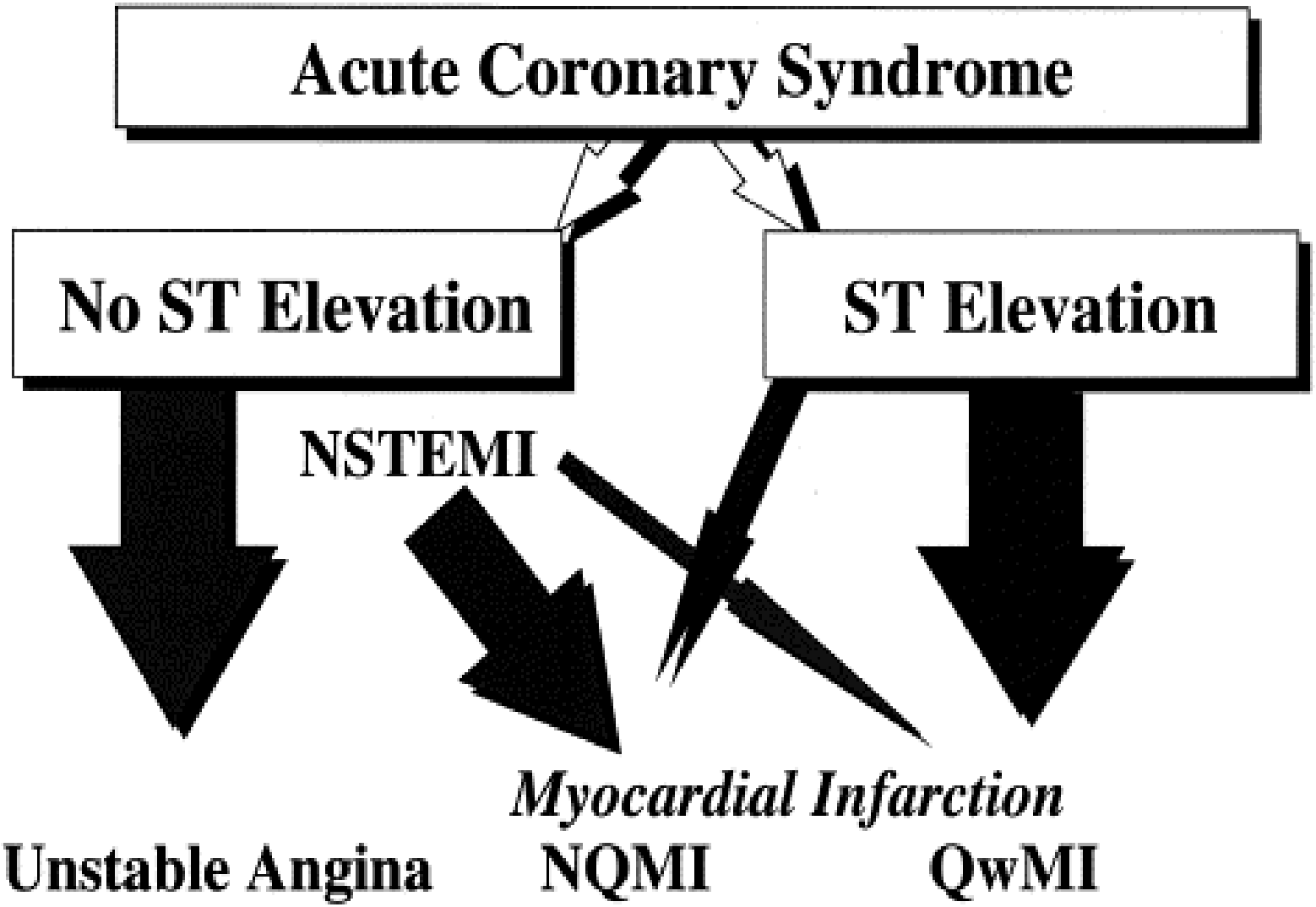
MYOCARDIAL INFARCTION


Acute Coronary Syndrome
No ST Elevation
ST Elevation
NSTEMI
Myocardial Infarction
Unstable Angina
NQMI
QwMI

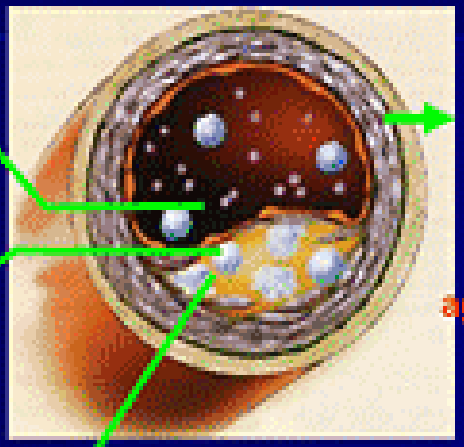
Unstable Angina

- **In patients with unstable angina，anticoagulant and antiplatelet drugs play a major role in therapy. Aggressive therapy with antilipid drugs, heparin, and antiplatelet agents is recommended.**

- **In addition, therapy with nitroglycerin and β-blockers should be considered; calcium channel blockers should be added in refractory cases.**

Myocardial Infarction is the rapid development of myocardial necrosis by a critical imbalance between oxygen supply and demand to the myocardium

Pathophysiology of Myocardial Infarction: Disrupted Plaque



Thin cap subject to enzymatic degradation

High macrophage content and activation

Mature Plaque

Large lipid core

Extracellular matrix proteins in fibrous cap

Plaque rupture

Platelet aggregation

In case of complete coronary occlusion

Spontaneous lysis, repair, and wall remodeling

In case of incomplete coronary occlusion

Acute myocardial infarction

Temporary resolution of instability Future high-risk lesion

Unstable angina or non–Q-wave myocardial infarction

Adapted from Yeghiazarians Y, et al. *N Engl J Med*. 2000;342:101–114.

Mechanisms of Myocardial damage

The severity of an MI is dependent of three factors

- The level of the occlusion in the coronary
- The length of time of the occlusion
- The presence or absence of collateral circulation

History

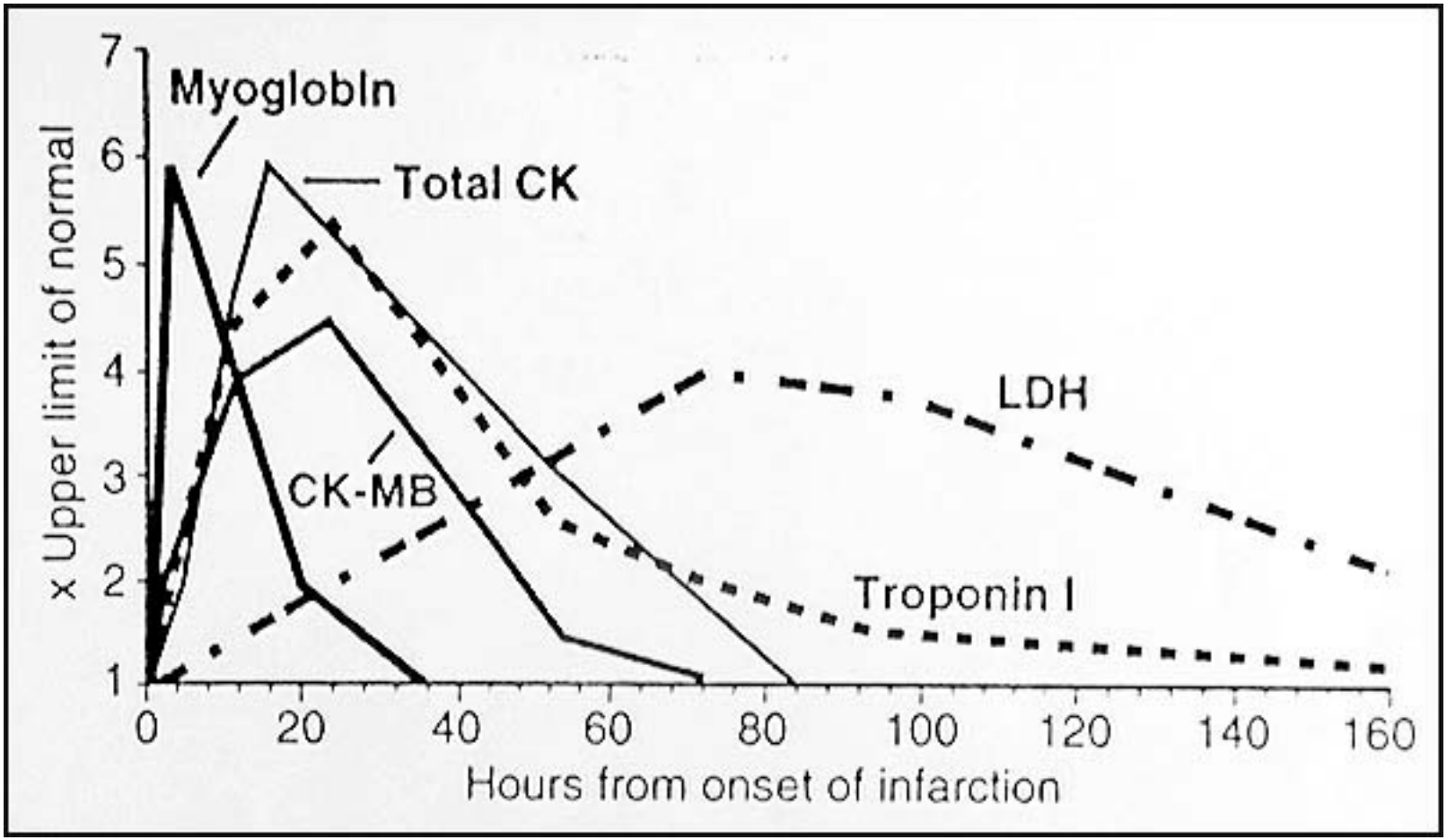
- Chest Pain- anterior precordium tightness
- Pain may radiate to jaw, neck and epigastrium
- Dyspnea-
- Anxiety
- Nausea with and without vomiting
- Diaphoresis or sweating
- Syncope or near syncope
- As many as half of MI are clinically silent

Physical Exam.

- The physical exam can often be unremarkable
- Hypertension
- Hypotension
- Acute valvular dysfunction may be present
- Rales
- Neck vein distention
- Third heart sound may be present
- A fourth heart sound poor LV compliance
- Dysrhythmias

Diagnosis:Cardiac Biomarkers

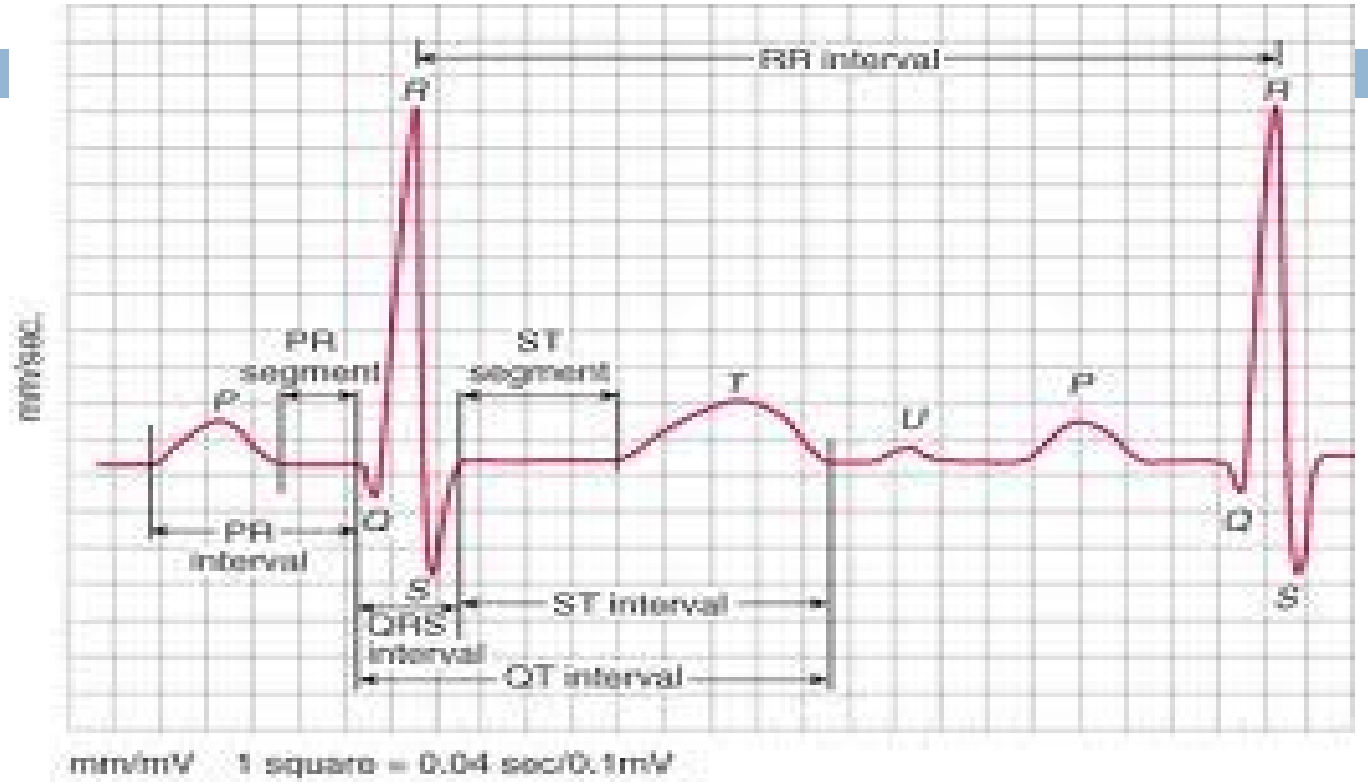
- Cardiac biomarkers are protein molecules released into the blood stream from damaged heart muscle



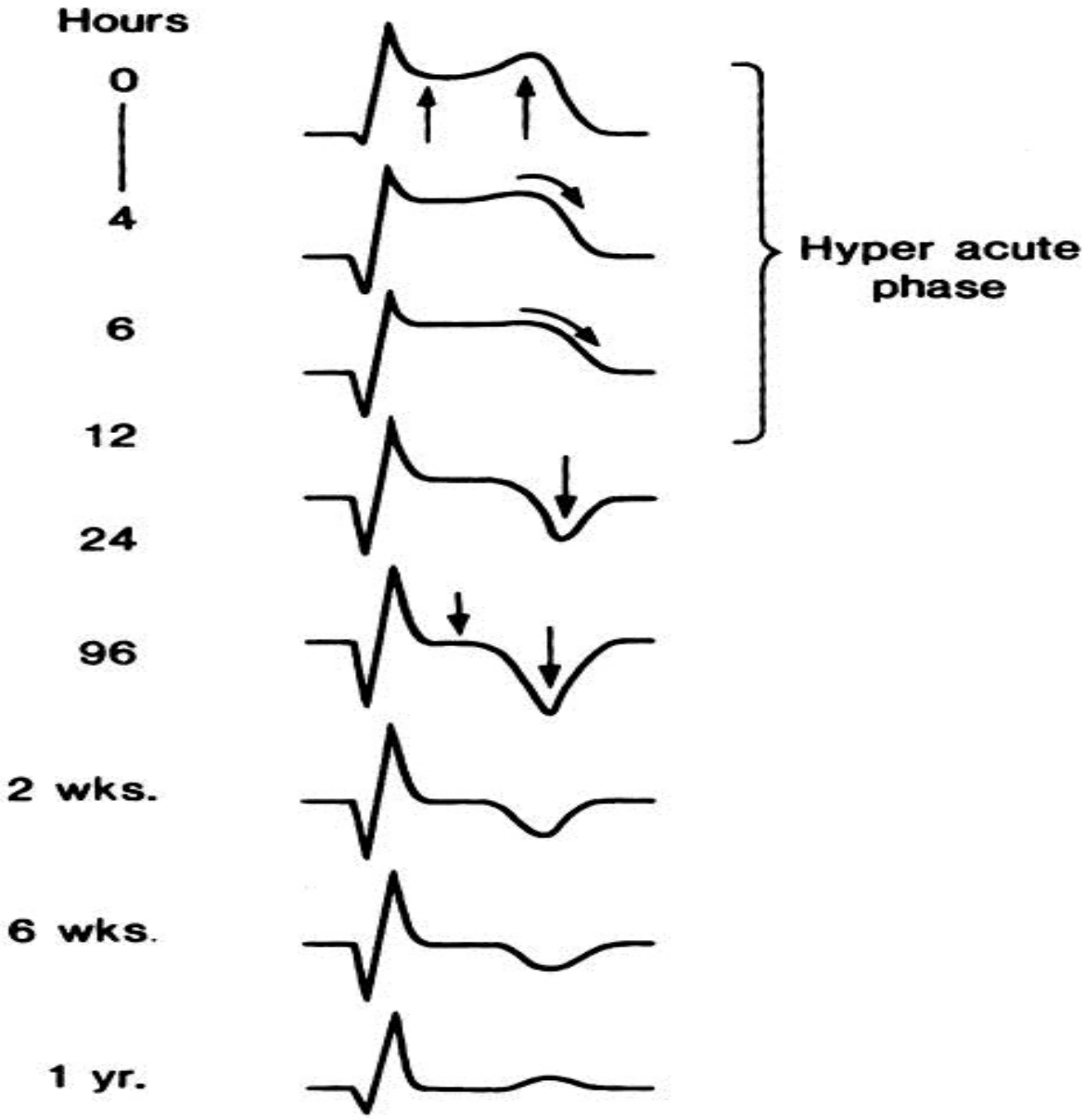
Electrocardiogram

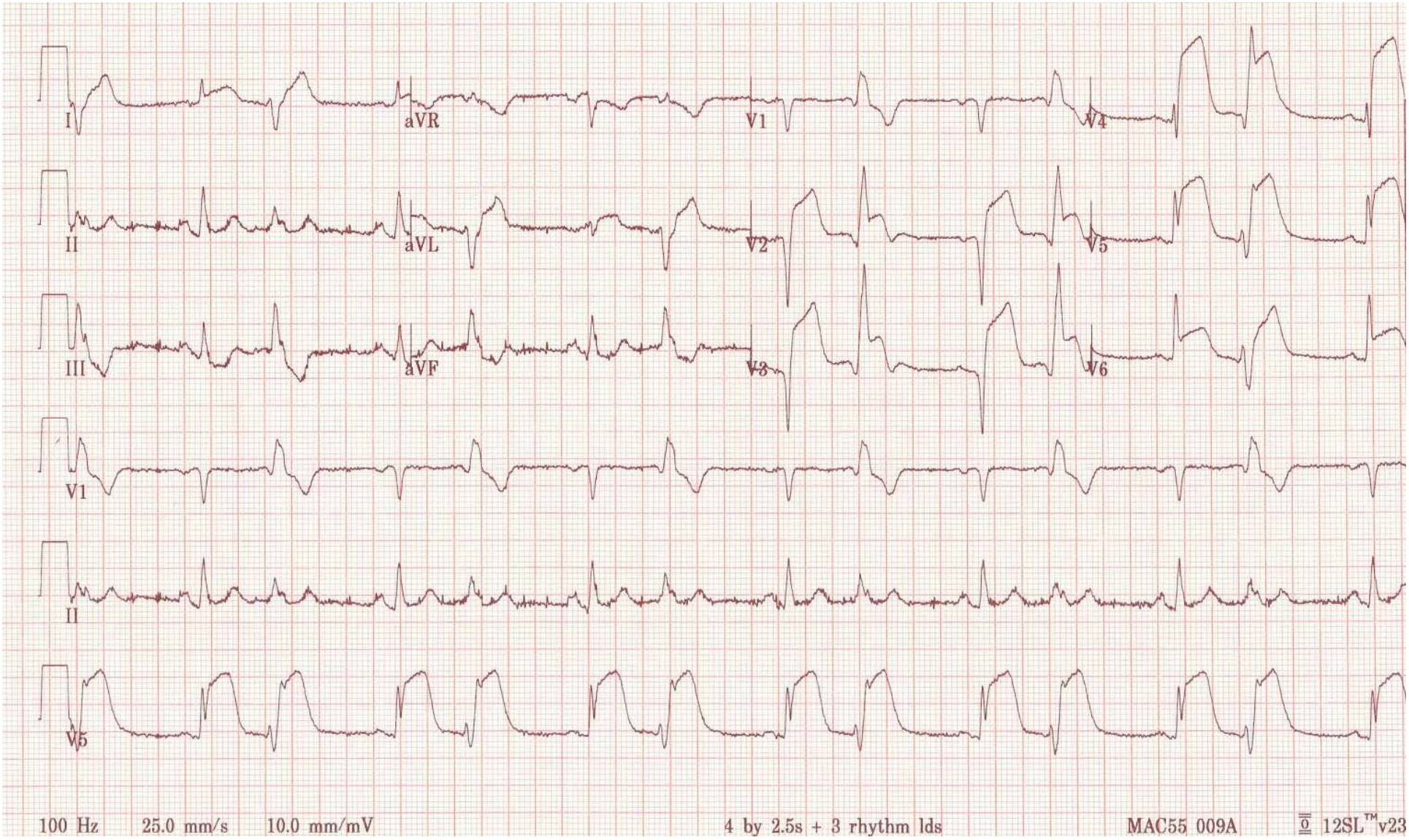
- A normal ECG does not exclude ACS
- High probability include ST segment elevation in two contiguous leads or presence of q waves
- Intermediate probability ST depression
- T wave inversions are less specific

Diagnosis


RR interval
R
R
PR
segment
ST
segment
P
T
U
P
PR
interval
Q
Q
S
S
QRS
interval
ST interval
QT interval
mm/sec
mm/mV 1 square = 0.04 sec/0.1mV

Evolution of ECG changes in Myocardial Infarction




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
100 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 3 rhythm lds
MAC55 009A
12SL™v23

Drug therapy of AMI

The goals of therapy in AMI are the expedient restoration of normal coronary flow and the maximum salvage of functional myocardium

Antiplatelet Agents

- Aspirin at least 160mg immediately
- Interferes with function of cyclooxygenase and inhibits the formation of thromboxane
- ASA alone has one of the greatest impact on the reduction of MI mortality.
- Clopidogrel, ticlopidine, have not been shown to be superior to Aspirin in acute MI

Supplemental Oxygen

- Because MI impairs the circulatory function of the heart, oxygen extraction by the heart and other tissues may be diminished

Nitrates

- IV nitrates to all patients with MI and congestive heart failure, persistent ischemia, hypertension, or large anterior wall MI
- Primary benefit: vasodilator effect
- Vasodilatation reduces myocardial oxygen demand and preload and afterload

Nitrates…

- When administered sublingually or intravenously, nitroglycerin has a rapid onset of action.
- Clinical trial data have supported the initial use of nitroglycerin for up to 48 hours in MI.

Pain Control

- Pain from MI is often intense and requires prompt and adequate analgesia. The agent of choice is morphine sulfate, given initially IV at 5 to 15 minute intervals at typical doses of 2 to 4 mg.
- Reduction in myocardial ischemia also serves to reduce pain, so oxygen therapy, nitrates, and beta blockers remain the mainstay of therapy

Thrombolytics

- Indicated with MI and ST segment elevation who present less than 12 hours but not more than 24 hours after symptom onset
- The most critical variable in achieving successful fibrinolysis is time form symptom onset to drug administration

Thrombolytics…

- As a class the plasminogen activators have been shown to restore coronary blood flow in 50-80% of patients
- Contraindication active intracranial bleeding, CVA 2months, CNS neoplasm, HTN, coagulopathy
- Intracranial bleed risk major drawback
- Eg .Streptokinase 15lakh units in 100ml NS infused IV over 1hr

Beta-blockers

- Recommended within 12 hours of MI symptoms and continued indefinitely
- Reduces Myocardial mortality by decreasing death due to arrhythmias
- Decrease the rate and force of myocardial contraction and decreases overall oxygen demand

Beta Blocker Therapy

- Metoprolol
- 15 mg IV × 1 then 200 mg/day PO in divided doses
- Atenolol
- 5-10 mg IV × 1, then 100 mg/day PO
- Carvedilol
- 6.25 mg bid titrated to 25 mg BID

Angiotensin-Converting Enzyme Inhibitors

- should be used in all patients with a STEMI without contraindications.
- Also recommended in patients with NSTEMI who have diabetes, heart failure, hypertension, or an ejection fraction less than 40%. In such patients, an ACE inhibitor should be administered within 24 hours of admission and continued indefinitely.
- Benefit of ACE inhibitor therapy can likely be extended to all patients with an MI.
-

Contraindications to ACE inhibitor use include hypotension and declining renal function. I

ACE inhibitors :Captopril

6.25 mg bid titrated to 50 mg bid started within 24 hr of MI

Lisinopril : 5 mg/day titrated to 10 mg/day

: started within 24 hr of MI

Ramipril:1.25 mg bid titrated to 5 mg bid, 3-10 days post-MI with symptoms of heart failure

Unfractionated heparin

- Forms a chemical complex with antithrombin III inactivates both free thrombin and factor Xa
- Recommended in patients with MI who undergo PTCA or fibrinolytic therapy with alteplase

Unfractionated Heparin Dosing

Loading Dose

- 60 U/kg IV bolus
- Max 5000 U if >65 kg or 4000 U if <65 kg

Maintenance Dose

- 12 U/kg/hr IV
- Max 1000 U/hr if >65 kg or 800 U/hr if <65 kg

Titration Goal

- PTT 50-70 sec

Low-molecular weight heparin

- Direct activity against factors Xa and IIa
- Proven to be effective in treating ACS that are characterized by unstable angina or non ST-elevation MI
- Their fixed doses are easy to administer and laboratory testing to measure their therapeutic effect is not necessary makes them attractive alternative of un-fractionated heparin

: Low-Molecular-Weight Heparin

- Dalteparin
- t1/2 : 3-5 hr
- 120 U/kg SC bid
- Prevention of ischemic complications in UA and NSTEMI
- Enoxaparin
- 4.5 hr
- 100 U/kg (1 mg/kg) SC q12h
- Prophylaxis of ischemic complications of UA and NSTEMI when administered with aspirin
- UA, unstable angina; NSTEMI, non−ST segment elevation myocardial infarction.

Statins

- A statin should be started in all patients with a myocardial infarction without known intolerance or adverse reaction prior to hospital discharge. Preferably, a statin would be started as soon as a patient is stabilized after presentation.
- Eg .Simvastatin 20-80mg, Pravastatin,
- Trials show benefit of starting patients on high-dose therapy from the start (e.g., atorvastatin 80 mg/day).

Long term Medications

- Most oral medications instituted in the hospital at the time of MI are continued long term
- Aspirin, beta blockers and statin are continued indefinitely
- ACEI indefinitely in patients with CHF, ejection fraction <.40, hypertension, or diabetes