Acute Myocardial Infarction – ST Elevation and Non-ST Elevation: Current Diagnosis and Management

August 4, 2009

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UTHSCSA and STVHCS

Onset of STEMI
- **Prehospital issues**
- **Initial recognition and management in the Emergency Department (ED)**
- **Reperfusion**

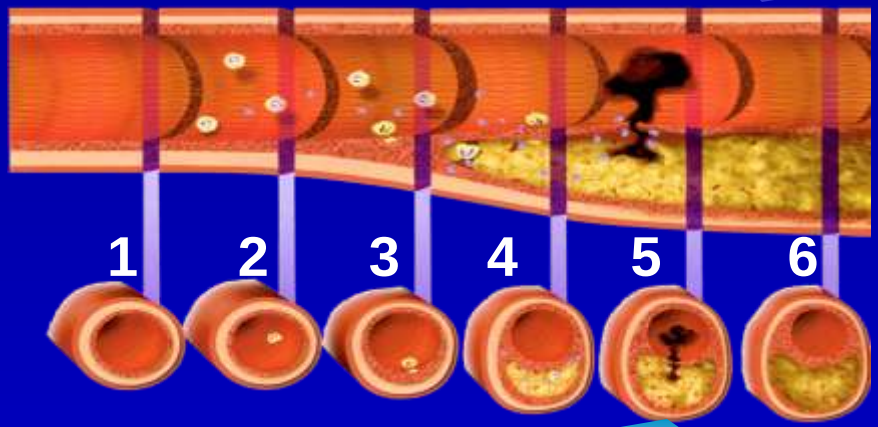
Hospital Management
- **Medications**
- **Arrhythmias**
- **Complications**
- **Preparation for discharge**

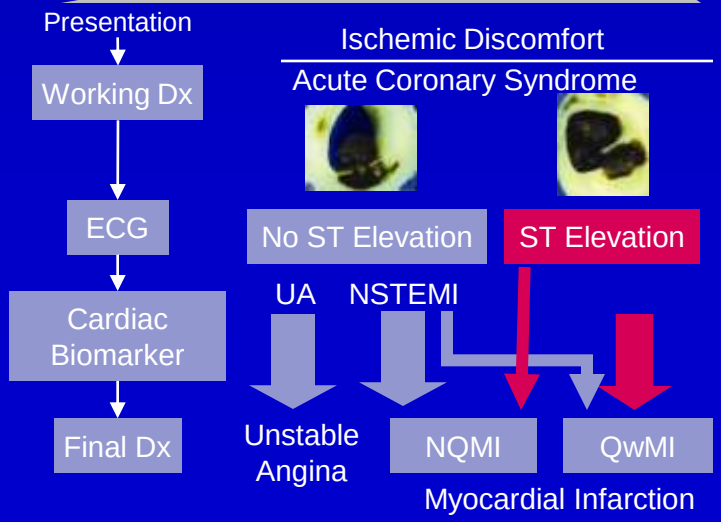
Modified from Libby. Circulation 2001;104:365, Hamm et al. The Lancet 2001;358:1533 and Davies. Heart 2000;83:361.

Management Before STEMI

Secondary Prevention/ Long-Term Management

Prevention:
Smoking
Blood pressure
Cholesterol
Diabetes





Chronology of the interface between the patient and the clinician through the progression of plaque formation and the onset of complications of STEMI.

The Coronary Artery in Acute MI

- Vulnerable plaque: lipid-rich, nonobstructive, abundant macrophages and inflammatory cells, at arterial branch points or bends (vulnerable plaques may be multiple)
- Rupture or erosion of fibrous cap, exposing subendothelium
- Platelet activation, adhesion and aggregation, thrombin generation and thrombus
- Thrombus; if inadequate collaterals, necrosis begins in 15 minutes, endo to epi, necrosis modulated by many factors, e.g. HR, BP, collateral flow
- ST elevation MI patients highly likely (90%) to have occlusive thrombus, Non ST elevation MI likely to have nonocclusive

Prehospital Symptomatic Management in MI

- NTG for chest pain, call 9-1-1 if pain persists over 5 minutes after **THE FIRST** NTG dose
- ASA chewable 162-325 mg
- Public safety first responders should be trained and equipped with AEDs
- EMS with 12-lead ECG and fibrinolytic agent and reperfusion checklist
- Transport STEMI to facility capable of emergent catheterization and revascularization
  - Cardiogenic shock in patient ≤75 yo and ≤18 hr of shock (I-A)
  - Patient with contraindications to fibrinolysis (I-B)

ACC/AHA Guidelines, STEMI 2004

Symptoms in Myocardial Infarction

- Chest pain or severe epigastric pain, nontraumatic, with typical features
  - Central or retrosternal compression or crushing
  - Pressure, tightness, heaviness, cramping, burning, aching
  - Unexplained indigestion, belching, epigastric pain
  - Radiating pain in neck, jaw, shoulders, back, or 1 or both arms
- Associated dyspnea
- Associated diaphoresis
- Associated nausea or vomiting
- Careful! Elderly may present with generalized weakness, stroke, syncope or change in mental status

Past Medical History in Myocardial Infarction

- Prior CAD, CABG, PCI, angina, or MI
  - How do current symptoms compare to prior symptoms?
- Nitroglycerin use
- Risk factors: smoking, Htn, HLP, DM, FH, Cocaine or methamphetamine
- Recent medication use

Initial ED Evaluation of MI - 1

- Brief H&P: pain history, prior CAD tests and procedures, examination (ABC, VS, general, JVP, rales, murmur/gallop, pulses, CVA, hypoperfusion)
- DDX – life-threatening: Ao dissection, Pulm Emb, Perf Ulcer, Tension pneumo, Boerhaave
- DDX – other: Pcard, Atypical angina, Repol, WPW, CNS-T waves, LVH strain, Brugada, Myocarditis, Hyperkalemia, BBB, Vasospasm, HCM
- DDX – noncardiac: GERD, Chest wall pain, pleurisy, PUD, Panic, C-spine-radiculop, biliary-pancreatic, somatization and psychogenic

ACC/AHA Guidelines, STEMI 2004

Initial ED Evaluation of MI - 2

- ECG – if no initial ST elevation, serial ECGs for continued symptoms or high clinical suspicion; HCP sees ECG within 10 minutes; in inferior wall STEMI obtain right chest leads
- Lab should not delay therapy – biomarkers esp troponin, CBC, INR, PTT, chem-7, Mg, FLP
- CXR should not delay therapy – unless suspect dissection
- For suspected dissection – TTE and/or TEE, and MRI or CT with contrast

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ED Management of MI

- O2 for O2 sat< 90%, or for 6 hours
- Nitroglycerin SL q5min x 3, then assess for IV NTG
  - IV NTG indicated for ongoing ischemic discomfort
  - No NTG: if SBP<90, HR<50, HR>100, suspected RVMI, PDE-5 inhibitor <24-48h
- Morphine 2-4 mg IV + 2-8 mg IV q5-15 min for pain of MI
- ASA chewable 162 -325 mg if not already taken
- Oral beta blocker promptly unless contraindicated
- IV beta blocker for tachycardia or hypertension
- All ST Elevation should have reperfusion – of some kind… If no ST elevation, no routine reperfusion

ACC/AHA Guidelines, STEMI 2004

STEMI Risk Assessment –TIMI

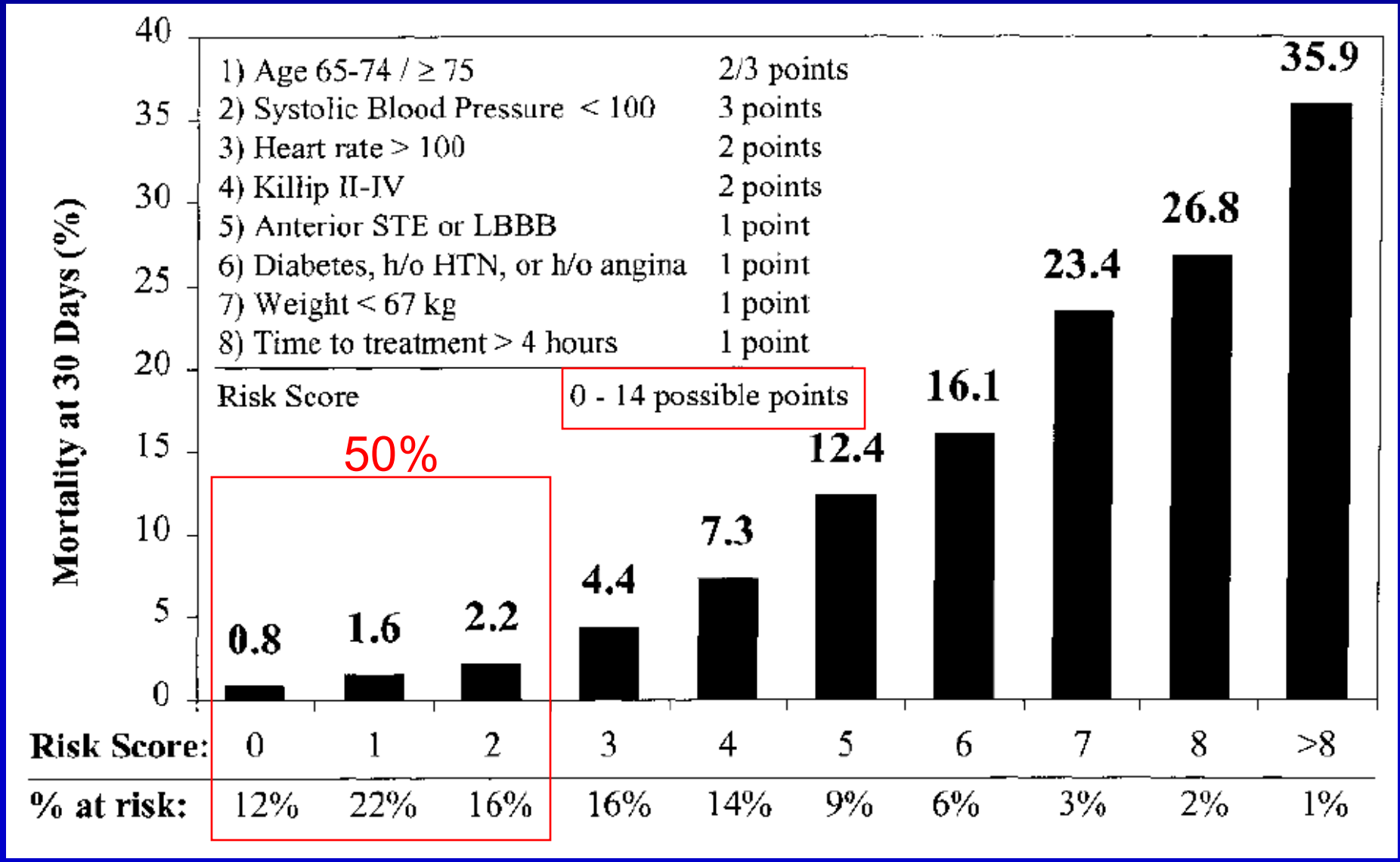
Prognostic variables	Points
Historical	
Age >75	3
Age 65-75	2
DM, htn, AP	1
PE	
SBP<100	3
HR>100	2
Killip 2-4	2
Wt <150	1
Presentation	
Ant STEL or LBBB	1
Time >4h to reperfusion	1

Score	30-da Mortality
0	0.8%
1	1.6%
2	2.2%
3	4.4%
4	7.3%
5	12%
6	16%
7	23%
8	27%
>8	36%

Retrospective analysis of 14,114 pts, InTIME II Trial; overall mortality 6.7% @30 days

Morrow DA et al. Circulation 2000;102:2031.

STEMI Risk Assessment –TIMI



Morrow DA et al. Circulation 2000;102:2031.

Management of ST Elevation MI

- Antithrombin (heparin, LMWH)
- **Reperfusion** (thrombolysis<30 or PCI<90)
- IIb/IIIa inhibitor with PCI
- ACE-I PO not IV <24hr if anterior or EF<40 or HF with BP>100 (and all patients later)
- ARB if ACE intolerant or with ACE if EF<40 and HF
- Insulin infusion if complicated course, or for 24-48 hr in any hyperglycemic patient
- Aldo antagonist if EF<40 with HF or DM and no contraindications

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Fibrinolysis <30 min

Generally Preferred

- Early presentation (≤3 h symptoms and delay to invasive strategy
- Invasive strategy not option (cath lab or skilled PCI not available, vascular access difficulty)
- Delay to invasive strategy (prolonged transport >60 min to balloon, or door to balloon >90 min)

Invasive Strategy

Generally Preferred

- Late presentation (>3 h symptoms)
- Skilled PCI lab available with surgical backup (door to balloon <90 min)
- Cardiogenic shock or Killip class 3-4
- Contraindications to fibrinolysis
- Diagnosis of STEMI in doubt

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Absolute Contraindication to Fibrinolysis in ST Elevation MI

- Any prior intracranial hemorrhage
- Known structural CV lesion (AVM) or malignant CNS neoplasm
- Stroke < 3 mo except stroke <3h
  - (>3 mo=relative contraindication)

CNS

- Suspected Ao dissection
- Active bleeding or bleeding diathesis (except menses; active peptic ulcer is relative contraindication)
- Significant closed-head or facial trauma within 3 mo.

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Relative Contraindication to Fibrinolysis in STEMI

- Hypertension
  - chronic severe poorly controlled
  - on presentation (SBP>180 or DBP >110)
- Prior stroke >3 mo, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (>10 min) CPR or major surgery (<3 wk)
- Recent (<2-4 wk) internal bleeding
- Noncompressible vascular punctures
- For streptokinase/ anistreplase: prior exposure (>5 da) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

ACC/AHA Guidelines for the Management of ST Elevation MI, August 2004

Acute MI: In-Hospital Complications

- Mechanical
  - Heart failure, cardiogenic shock from LV dysfunction
  - Rupture (papillary muscle, IV septum, LV free wall)
- Pericarditis (history, rub, ECG)
  - Epicardial irritation from transmural infarction
  - Auto-immune (Dressler's syndrome)
  - Epicardial irritation from blood (impending rupture)
- Electrical

Heart Failure in Acute MI

- Killip Classification (Mortality)
  - Class I: normal (2-5%)
  - Class II: rales, mild increase in respiratory rate without dyspnea (10-15%)
  - Class III: pulmonary edema (20-30%)
  - Class IV: Cardiogenic shock (50-60%)
- Shock is hypotension, poor perfusion, confusion, cyanosis, oliguria (context is adequate preload)

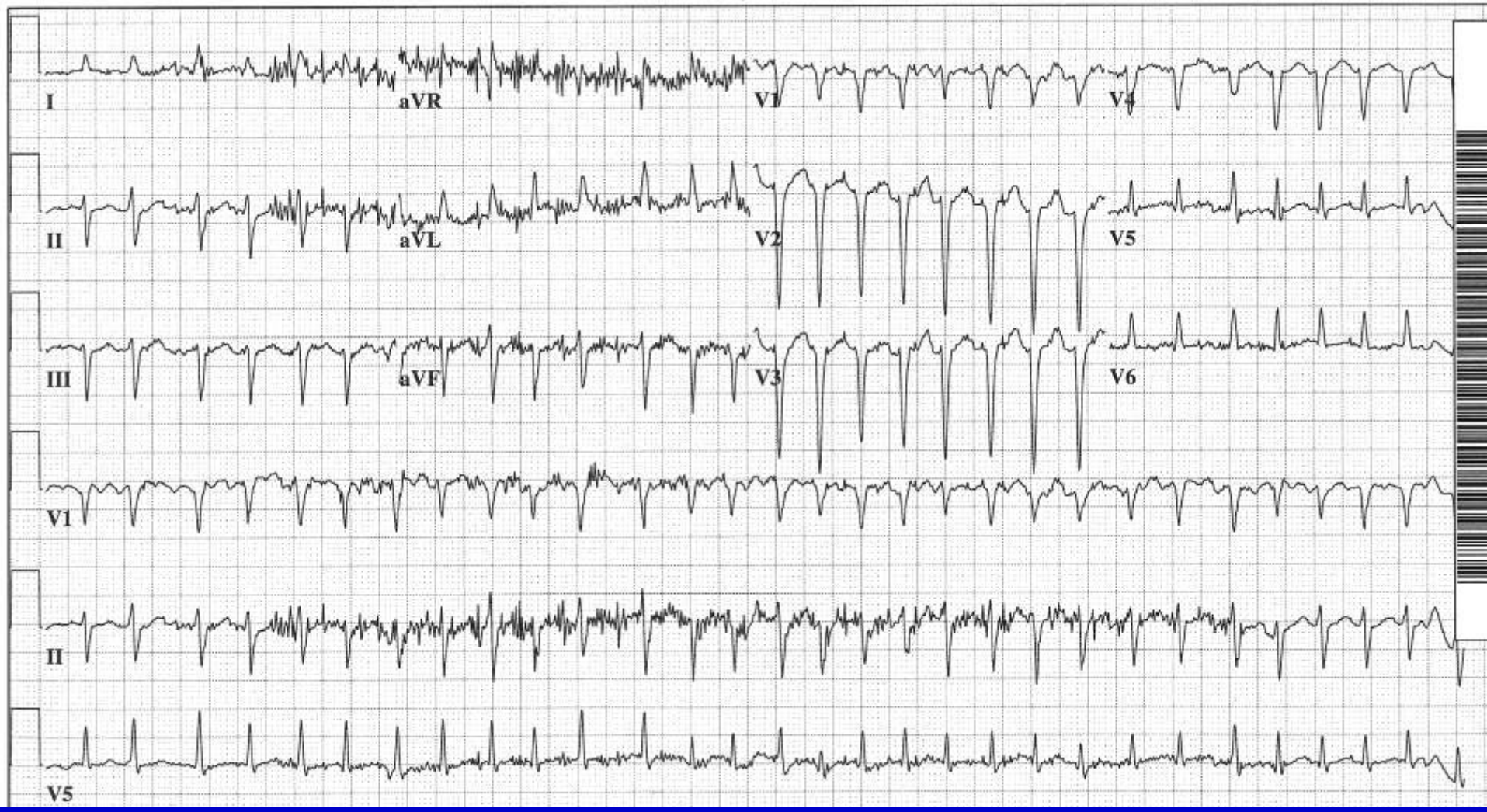
Mechanical Rupture in MI

Often about 3-5 days after onset of MI

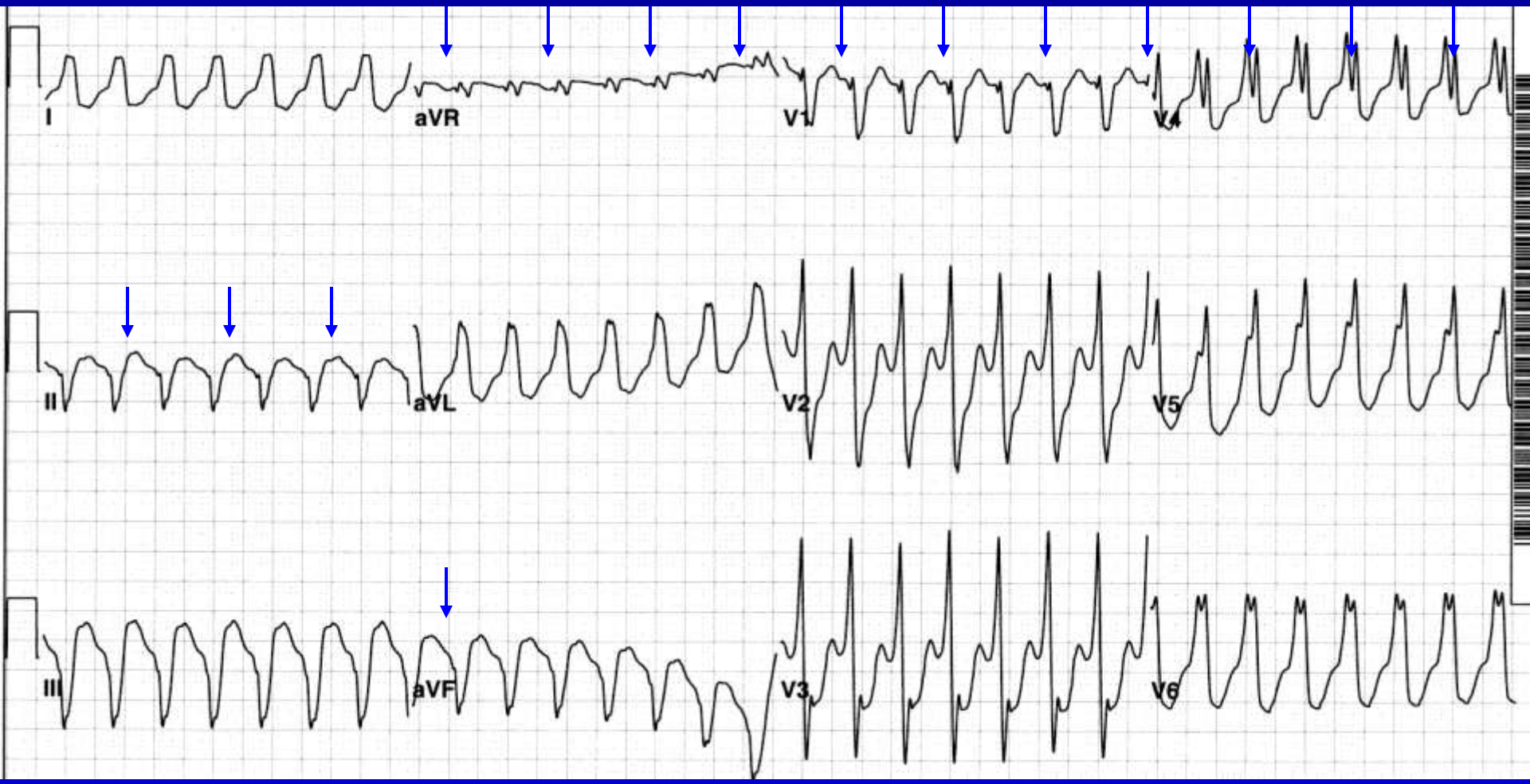
- Papillary muscle rupture – acute severe MR with systolic apical murmur and sudden pulmonary edema, needs emergency surgery
- Ventricular septal rupture – acute left-to-right shunt with systolic murmur at LLSB or RLSB, often pulmonary edema and often needs surgery
- LV free wall rupture – acute cardiac tamponade – usually fatal, needs emergent surgery, can heal on its own as a pseudoaneurysm.

Acute MI: Electrical Complications

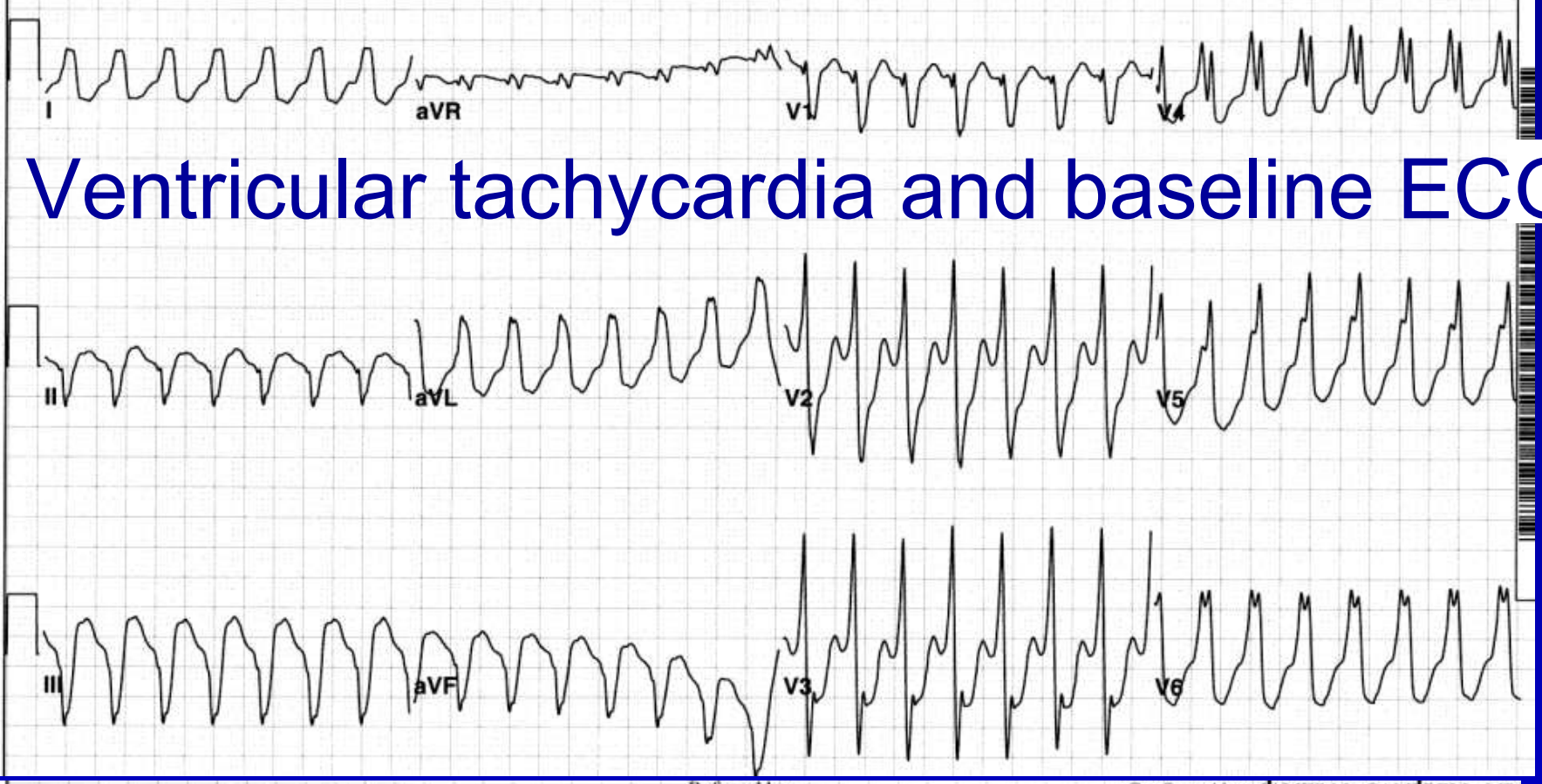
- Tachyarrhythmias
  - Sinus tachycardia secondary to HF or hypoxia or pain, etc
  - Atrial fibrillation or other atrial arrhythmia
  - Ventricular tachycardia/fibrillation
- Bradycardias
  - Sinus bradycardia associated with inferior wall MI
  - AV block associated with inferior wall MI or anterior wall MI

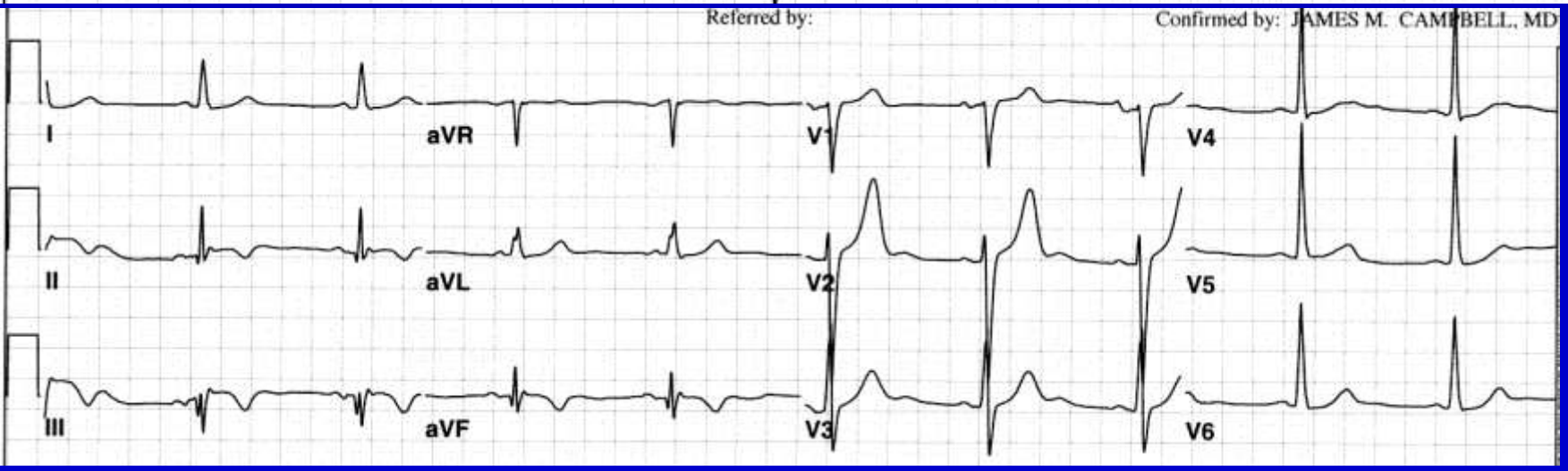

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

Ventricular tachycardia, sinus rhythm with sinus rate slightly less than half the ventricular rate

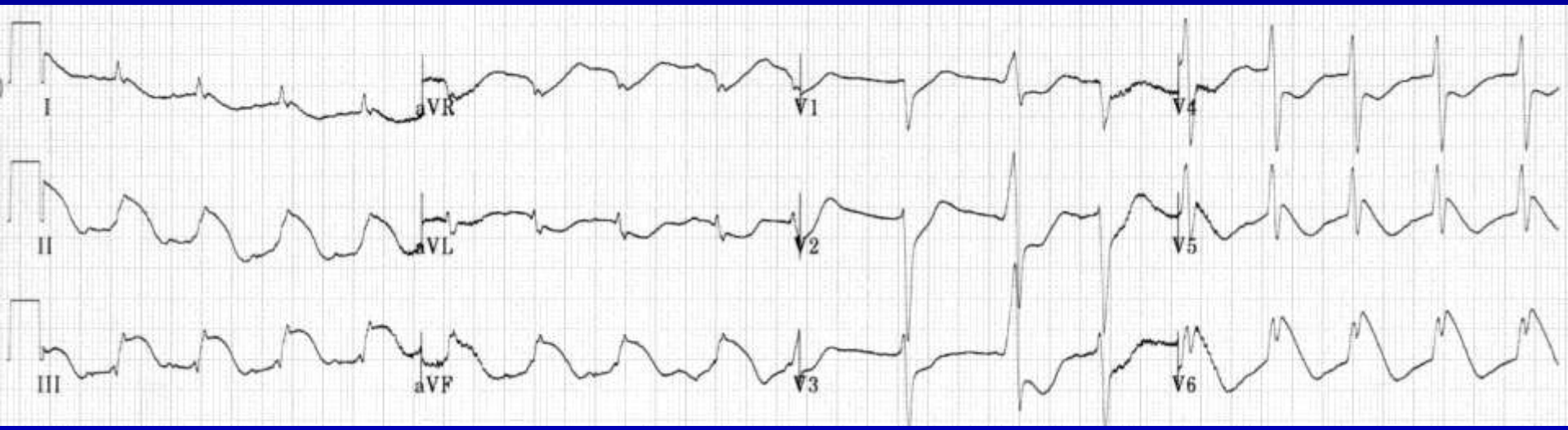


Ventricular tachycardia and baseline ECG



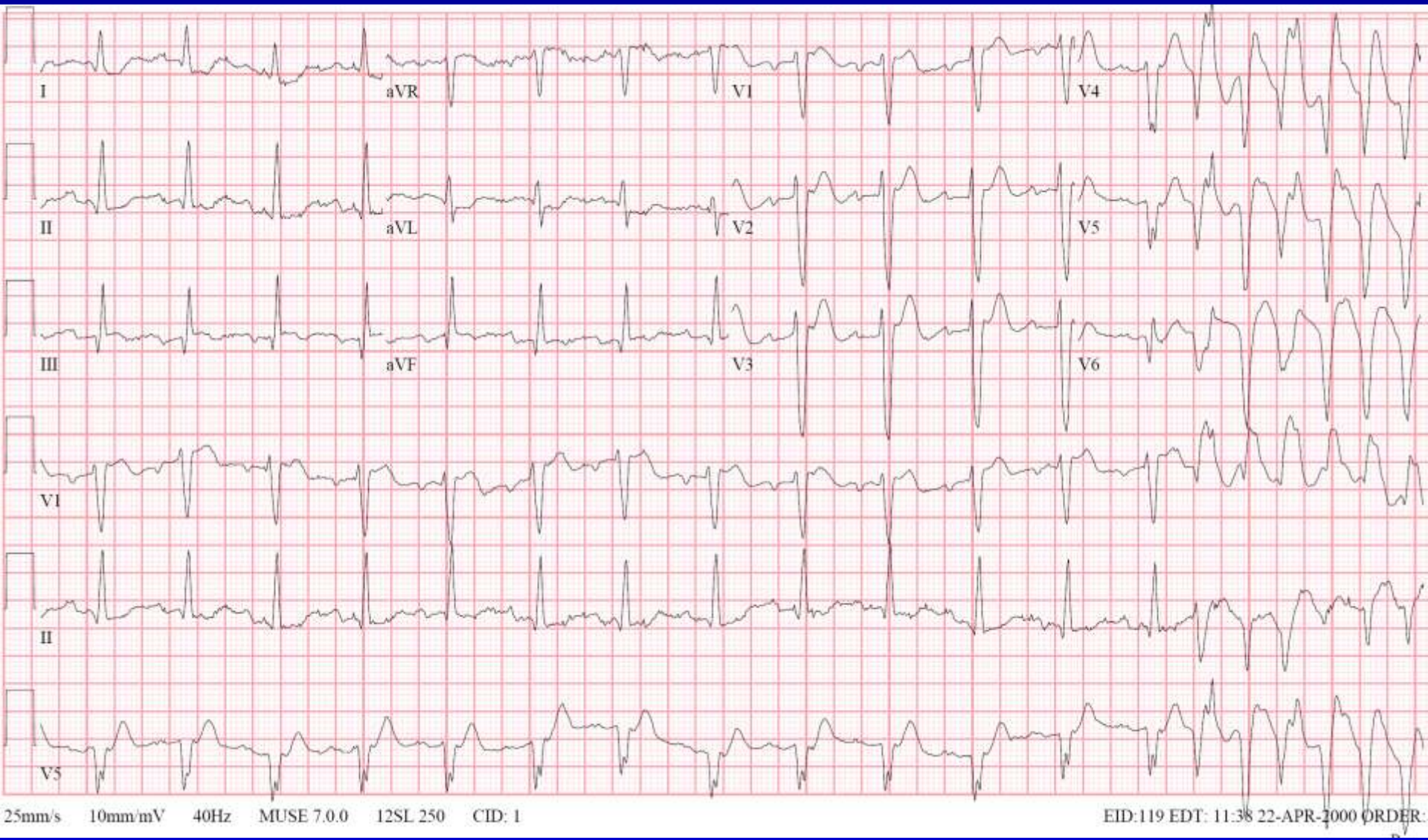


Case 2



Case 8

April 11, 2000, 08:15:11



Case 8

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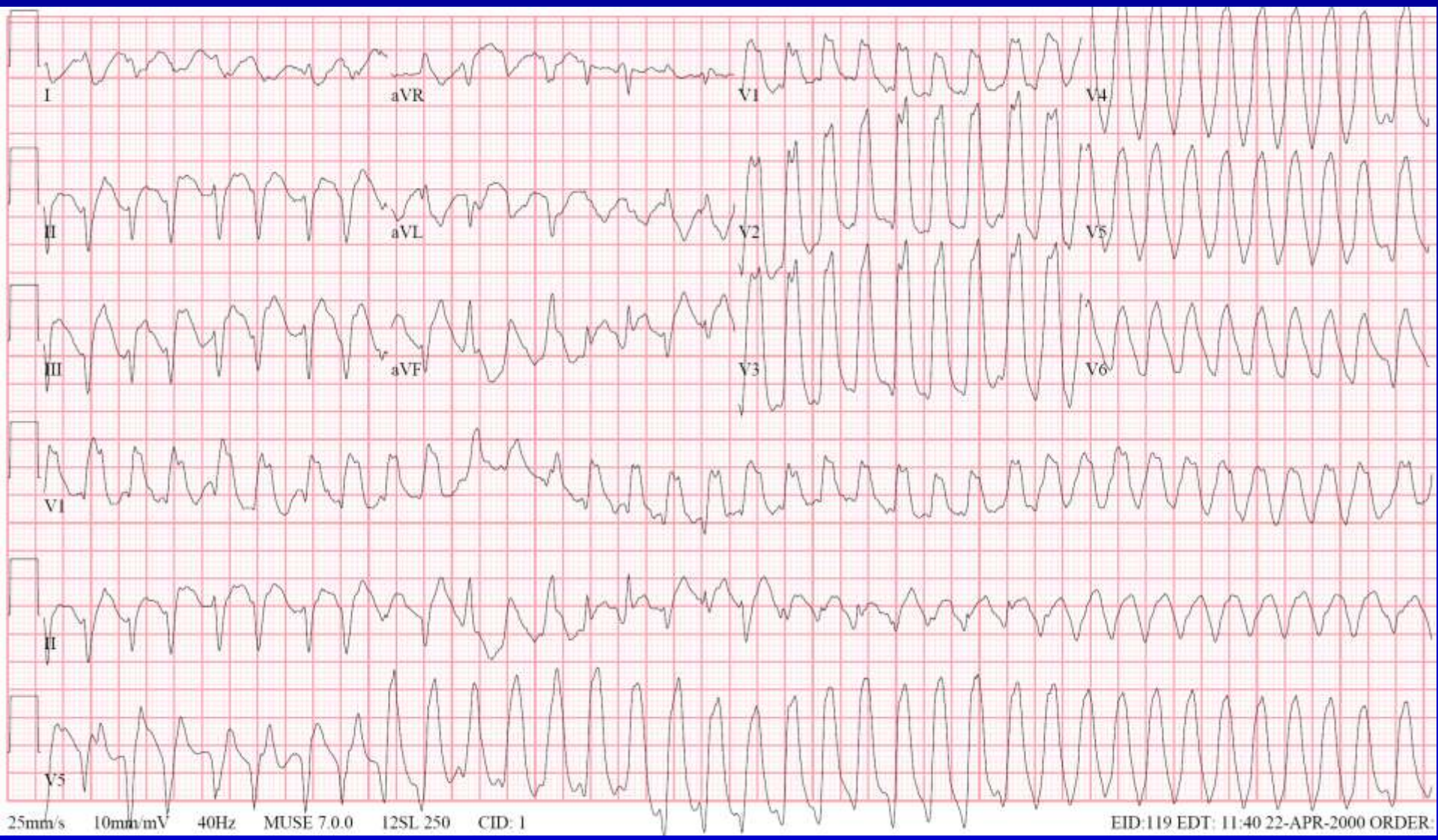
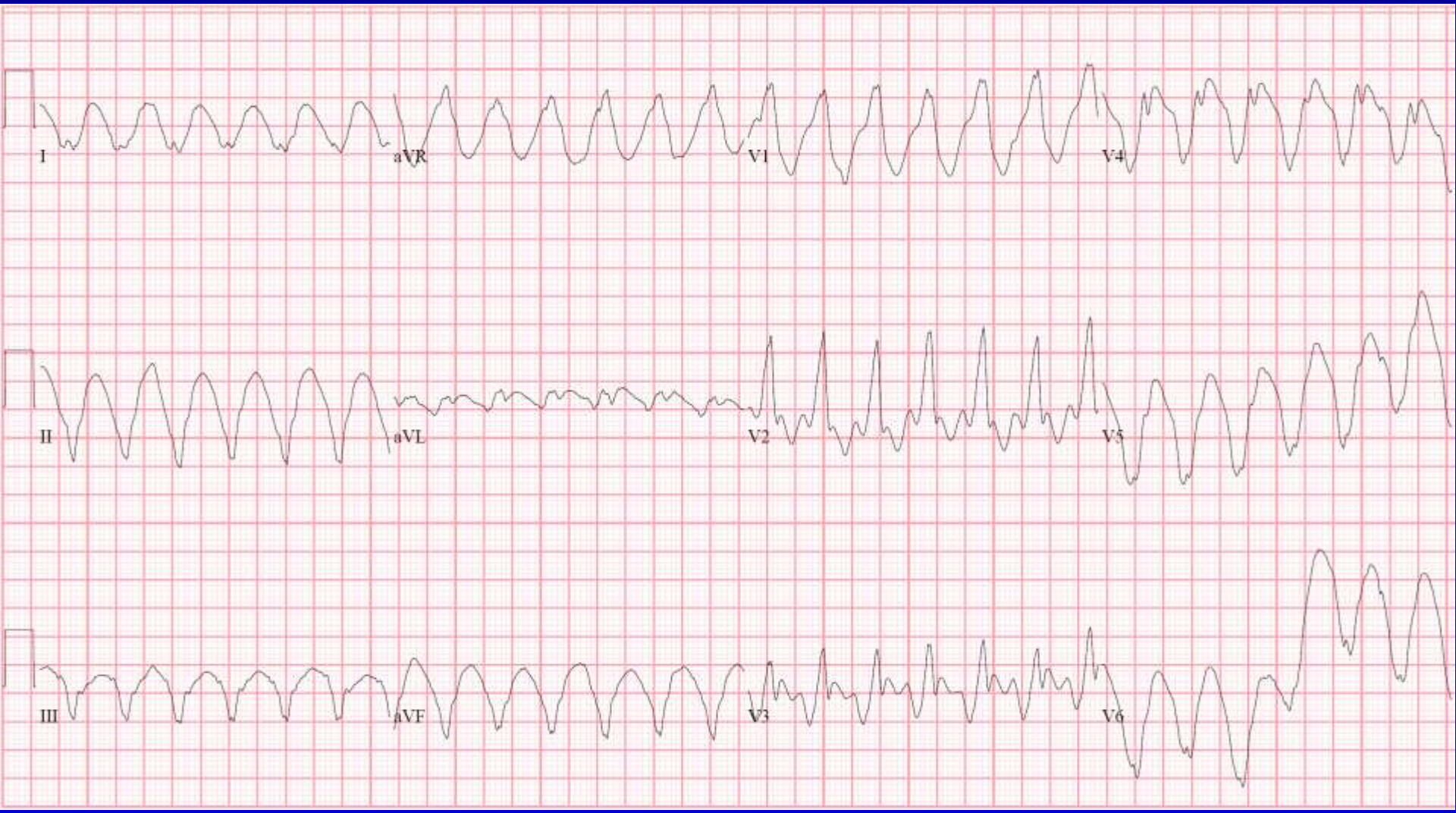


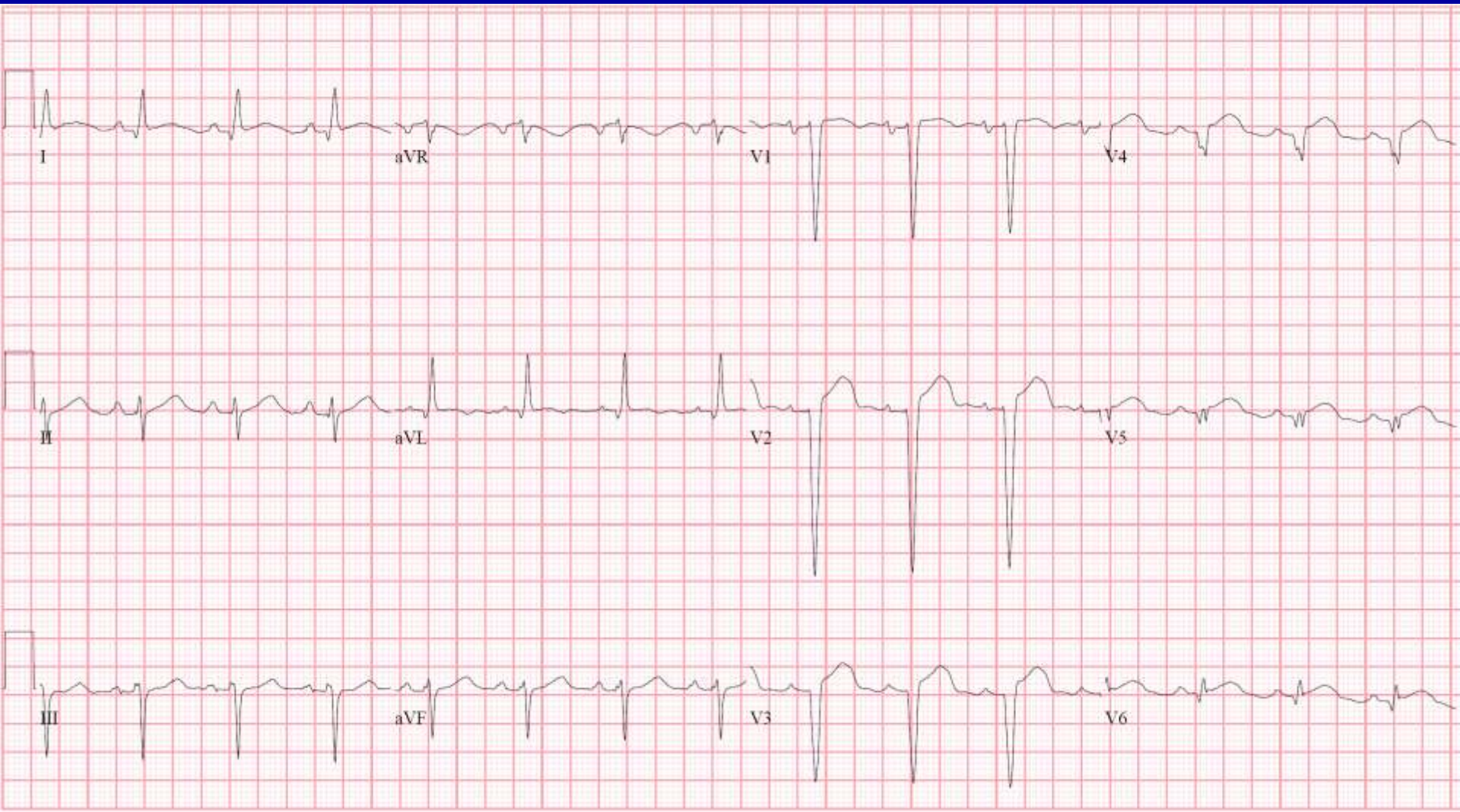
October 1987, rate 160

Case 20

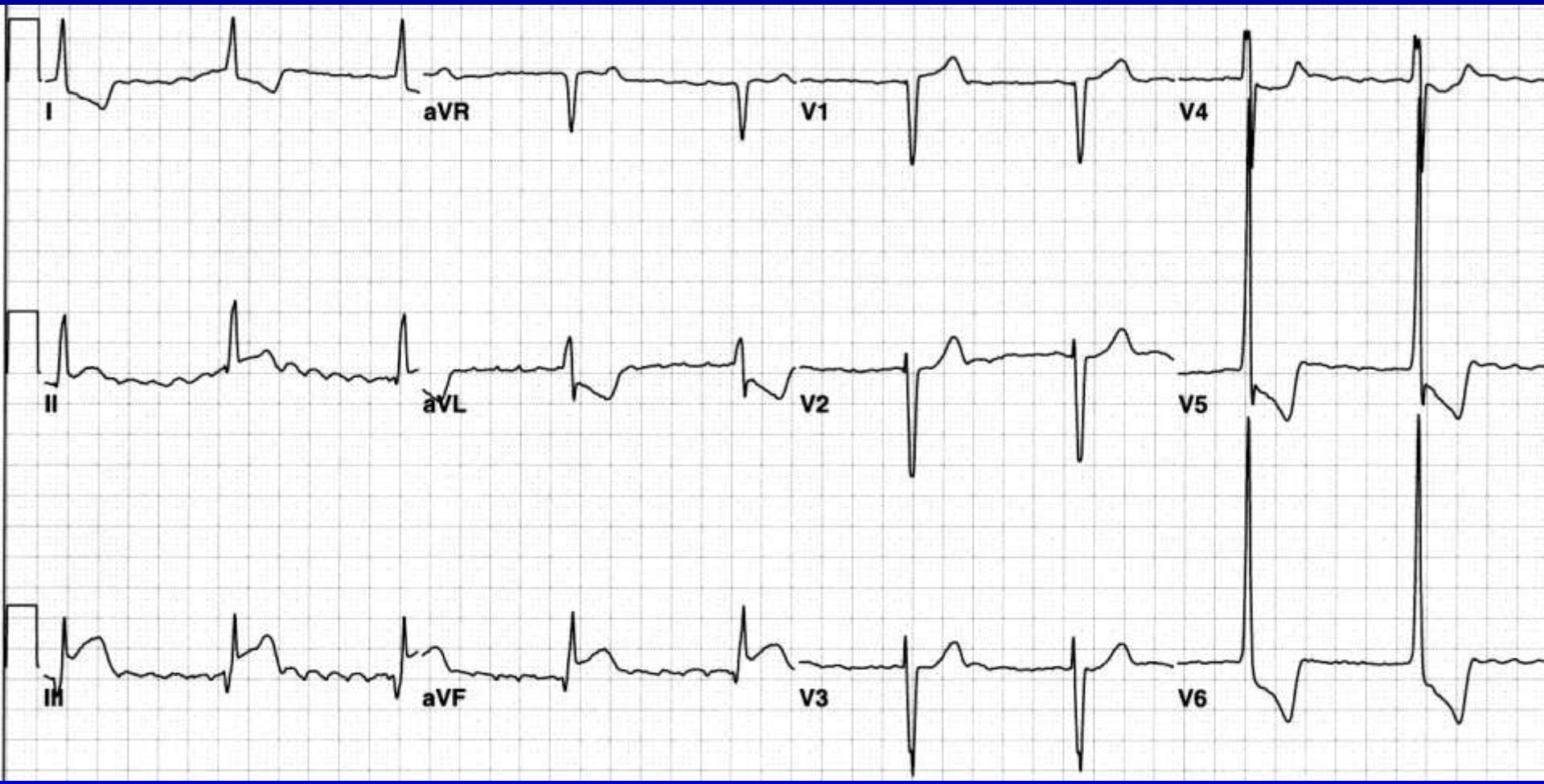


October 1987, 2 hr later

Case 20



Third Degree AV block



Atrial fibrillation with narrow QRS - junctional escape.
Acute inferior injury pattern!

Procedures in Management of ST Elevation MI - Indications

- Swan-Ganz: hypotension unresponsive to fluid or with congestion, suspected VSD or severe MR or free wall rupture or tamponade and no echo done
- Art line: BP <80, or inotropes or cardiogenic shock
- Echo: BP<90, low output state, urgent for pulmonary congestion, possible RV MI, stroke as complication of MI
- IABP: cardiogenic shock not quickly responsive to meds

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Later Management in ST Elevation MI

- Mechanical complications of STEMI occur at <24h or at 3-5da: Rupture of ventricular septum, mitral papillary muscle, LV free wall are emergencies, need surgery
- Pericarditis –use ASA 650 q4-6h, alternative is colchicine 0.6 mg q12h or acetaminophen 500 q6h, steroids last resort; avoid indomethacin
- Evaluate LV systolic function if not known prior (Echo, LV gram with catheterization, MUGA)

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Acute Coronary Syndromes (UA/NSTEMI)

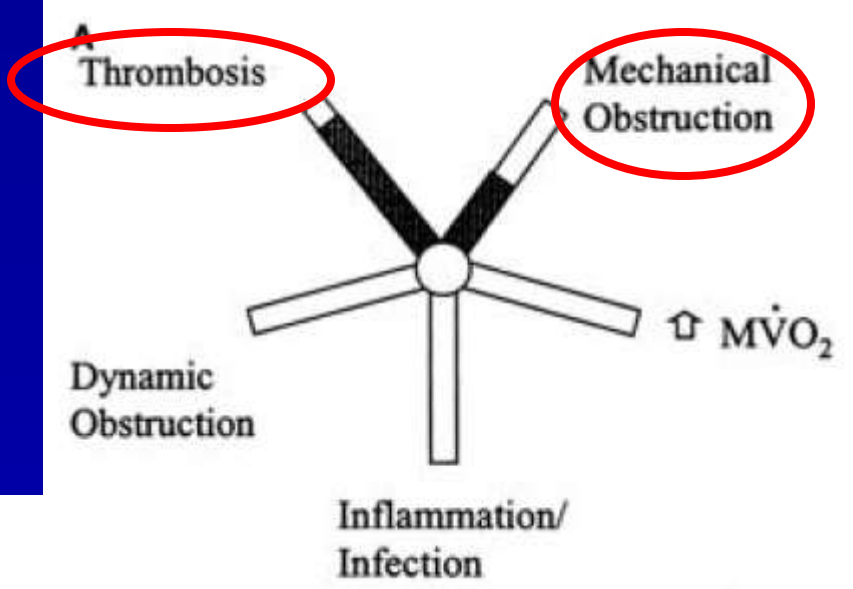


Table 2. Causes of UA*

Nonocclusive thrombus on pre-existing plaque
Dynamic obstruction (coronary spasm or vasoconstriction)
Progressive mechanical obstruction
Inflammation and/or infection
Secondary UA

*These causes are not mutually exclusive; some patients have greater than or equal to 2 causes.

Reprinted with permission from Braunwald E. Unstable angina: an etiologic approach to management. Circulation 1998;98:2219–22.

Acute Coronary Syndromes - 1

- Presentations:
  - Rest angina, usually >20 minutes;
  - New onset angina, usually frequent and disabling (CCS-3);
  - Increasing angina distinctly more bothersome (CCS-3), within <2 weeks-2 months
- 5 million ER chest pain visits/yr in US; 1.7 million admissions for ACS (1.5 million discharge diagnoses, 600k deaths)
- Age distribution: 45% are <65 yo, only 5% <40 yo

Acute Coronary Syndromes - 2

- NSTEMI: positive biomarkers (trop I, trop T, CK-MB), majority no Q wave
- UA: negative biomarkers
- Pathophysiology: supply/demand imbalance, usually ASCAD and plaque rupture and thrombi (NSTEMI and UA usually nonocclusive, STEMI usually occlusive), occasionally NSTEMI and UA are mere severe atherosclerotic narrowing, particularly in restenosis
  - Rare vasospasm (Prinzmetal's)
  - Secondary UA from hypotension, hypoxemia, anemia, tachycardia, or thyrotoxicosis

Prognostic Factors in NSTEMI

- **Clinical**: rest angina, 2 anginal episodes in 24 h*, age >65*, 3 or more traditional risk factors*, DM, Hx CAD with >50% obst*, ASA use in past week*, need for IV NTG
  - **PE**: low BP, diaphoresis, pulm edema, S3, transient MR
- **ECG**: ST depression (0.5mm) or transient elevation*, T inversion with pain
- **Biomarker**: any elevation*
  - **Additional marker**: CRP, BNP (not routine needed)
  - **Angiographic**: Coronary thrombus, high-grade CAD
  - **Noninvasive testing**: WMA at rest or stress echo, reversible defects on scan

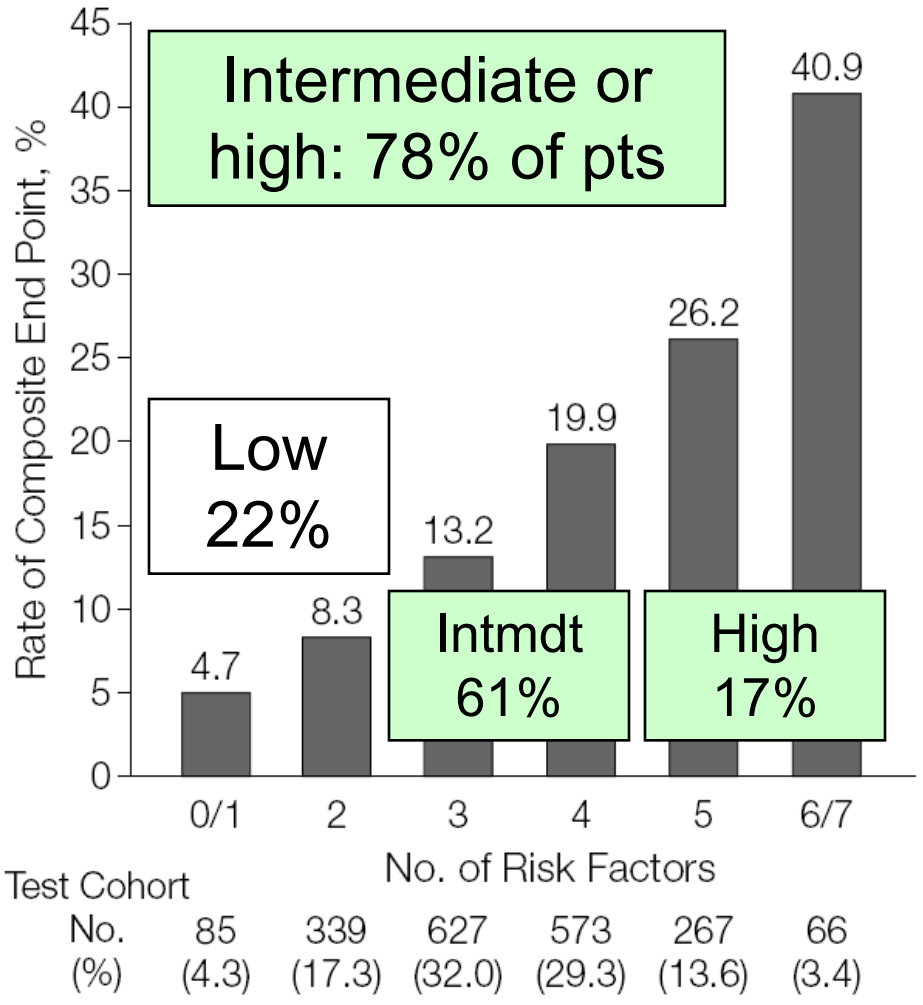
*= **7 TIMI points** (5 clinical, one ECG, one biomarker)

UA/NSTEMI TIMI Risk Score

- 2 anginal episodes in 24 h
- age >65
- 3 or more traditional risk factors
- Hx CAD with >50% obst
- ASA use in past week
- ST depression (0.5mm) or transient elevation
- any biomarker elevation

Score ≥3 → Invasive management and Gp 2b/3a inhibitor (intmdt or high risk)

N=1957 pts, UFH, TIMI-18 trial

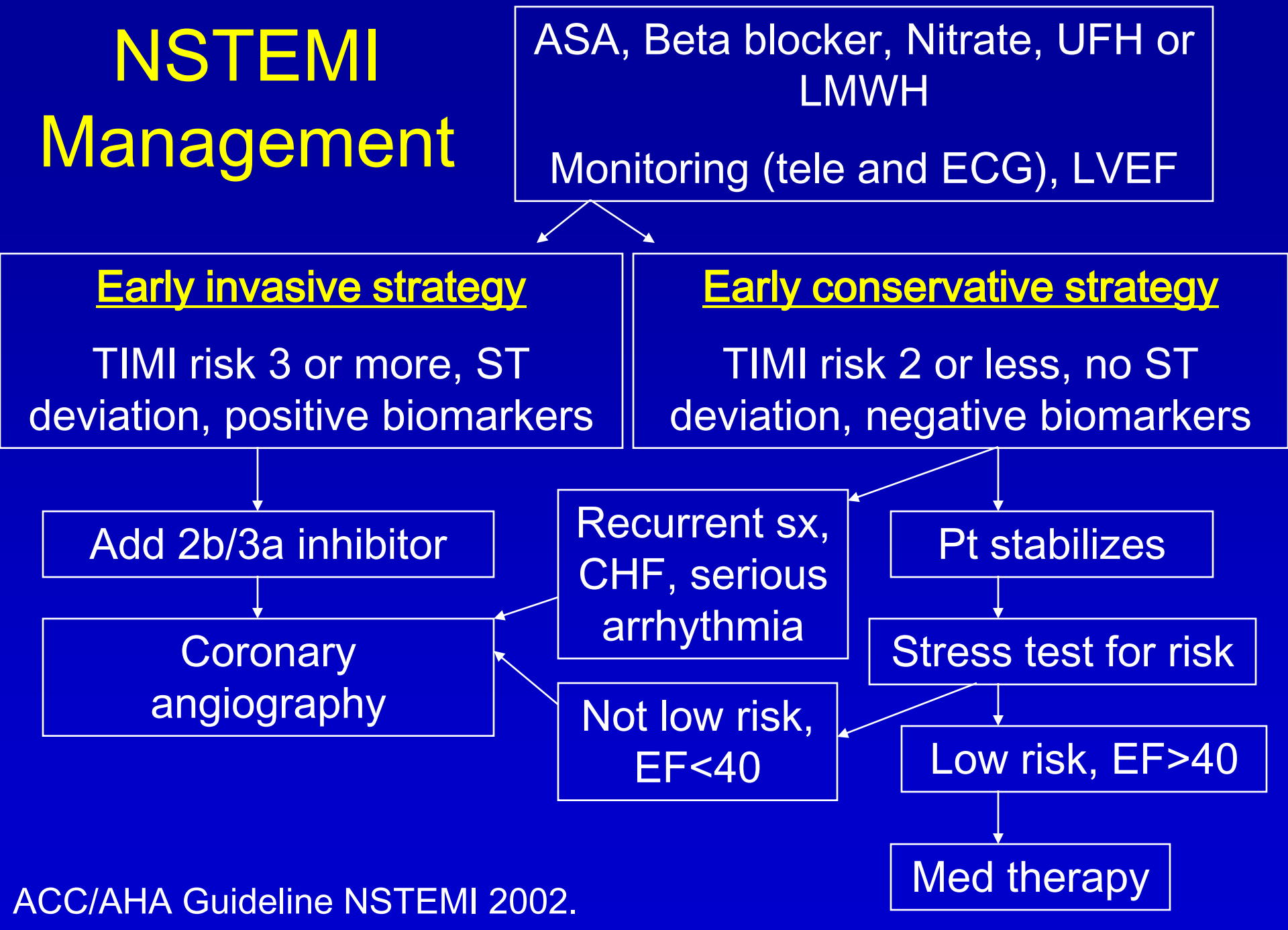


End Point 14 day:

- -Death
- -Recur MI
- -Urgent Revasc

Antman EM et al. JAMA 2000;284:835

NSTEMI Management



ACC/AHA Guideline NSTEMI 2002.

Management of NSTEMI

- NTG prn SL, then IV if recur
- MSO4 if needed
- β-blocker IV if ongoing pain, and then PO
- (Calcium blocker if β-blocker contraindicated or persistent pain)
- ACE-I for hypertension or systolic dysfunction or DM
- IABP if refractory

- ASA 162-325 then 75-160
- Clopidogrel for ASA intolerant or hypersensitivity or for 1 month if no cath or for 9 months if PCI planned
- LMWH or UFH
- 2b/3a antagonist if PCI planned (added to ASA and Heparin)

ACC/AHA Guideline NSTEMI 2002.

Summary: Treatment for MI

	STEMI	NSTEMI
Thrombolytic	Yes	No
Beta blocker	Yes	
Heparin	Yes	
Gp 2b/3a	Pre PCI	Pre PCI or high risk
ASA	Yes	
Clopidogrel	Stent or ASA allergy	
Statins	Yes	
ACE-I	HF htn low EF	
Ca blocker	Not first line, adjunct for htn or angina	
Nitrate	Yes	

STEMI and Later Cardiac Catheterization

- Cardiac catheterization for risk stratification at hospital discharge is reasonable in STEMI patients with any of the following:
  - diabetes mellitus,
  - LVEF<40,
  - heart failure,
  - prior revascularization, or
  - life-threatening arrhythmias

ACC/AHA STEMI Guidelines, 2004, p. 137

Noninvasive Strategy in NSTEMI

- Stress test in low risk with no angina or failure for 12-24 hr (intermediate without angina or failure for 2-3da)
- Catheterization if destabilizes, or if stress test is not low risk, or LVEF<40%
- LVEF somehow: Echo or MUGA if no LV gram at cath
- All NSTEMI and STEMI should receive statin therapy as inpatients unless contraindicated

ACC/AHA Guideline NSTEMI 2002.

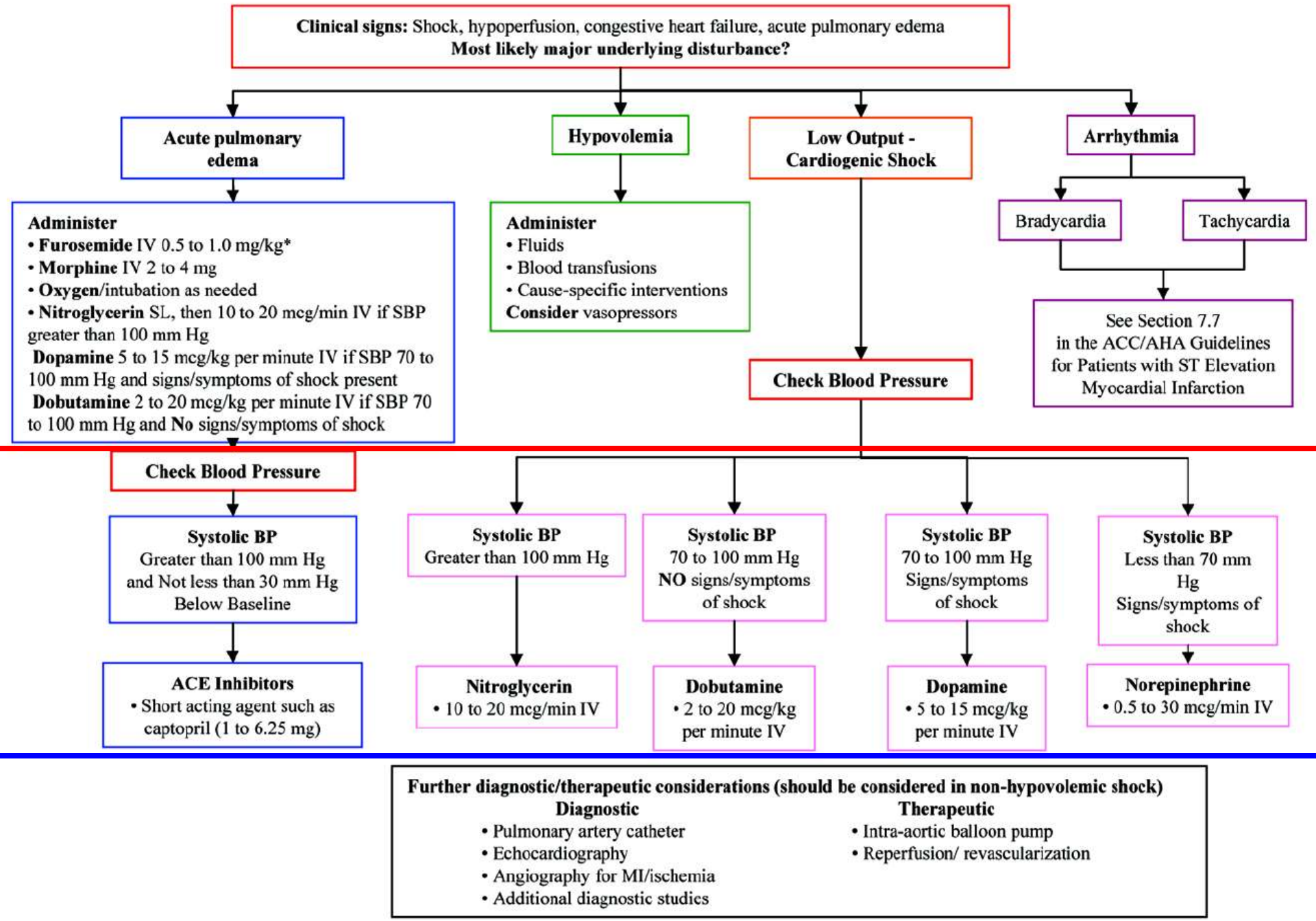
Revascularization in NSTEMI

Extent of Disease	Treatment	Class/Level of Evidence
Left main disease,* candidate for CABG	CABG	I/A
	PCI	III/C
Left main disease, not candidate for CABG	PCI	IIb/C
Three-vessel disease with EF <0.50	CABG	I/A
Multivessel disease including proximal LAD with EF <0.50 or treated diabetes	CABG or PCI	I/A IIb/B
Multivessel disease with EF >0.50 and without diabetes	PCI	I/A
One- or 2-vessel disease without proximal LAD but with large areas of myocardial ischemia or high-risk criteria on noninvasive testing (see Table 17)	CABG or PCI	I/B
One-vessel disease with proximal LAD	CABG or PCI	IIa/B†
One- or 2-vessel disease without proximal LAD with small area of ischemia or no ischemia on noninvasive testing	CABG or PCI	III/C†
Insignificant coronary stenosis	CABG or PCI	III/C

*≥50% diameter stenosis.

†Class/level of evidence I/A if severe angina persists despite medical therapy.

ACC/AHA Guideline NSTEMI 2002.



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Lipid Management in the ACS Patient

- Patient selection
  - Which patients - all
  - When to start - ASAP
- Therapeutic options
- Tailoring therapy
- Therapeutic goals

Lipids and STEMI

- Serum lipid values should be obtained on initial assessment in the emergency department along with other lab tests such as biomarkers of cardiac damage, but waiting for results should not delay treatment (FLP within 24 h of symptom onset is reliable, but LDL is significantly reduced by 48 h and may remain low for weeks)
- Treatment of lipids in the ED is not necessary
- In hospital, patients formerly on statins may be continued (if high dose and small patient who is ill, might decrease dose)
- In hospital diet should be ATP III TLC diet:
  - <7% calories as saturated fat
  - <200 mg chol/da
  - Increased consumption of omega-3 fatty acids
  - Appropriate caloric intake for energy needs
  - Encourage fruits, vegetables, soluble fiber, whole grains

ACC/AHA STEMI Guidelines, 2004, p. 30

Lipids: Secondary Prevention

- Patient education before discharge - all aspects of secondary prevention including physical activity and weight management and smoking cessation
- LDL: optional goal is <70
- If TG<200:
  - If LDL<100 use statin to lower LDL, start IN HOSPITAL
  - If LDL>100 intensify LDL therapy, preference to statins
- If TG is 200-499:
  - Goal is non-HDL substantially<130 (drug therapy for this IIa)
  - After LDL-lowering therapy, consider adding fibrate or niacin
- If TG is >500:
  - Consider fibrate or niacin before LDL-lowering therapy
  - Consider omega-3 fatty acids as adjunct
- If HDL<40: special emphasis on nonpharmacologic therapy (exercise, weight loss, smoking cessation) to increase HDL

ACC/AHA STEMI Guidelines, 2004, p. 30

Secondary Prevention ATP III Update July 2004

- Reviewed 5 trials published since original ATP III May 2001
  - HPS, PROSPER, ALLHAT-LLT, ASCOT-LLA, PROVE IT – TIMI 22
- LDL modifications (no modifications for TG or HDL):
  - Optional LDL goal <70 for very high risk*
  - Consider adding fibrate or niacin to LDL lowering drug if HDL<40 or TG>200 for high risk
  - Optional LDL goal <100 for moderately high risk (10-20%) with 30-40% LDL reduction
- *Very high risk description – Established CAD PLUS:
  - Multiple major risk factors, especially diabetes
  - Severe and poorly controlled risk factors, especially smoking
  - Multiple risk factors of the metabolic syndrome, especially TG>200 and non-HDL>130 with HDL<40
  - Acute coronary syndrome

Grundy SM et al. Circulation. 2004;110:227.

Diabetes and Acute Coronary Syndrome

- CAD accounts for 75% of all deaths in diabetics
- Of patients with ACS, 20-25% are diabetic
- In ACS patients, the diabetics have
  - More severe CAD
  - More adverse outcomes
    - Death
    - MI
    - Readmission with UA in 1 year
- Many diabetics with ACS are post CABG
- Diabetics have more non-coronary comorbidities (htn, LVH, cardiomyopathy, heart failure)

ACC/AHA ACS Guidelines, 2002, p. 65.

Diabetes and CAD

- Autonomic dysfunction
  - Occurs in 1/3 of all diabetics (1/2 if over 10 years)
  - Influences HR and BP responses
  - Raises anginal threshold
  - May predispose to LV dysfunction
- Coronary disease is less stable in diabetics
  - UA patients have more ulcerated plaques
  - UA patients have more intracoronary thrombi
- Diabetes and effects on medical therapy
  - Although beta-blockers mask hypoglycemic symptoms and may blunt a hyperglycemic response, they should be used with appropriate caution in diabetic ACS patients
  - Diuretics that cause hypokalemia may inhibit insulin release and impair glucose tolerance

ACC/AHA ACS Guidelines, 2002, p. 65.

Effects of Insulin in Diabetics with Acute MI

- Milieu of STEMI:
  - **Elevated catecholamines** with low insulin and high cortisol and glucagon levels lead to insulin resistance;
  - **Elevated FFA** worsen ischemic injury (myocellular toxicity, increased O2 demand, decreased glucose utilization)
- Insulin benefits:
  - Promotes glucose oxidation
  - Increases cellular ATP levels
  - Reduces FFA (lower lipolysis and higher glycolysis)
  - Increases cellular glucose, lactate and pyruvate uptake

ACC/AHA STEMI Guidelines, 2004, p. 78

Diabetes and ST Elevation MI in the ED

- History should include information about diabetes mellitus
  - Impaired angina (pain) recognition, especially with autonomic neuropathy
    - 50% of diabetics for >10 yr have autonomic neuropathy
  - Confusion: dyspnea, nausea, vomiting, diaphoresis can be symptoms of both MI and disturbances in DM control
  - Diabetics should be evaluated for renal dysfunction
- Laboratory should include Chem 7 (glucose) and lipid profile and magnesium and CBC and PTT and INR and biomarkers – these examinations should not delay the implementation of reperfusion

ACC/AHA STEMI Guidelines, 2004, p. 78

Diabetes and ST Elevation MI in CCU

- What about GIK for everyone?
  - GIK: glucose-insulin-potassium first used in 1962 by Demetrio Sodi-Pallares; attempt to provide energy substrate to the cells
  - High-dose: 25% glucose + 50U/L insulin + 80 mmol/L KCl at 1.5 ml/kg/h for 24h
  - Low-dose: 10% glucose + 20U/L insulin + 40 mmol/L KCl at 1.0 ml/kg/h for 24h
  - **no recommendations** yet
- Management of Glucose:
  - Insulin infusion is indicated for STEMI and complications or not
  - Target glucose 80-110 or 100-130 mg/dL (precise target glucose is not known)

ACC/AHA STEMI Guidelines, 2004, p. 78

ST Elevation MI: Long Term Glucose control in Diabetics

- Oral agents are about equally effective in lowering glucose levels
- Goal level is HbA1c of <7.0% (Class I)
- DM 2 patients are likely to need insulin to obtain goal
- Insulin and metformin is an attractive combination due to lower weight gain, lower insulin requirements and fewer hypoglycemic episodes than combination of insulin with sulfonylureas
- Metformin is contraindicated in heart failure and renal failure and should be withheld for 48 hours after iodinated contrast injection
- Thiazolidinediones should NOT be used in patients recovering from STEMI and have NYHA Class III or IV heart failure

ACC/AHA STEMI Guidelines, 2004, p. 80

Diabetes in Non ST Elevation MI or Unstable Angina (Acute Coronary Syndrome)

- Diabetes is an independent risk factor in patients with UA/NSTEMI (Class I)
- Medical treatment should be similar in diabetic and nondiabetic patients (Class I)
  - Stress testing
  - Angiography (slightly different in STEMI 2004)
  - Revascularization
- Attention should be directed toward tight glucose control (Class I)
- Patients with multivessel disease: CABG with LIMA is preferred over PCI
- PCI is indicated (Class IIa) in diabetics with 1-vessel disease and inducible ischemia
- Abciximab is indicated (Class IIa) in diabetics undergoing stenting (context of bare metal stents)

ACC/AHA ACS Guidelines, 2002, p. 61, 64-5.

Lipid and Glucose Management with Myocardial Infarction: Conclusions

- Investigate for lipid abnormalities and glucose status at presentation
- Goals for therapy are similar to those of stable coronary disease and initiation of therapy should not be delayed
  - LDL goal of <70 is now a reasonable therapeutic target (nonHDL<100)
  - HbA1c<7.0

Usual Discharge Medications

- Nitroglycerin SL PRN (tab or spray)
- Beta-blockade
- Aspirin 81-160 (stent: 160-325)
- Clopidogrel 75 for a month or year (stent, longer)
- Statin
- ACE-inhibitor (EF<40 or htn or DM)

Send-Home Messages

- Educate on all medications
  - Value and benefits, side-effects
- TLC: Diet, exercise, weight, smoking cessation
- Blood pressure control and targets
- Diabetes control and targets
- Heart failure control if present
- Rehabilitation issues – return to work
- Long term considerations: ICD