
• Titrate to MAP of 70 mm Hg
or
Euvolemia
(If still hypotensive, begin intravenous vasopressor agents)
3 Review imaging study for
• Pericardial tamponade
• Contained rupture
• Severe aortic insufficiency
Type B dissection
1 Intravenous fluid bolus
• Titrate to MAP of 70 mm Hg
or
Euvolemia
(If still hypotensive, begin intravenous vasopressor agents)
2 Evaluate cause of hypotension
• Review imaging study for evidence of contained rupture
• Consider TTE to evaluate cardiac function
3 Urgent surgical consultation
Systolic BP >120 mm Hg?
No
Yes
Secondary pressure control
BP control
Intravenous vasodilator
Titrate to BP <120 mm Hg
(Goal is lowest possible BP that maintains adequate end-organ perfusion)
Yes
Cause of hypotension amenable to operative management?
No
STEP 3
Definitive management
Dissection involving the ascending aorta?
Yes
No
Ongoing medical management
Close hemodynamic monitoring
Maintain systolic BP <120 mm Hg
(Lowest BP that maintains end-organ perfusion)
Operative or interventional management
Ongoing medical management
Close hemodynamic monitoring
Maintain systolic BP <120 mm Hg
(Lowest BP that maintains end-organ perfusion)
Complications requiring operative or interventional management?
Malperfusion syndrome
Progression of dissection
Aneurysm expansion
Uncontrolled hypertension
Yes
Yes
Complications requiring operative or interventional management?
Malperfusion syndrome
Progression of dissection
Aneurysm expansion
Uncontrolled hypertension
No
No
STEP 4
Transition to outpatient management and disease surveillance
Transition to oral medications
(Beta blockade/antihypertensive regimen)
Outpatient disease surveillance imaging

Lethal arrythmias

1. SVTA: High risk WPW :

a-

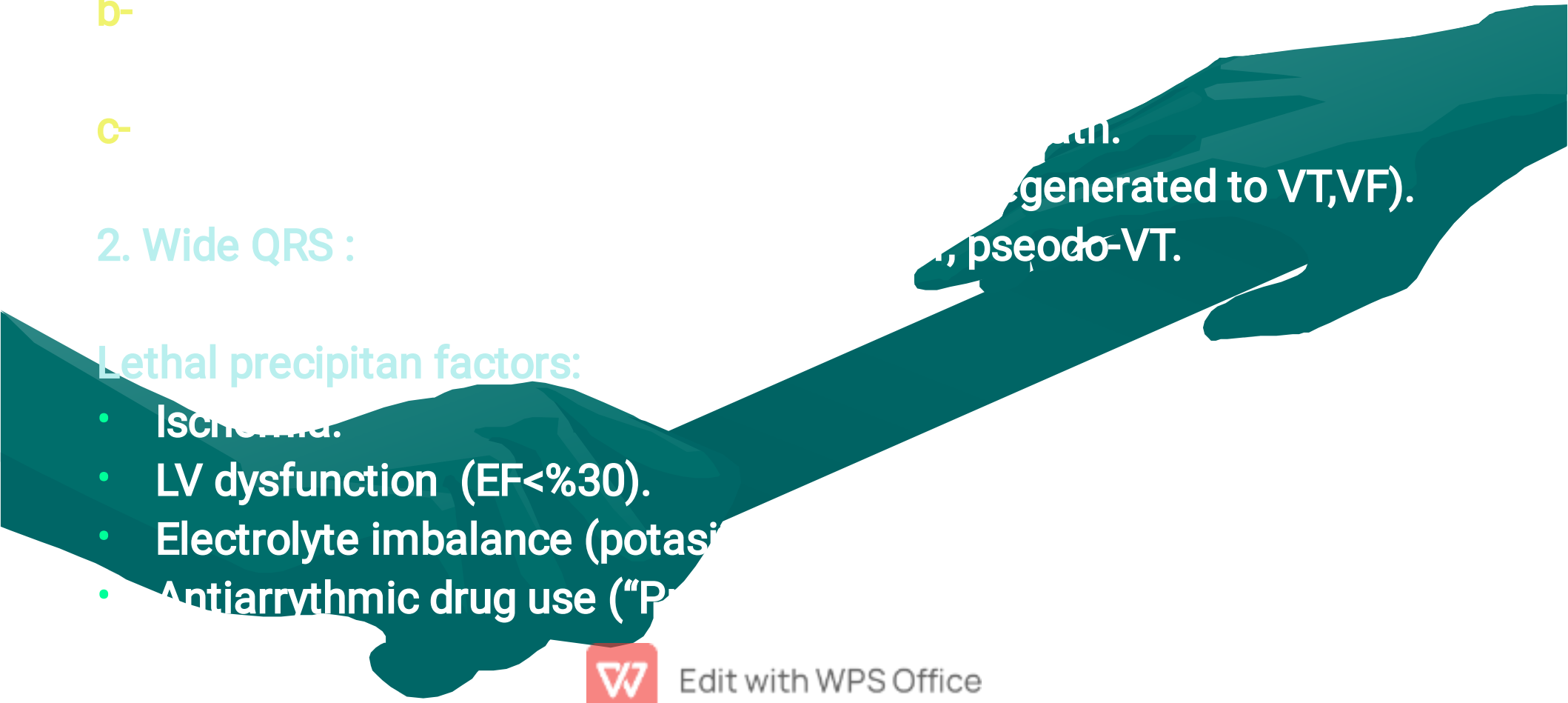
b-

c-

2. Wide QRS :

Lethal precipitan factors:

- Isch
- LV dysfunction (EF<%30).
- Electrolyte imbalance (potas
- Antiarrythmic drug use ("P



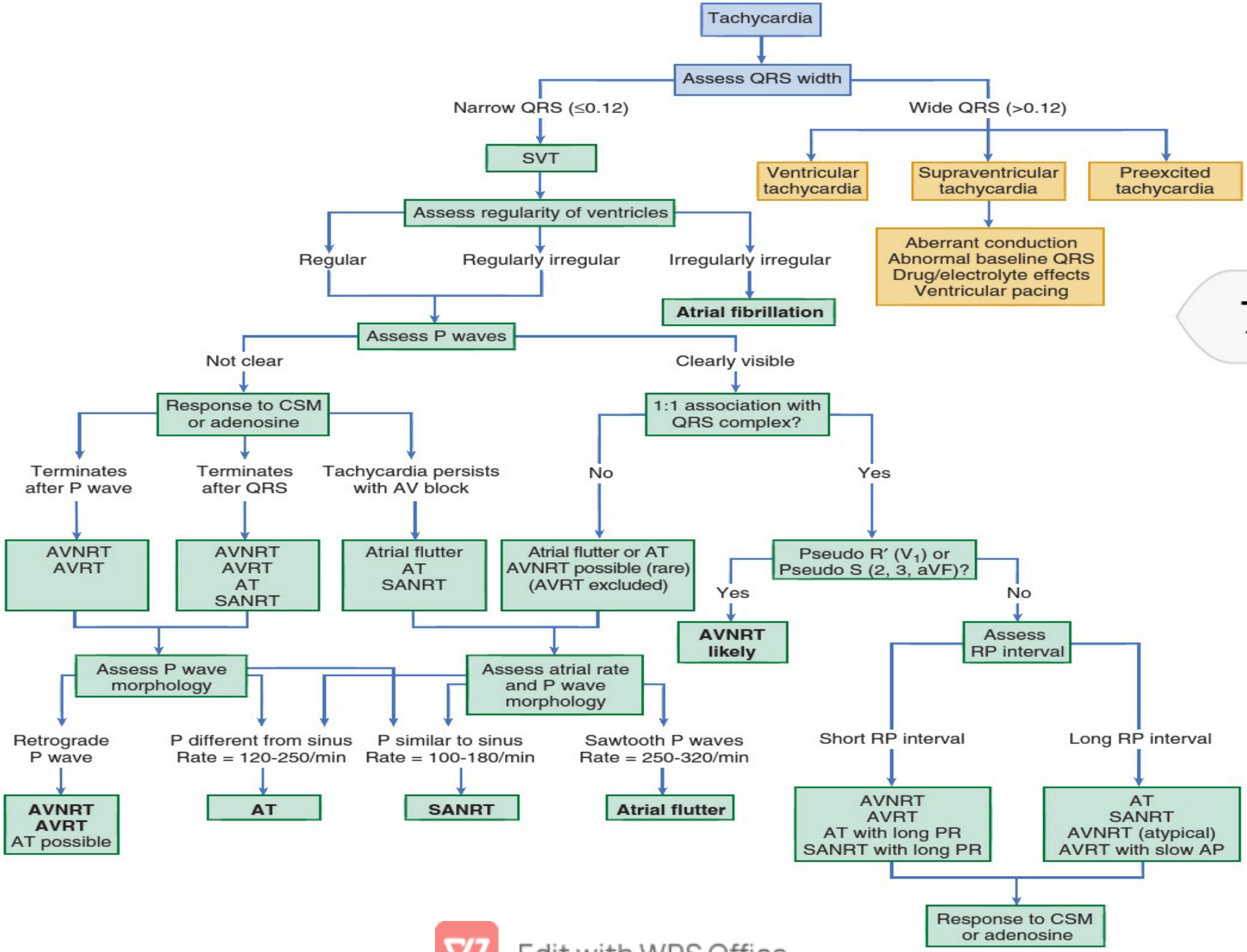
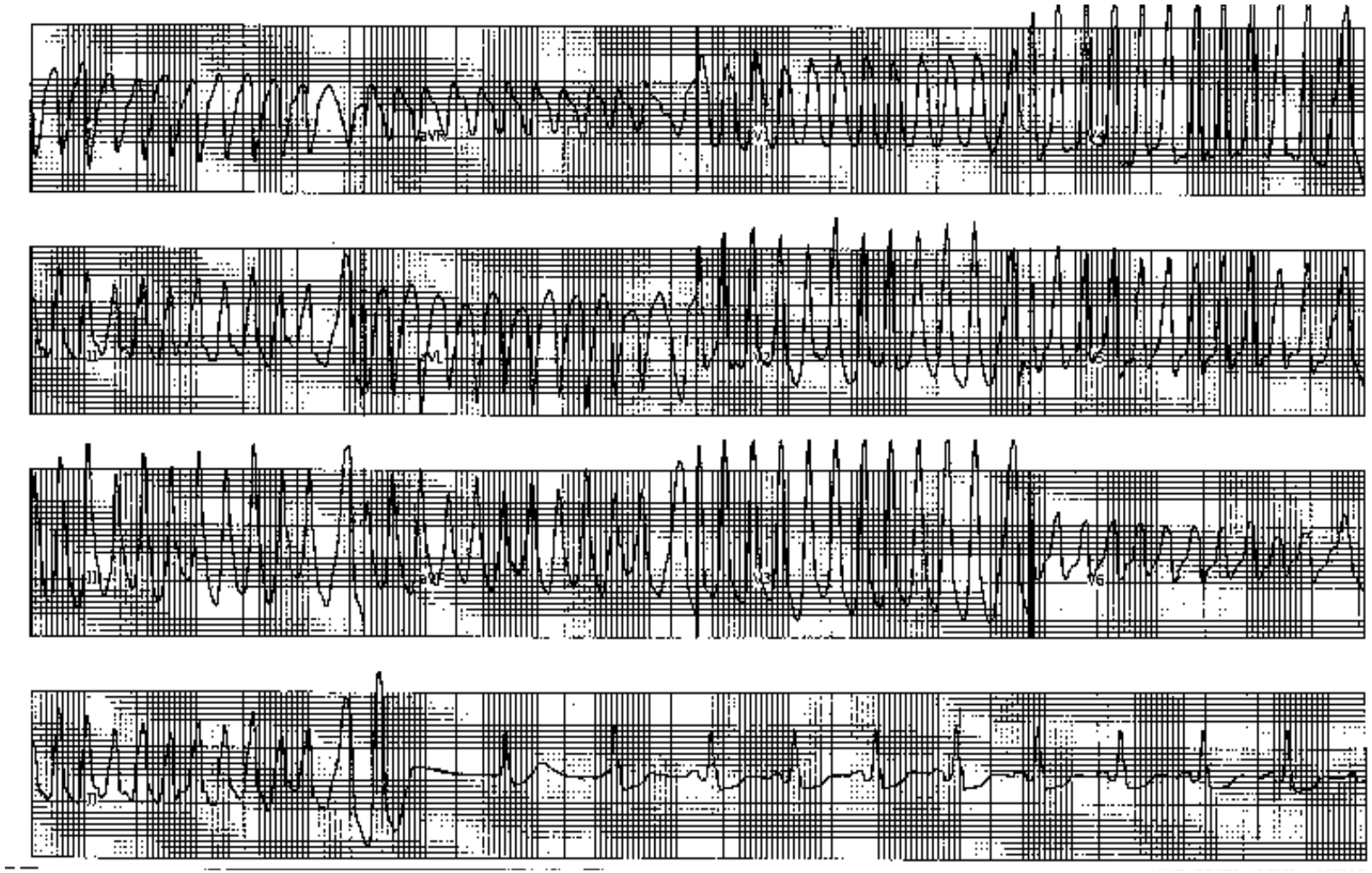
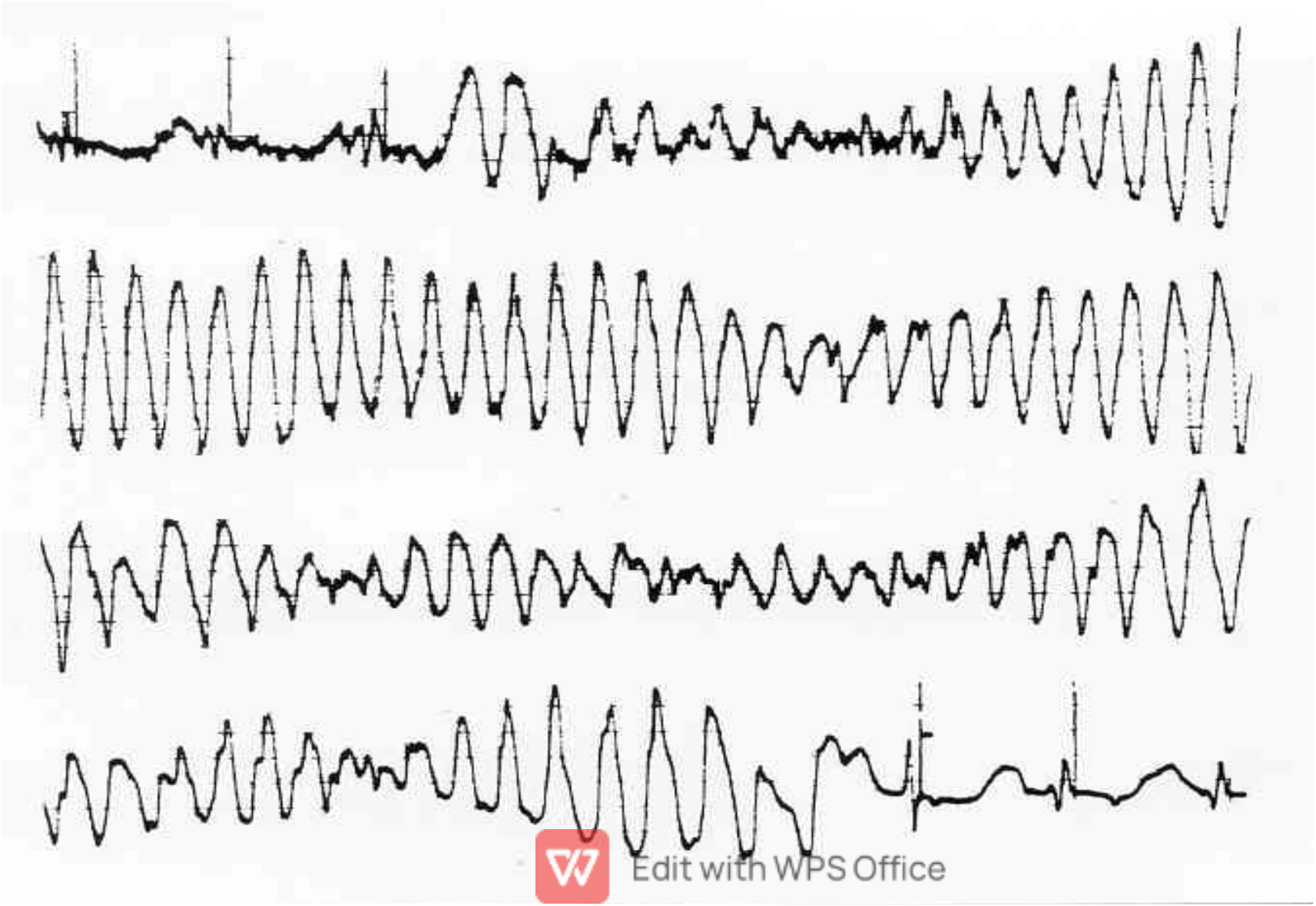


FIGURE 34-1 Stepwise approach to diagnosis of the type of tachycardia based on a 12-lead ECG during the episode. The initial step is to determine whether the tachycardia has a wide or narrow QRS complex. For wide-complex tachycardia, see Table 34-1; the remainder of the algorithm is helpful in diagnosis of the type of narrow-complex tachycardia. AP = accessory pathway; AT = atrial tachycardia; AVNRT = atrioventricular nodal reentrant tachycardia; AVRT = atrioventricular reciprocating tachycardia; CSM = carotid sinus massage; SANRT = sinoatrial nodal reentry tachycardia.

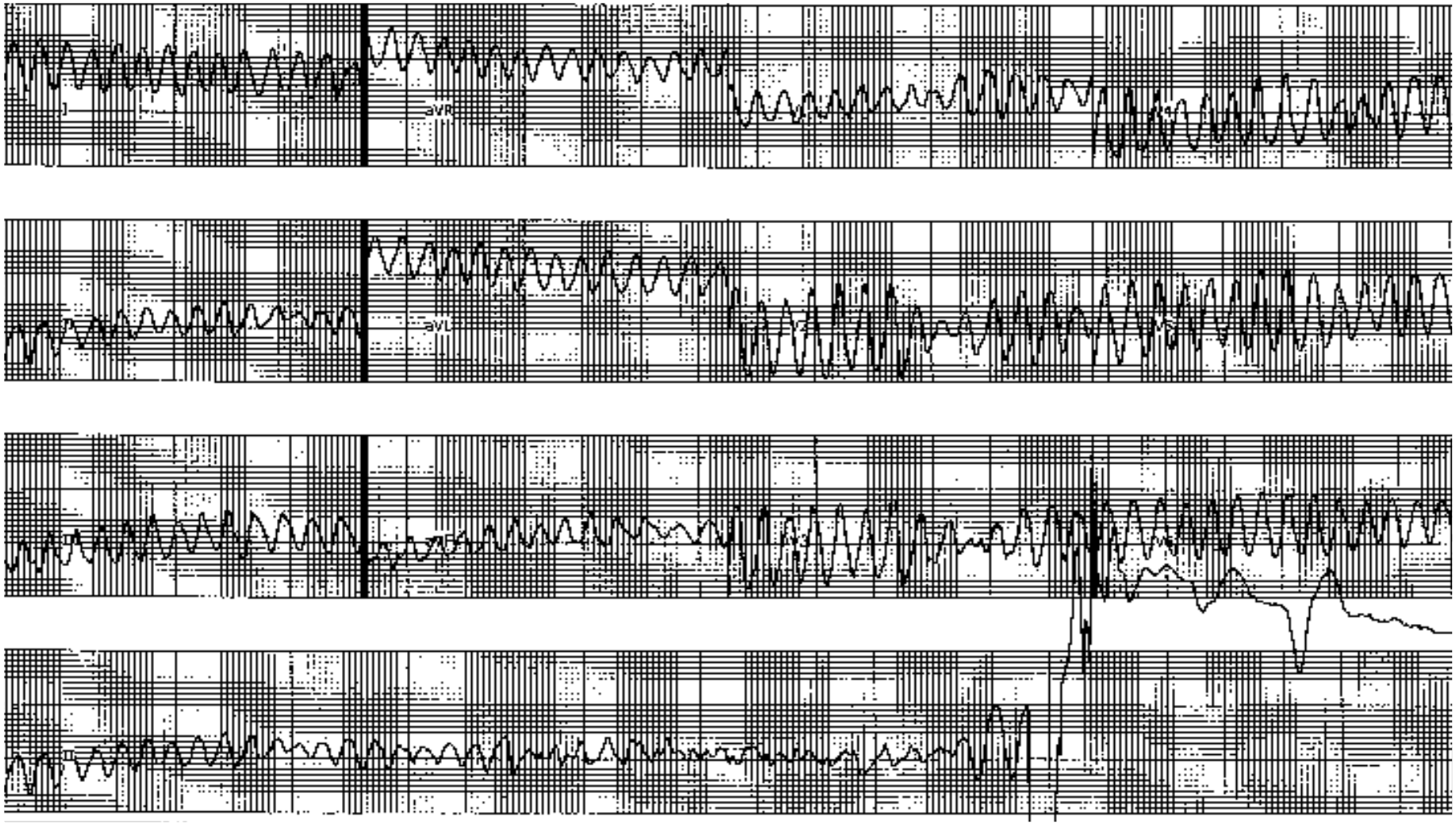
Ventrikular –Flatter, VT:
(Rate: 150200/min, arrythmic).



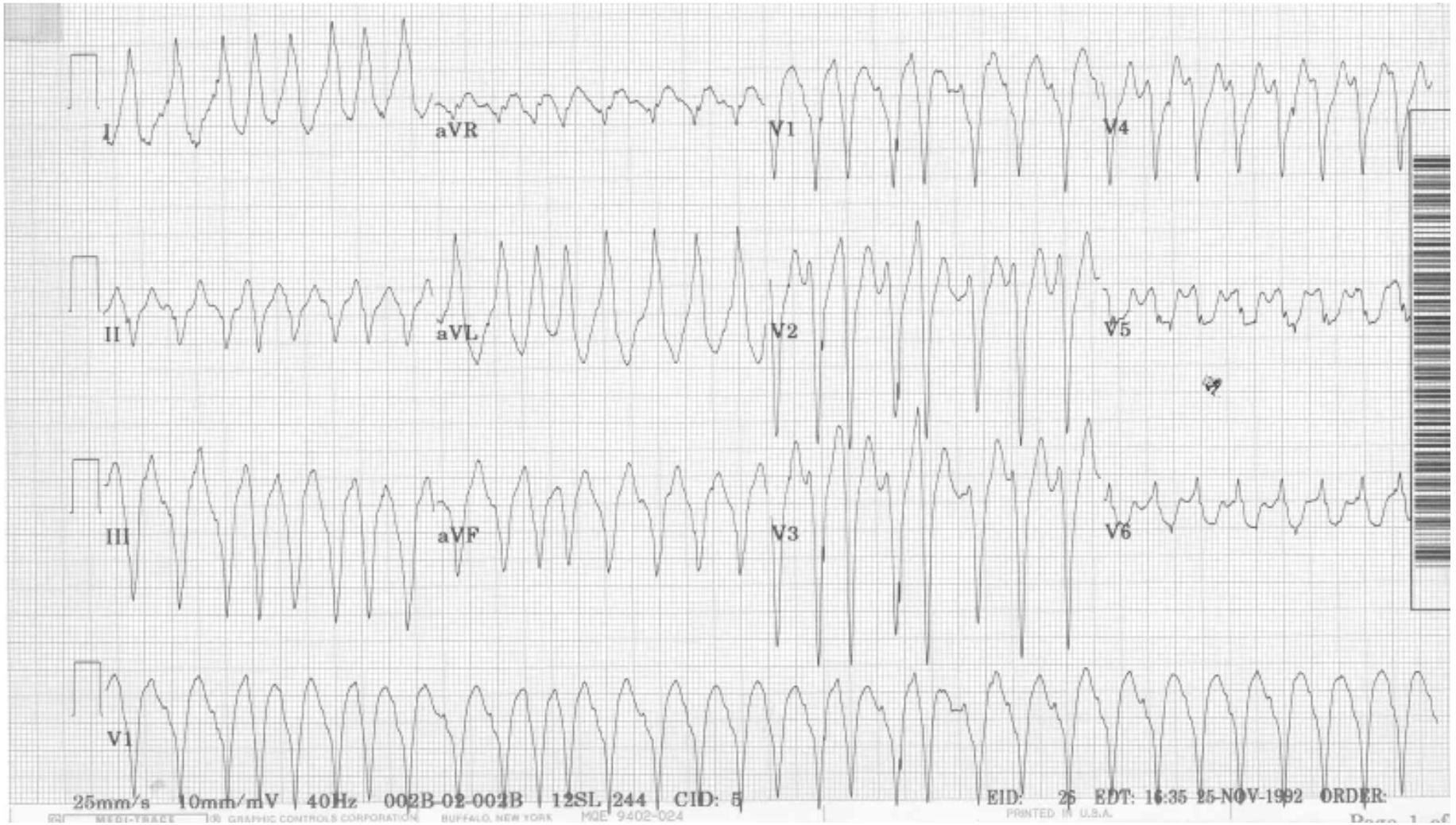
Torsades de Pointes-TdeP: Slow polimorphic VF/ T; Polimorphic VTA; Amplitude of ventricular activity changes constantly around isoelectric line.



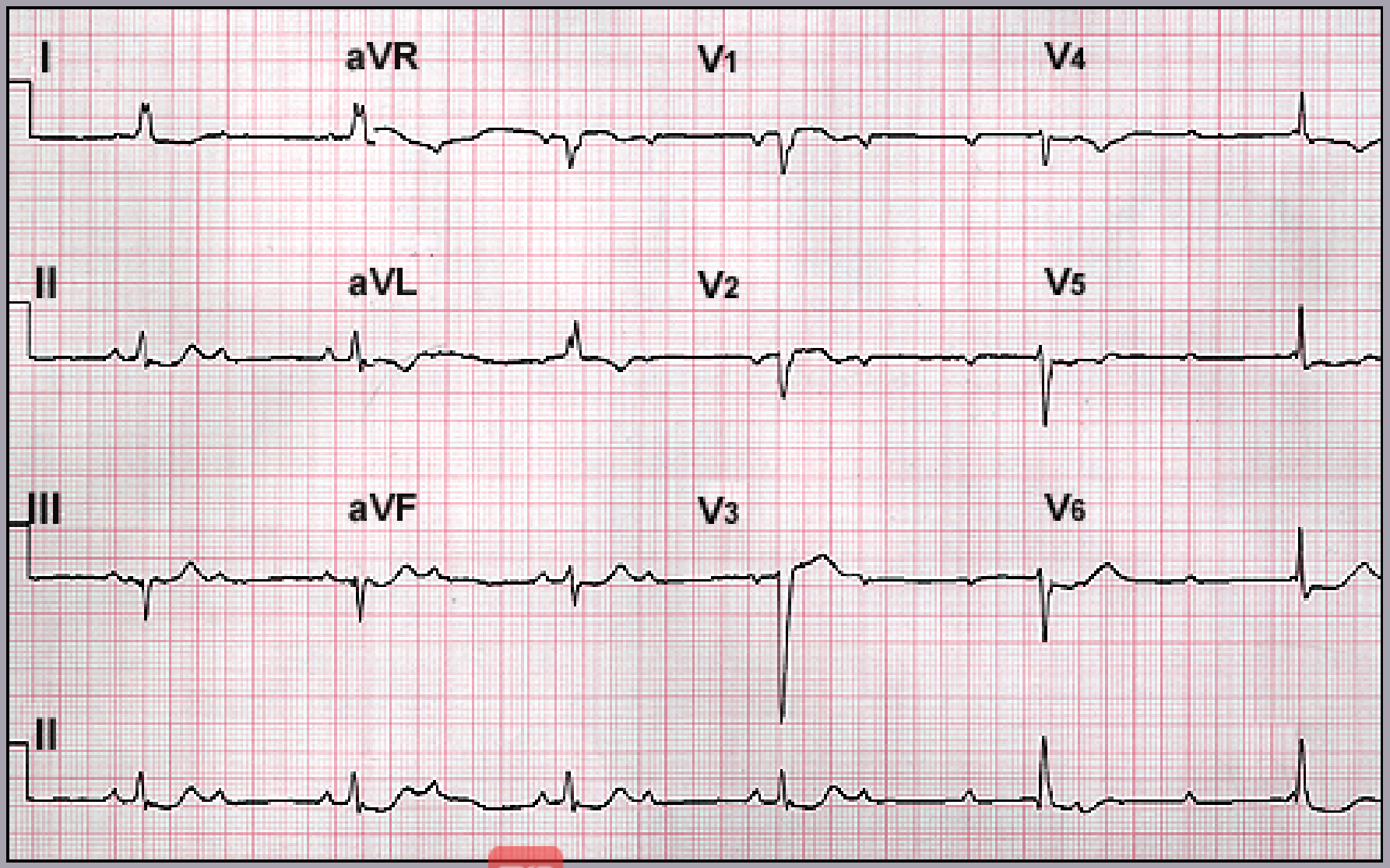
VF and Defibrillation.



Wide QRS and arrythmic tachycardia: AF, accessory way antidromic conduction; - not VT, ("Psödo-VT").



3. Degree AV Block.



Syncope: