Myocardial Infarction

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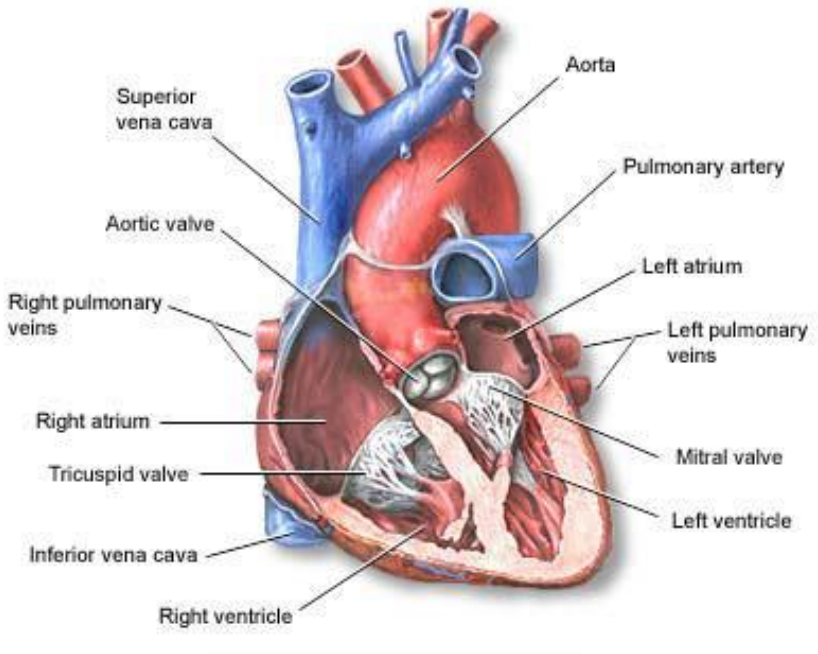
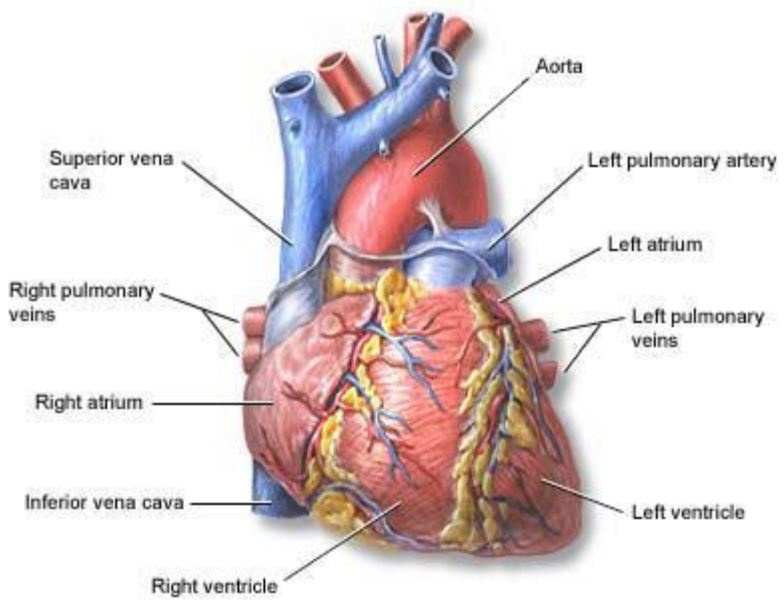
The interior of the heart is composed of valves, chambers, and associated vessels.

Definition :

A heart attack (myocardial infarction) occurs when an area of heart muscle dies or is permanently damaged because of an inadequate supply of oxygen to that area.





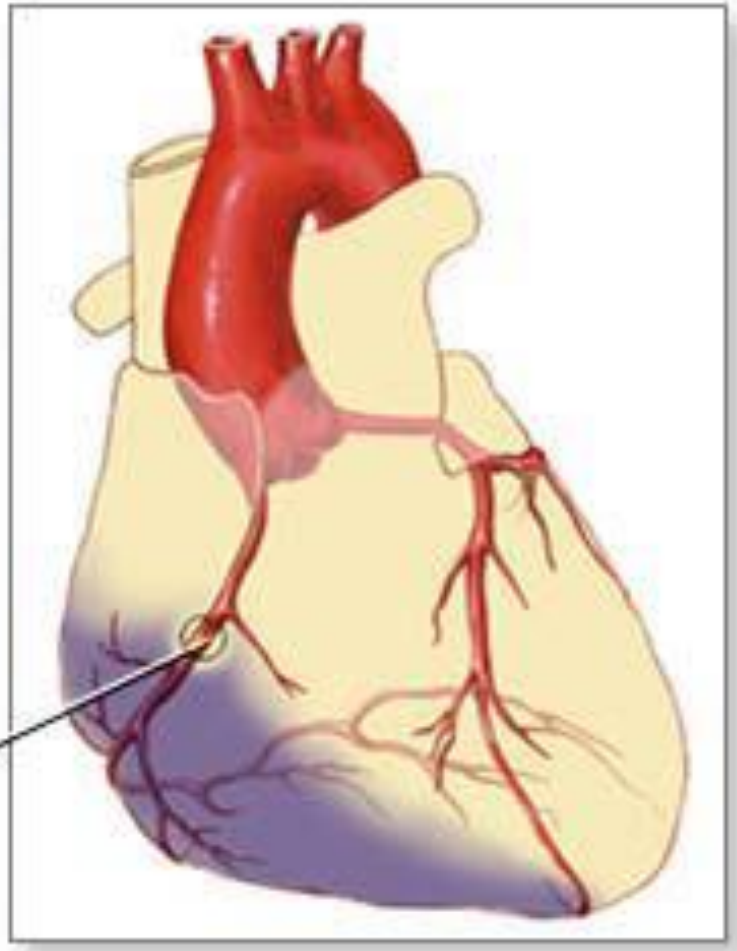
The external structures of the heart include the ventricles, atria, arteries and veins. Arteries carry blood away from the heart while veins carry blood into the heart. The vessels colored blue indicate the transport of blood with relatively low content of oxygen and high content of carbon dioxide. The vessels colored red indicate the transport of blood with relatively high content of oxygen and low content of carbon dioxide.

DEFINITION

- Myocardial infarction (MI) – ischaemic necrosis is almost always due to the formation of occlusive thrombus at the site of rupture or erosion of an atheromatous plaque in a coronary artery.
- More rarely, MI may result from prolonged vaso-spasm, inadequate myocardial blood flow or excessive metabolic demand.
- Very rarely, MI may be caused by embolic occlu-sion, vasculitis, aortic root or coronary artery dissection or aortitis.

Plaque build up in the coronary artery blocking blood flow and oxygen to the heart

Damage and death to heart tissue shown in purple



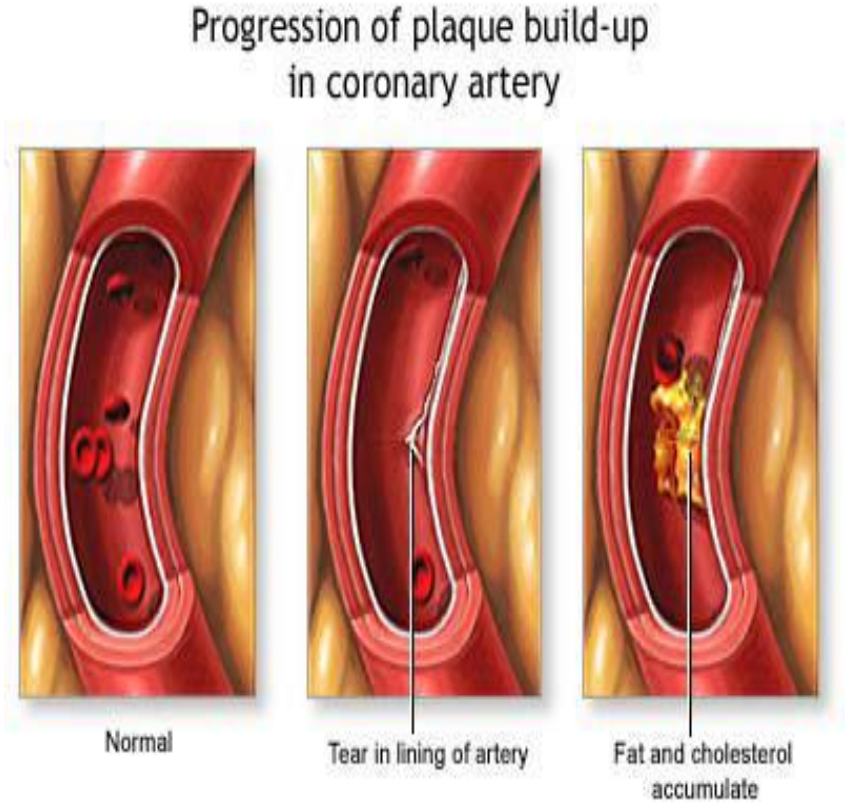
Universal definition of myocardial infarction

Excluding myocardial infarction associated with revascularization procedures

- **Detection of rise and/or fall of cardiac biomarker values (preferably troponin) with at least one value above the 99th percentile of the upper reference limit and with at least one of the following**:
  - Symptoms of ischaemia;
  - New or presumably new significant ST-T changes or new LBBB;
  - Development of pathological Q waves in the ECG;
  - Imaging evidence of new loss of viable myocardium, or new regional wall motion abnormality;
  - Identification of an intracoronary thrombus by angiography or autopsy.
- **Cardiac death with symptoms suggestive of myocardial ischaemia, and presumably new ECG changes or new LBBB, but death occurring before blood cardiac biomarkers values are released or before cardiacbiomarker values would be increased.**
- **Stent thrombosis associated with MI when detected by coronary angiography or autopsy in the setting of myocardial ischaemia and with a rise and/or fall of cardiac biomarker values with at least one value above the 99th percentile URL.**

ECG = electrocardiogram; LBBB = left bundle branch block.

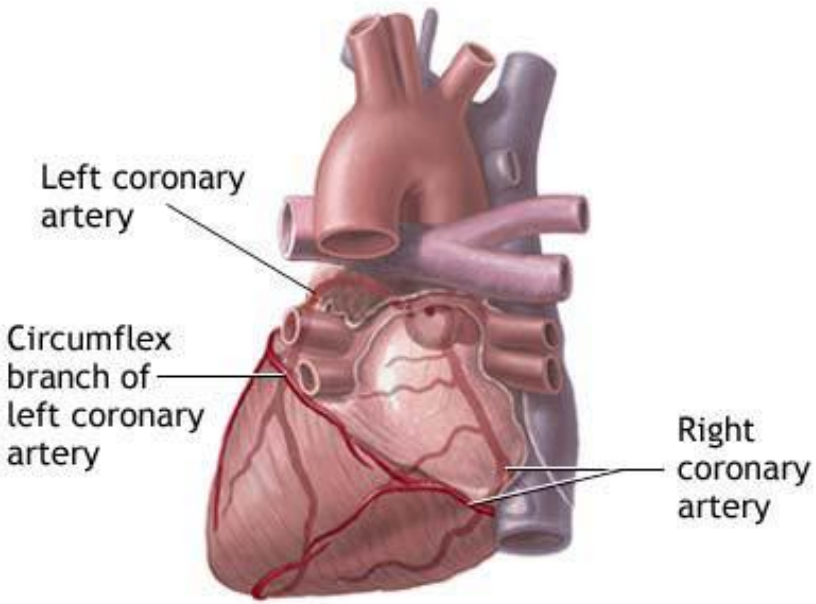
European Heart Journal (2012) 33, 2569–2619
doi:10.1093/eurheartj/ehs215



(Progressive Build-Up of Plaque in Coronary Artery)

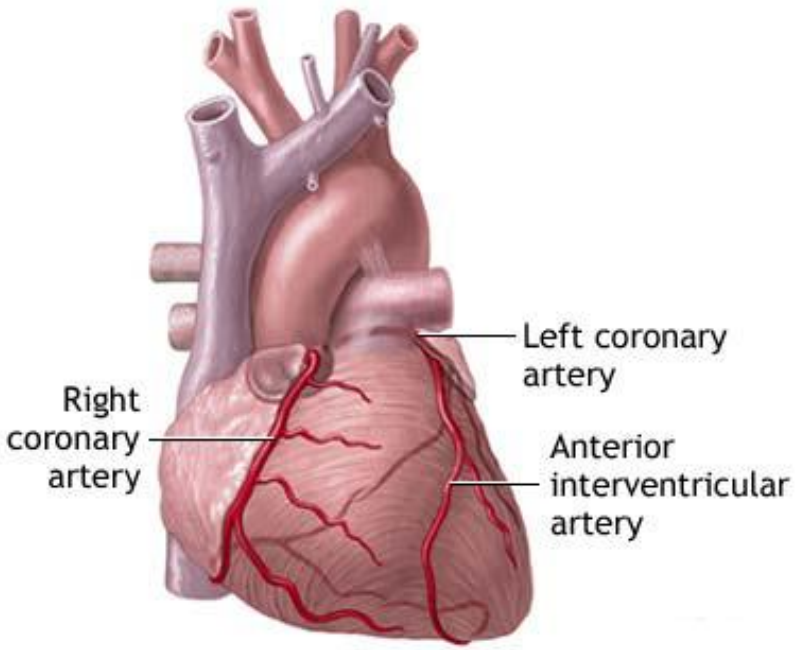
Plaque may build-up in a coronary artery at the site of a tear in the lining of the vessel.

Posterior Heart Arteries



The coronary arteries supply blood to the heart muscle. The right coronary artery supplies both the left and the right heart; the left coronary artery supplies the left heart.

Anterior Heart Arteries



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INTERNATIONAL CLASSIFICATION OF DEASESES – 10

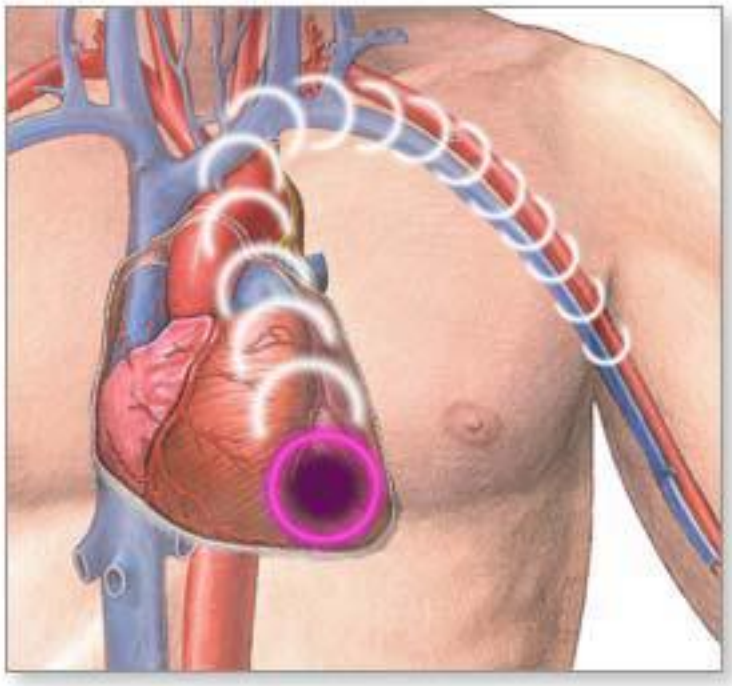
- **ACUTE MI -** WITH PATHOLOGICAL **Q-WAVE**
- **ACUTE MI** – WITHOUT PATHOLOGICAL **Q-WAVE**
- **ACUTE MI** SUBENDOCARDIAL
- **ACUTE** UNCERTAIN MI
- **RELAPSING MI** (FROM 3 to 28 DAYS)
- **REPETATIVE MI** (AFTER 28 DAYS)
- **ACUTE CORONARY FAILURE** – acute ischaemia before development of attributes of myocardial necrosis or sudden coronary death.

Classification of MI

- **TYPE 1 –** Acute coronary syndrom:primary coronary event- plaque rupture, erosion, ulceration, coronary dissection
- **TYPE 2** – Infarction secondary to oxygen supply and demand imbalance- spasm,endothelial dysfunction,left ventricule hypertrophy,anemia,hypoxemia,arrhythmia,hypotension,cocaine
- **TYPE 3 –** Cardiac arrest/ Sudden death- No biomarcer assays
- **TYPE 4a** - Infarction secondary to PCI
- **TYPE 4b -** Infarction secondary to stent thrombosis
- **TYPE 5 -** Infarction secondary to CABG

Heart Attack Symptoms

Symptoms of a possible heart attack include chest pain and pain that radiates down the shoulder and arm.



Pain radiating down arm might signal heart attack

Causes, & Risk Factors

- Most heart attacks are caused by a clot that blocks one of the coronary arteries (the blood vessels that bring blood and oxygen to the heart muscle). The clot usually forms in a coronary artery that has been previously narrowed from changes related to atherosclerosis. The atherosclerotic plaque (buildup) inside the arterial wall sometimes cracks, and this triggers the formation of a clot, also called a thrombus.

  A clot in the coronary artery interrupts the flow of blood and oxygen to the heart muscle, leading to the death of heart cells in that area. The damaged heart muscle loses its ability to contract, and the remaining heart muscle needs to compensate for that weakened area.

  Occasionally, sudden overwhelming stress can trigger a heart attack.

RISK FACTORS

- ***Nonmodifable :***

- Age (> 45)
- Male gender
- Family history (genetic predisposition)
- Aethnic origin

- ***Modifable :***
- Dyslipidaemia
- Arterial hypertension
- Smoking
- Diabetes mellitus
- Obesity
- Fatty food diet
- Physical inactivity
- Stress
- Hypoestrogenemia in female

Heart Attack Symptoms & Signs :

Chest pain behind the sternum (breastbone) is a major symptom of heart attack, but in many cases the pain may be subtle or even completely absent (called a "silent heart attack"), especially in the elderly and diabetics. Often, the pain radiates from the chest to the arms or shoulder; neck, teeth, or jaw; abdomen or back. Sometimes, the pain is only felt in one these other locations.

The pain typically lasts longer than 20 minutes and is generally not fully relieved by rest or nitrioglycerine, both of which can clear pain from angina.

PRESENTATION (urgent diagnosis)

- Sudden intensity chest pain usually similar in nature to angina, but of greater severity, longer duration (>20 min) and not relieved by nitroglicerin.
- Unusual,intensive, prolonged pain which located on arms,in epigastrium,in low jaw, in back.
- Sudden appearance of severe disturbances of rhythm or acute heart failure.
- Sudden, acute change for the worse of the patient condition which associated with hypotension .
- Acute appearance of the new left bundle branch block of His (LBBB).

HEART ATTACK SYMPTOMS

The pain can be intense and severe or quite subtle and confusing. It can feel like:

- **squeezing or heavy pressure**
- **a tight band on the chest**
- **"an elephant sitting on the chest"**
- **bad indigestion**
- ***Other symptoms you may have either alone or along with chest pain include:***
- **Shortness of breath**
- **Cough**
- **Lightheadedness - dizziness**
- **Fainting**
- **Nausea or vomiting**
- **Sweating, which may be profuse**
- **Feeling of "impending doom"**
- **Anxiety**

Variants of AMI clinical course

- **Anginous** – typical (70-90%);
- **Asthmatic** – cardiac asthma and pulmonary oedema-like type (10 %);
- **Abdominal** – stomach-ache, dyspepsia;
- **Arrhythmic** – sudden development of impaired rhythm and conductibility;
- **Cerebrovascular** – fainting, loss of consciousness, acute impairment of cerebral blood flow;
- **AMI with atypical pain syndrome** – pain in the jaw, back, arm, the right side of chest;
- **Painless AMI** – diagnosed by ECG.

Clinical course of MI

- **Latent period** till 28 days (in which presenting features includes signs of unstable angina pectoris)
- **Superacute period** from 30 min till 2 hours (time from appearance of acute ischaemia till first signs of myocardial necrosis)
- **Acute period** till 10 to 14 days (occur after 2-3 hours and prolonged till final formation of focal necrosis and complicated by resorbtion of necrosis products with increases cardiospeciphic enzymes)
- **Subacute period** till 4-8 weeks (time from limitation of focal necrosis till substitution of primary connective tissue)
- Postmyocardial infarction period from 3-6 monthes (final formation of scar and named by **cardiosclerosis**).

Criterias of diagnosis

- Typical clinical signs (combination of history)
- Typical ECG changes (Q-wave, ST-segment, T-wave)
- Biochemical markers of cardiac injury (myoglobin,CK,CK-MB,troponins T and I)

DIAGNOSIS OF MI

- The diagnosis is based on thorough analysis of **clinical manifestations, ECG, and necrosis marker levels;**
- ECG should be taken **within the first 10 minutes** since a physician sees a patient and is repeated 6 and 24 hours later;
- The level of **troponins T and I** should be determined **within 60 minutes** since admission to hospital and is repeated 6 and 12 hours later in case of negative test results;

Heart Attack Diagnosis & Tests :

During a physical examination, the doctor will usually note a rapid pulse. Blood pressure may be normal, high, or low. While listening to the chest with a stethoscope, the doctor may hear crackles in the lungs, a heart murmur, or other abnormal sounds.

The following tests may reveal a heart attack and the extent of heart damage:

- Electrocardiogram(ECG) -- single or repeated over several hours
- Echocardiography
- Coronary angiography
- Nuclear ventriculography (MUGA or RNV)

- *The following tests may show the by-products of heart damage and factors indicating you have a high risk for heart attack*:
- Troponin I and troponin T
- CK and CK-MB
- Serum myoglobin

The ECG in acute myocardial infarction (MI)

Acute MI may cause changes in the QRS complex, ST segment or the T wave. However, the only definitive diagnostic changes of myocardial infarction are changes in the QRS complex.

The QRS complex in infarction

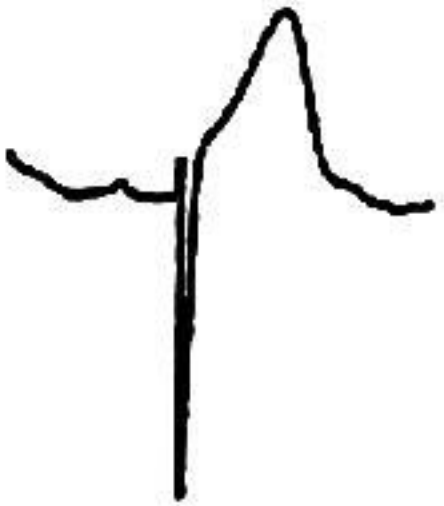
Two types of QRS abnormalities may indicate infarction:
1) Inappropriately low R wave voltage in a local area and
2) Abnormal Q waves

The above two abnormalities are actually part of the same process - i.e. the development of a negative Q wave and the reduction in size of the positive wave.

The loss of positivity is the result of myocardial necrosis beneath the exploring electrode. The size of the positive wave in each precordial lead is related to the thickness of viable myocardium underneath that electrode.

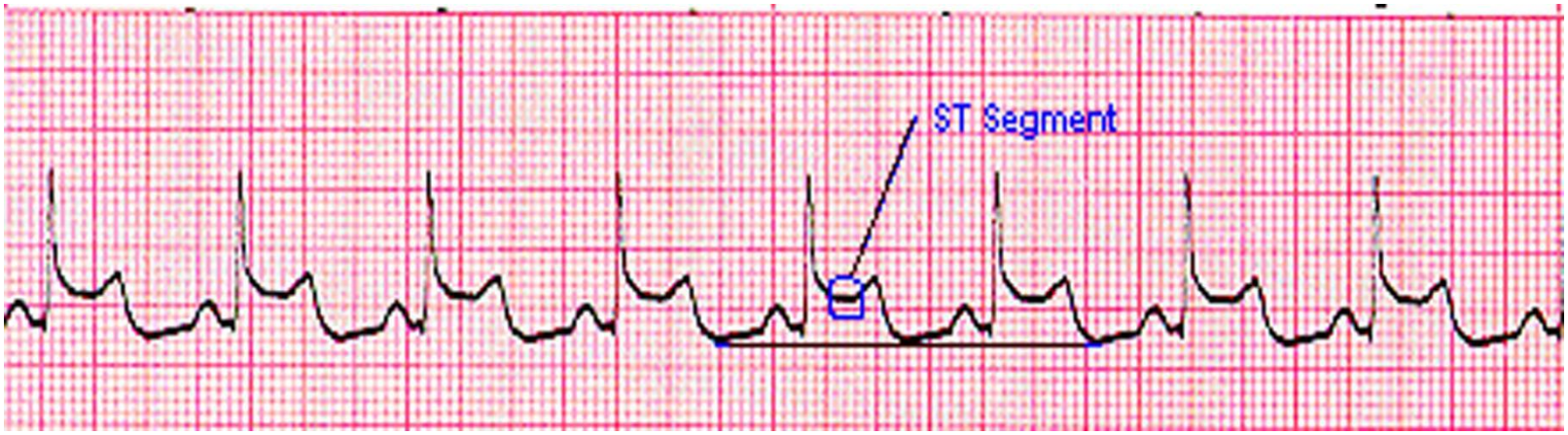
The ECG and Myocardial Infarction

- During an MI, the ECG goes through a series of abnormalities. The initial abnormality is called a **hyperacute T wave**. This is a T wave that is taller and more pointed than the normal T wave.



The ECG and Myocardial Infarction

The abnormality lasts for a very short time, and then elevation of the ST segment occurs. This is the hallmark abnormality of an acute MI. It occurs when the heart muscle is being injured by a lack of blood flow and oxygen and is also called a *current of injury*.



leads V2 to V5.

- **Abnormal Q waves and QS complexes**

Therefore, the four possible QRS changes indicative of infarction are:
1) Reduced R wave voltage (confirmed by previous ECGs)
2) Abnormal Q waves without any conclusive evidence of R wave reduction
3) Reduced R wave voltage in association with abnormal Q waves and
4) QS complexes.

In a **transmural infarction** (endocardium to epicardium), there will be **total loss of R waves** in leads overlying the infracted zone. This gives rise to entirely negative waves - i.e. **QS complexes**. These negative waves are the result of depolarisation of the posterior wall of the ventricle travelling from endocardium to epicardium (i.e. away from the anterior leads).

These four changes represent increasing thickness of infarction as part of a common process. A combination of these findings is seen in an infarction of non-uniform thickness.

The reduction in R wave voltage can only be confirmed if either a previous ECG shows a significantly greater R wave height in the appropriate leads before the infarction occurred, or the leads involved are two or more of the leads V2 to V5.

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- **Abnormal Q waves**

  Q waves may be recognised to be abnormal because of:
  1) Abnormal width (duration) - i.e. Q wave = 0.04 s or
  2) Abnormal depth (relative to the following R wave) - i.e. depth of Q wave >25% of the height of the following R wave is abnormal.

  ST segment changes in myocardial infarction

  Dramatic ST segment changes occur in the early stages of myocardial infarction. Such changes indicate **myocardial injury rather than infarction**.

  The injury state is unstable, and acute ST segment elevation **always** resolves to some extent and **usually** resolves completely. The resolution of the acute ST elevation is **usually** accompanied by development of the QRS changes of frank infarction, although **occasionally**, it may resolve without the development of diagnostic changes of infarction.

  The ST segment shift is produced by myocardial injury, which causes a disturbance in the current flow across the cell membrane.

Primary ST segment depression is seen in leads facing the infarct when a subendocardial infarction occurs.

The essential change of myocardial injury is ST segment elevation above the isoelectric line.

T wave changes of infarction

The normal ST segment does not deviate by more than 1 mm above or below the isoelectric line.

The spectrum of changes in the T waves during infarction includes flattening of the T waves, bi-phasic T waves, inverted T waves and abnormally tall T waves.

Abnormal ST segment elevation occurs in leads facing the infarction, both in transmural and subepicardial infarction. Reciprocal ST segment depression may be seen at the same time as the above primary changes in leads recording from positions opposite to the infarct.

The most typical T wave change in acute MI is deep, symmetrical T wave inversion.

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T wave changes of infarction

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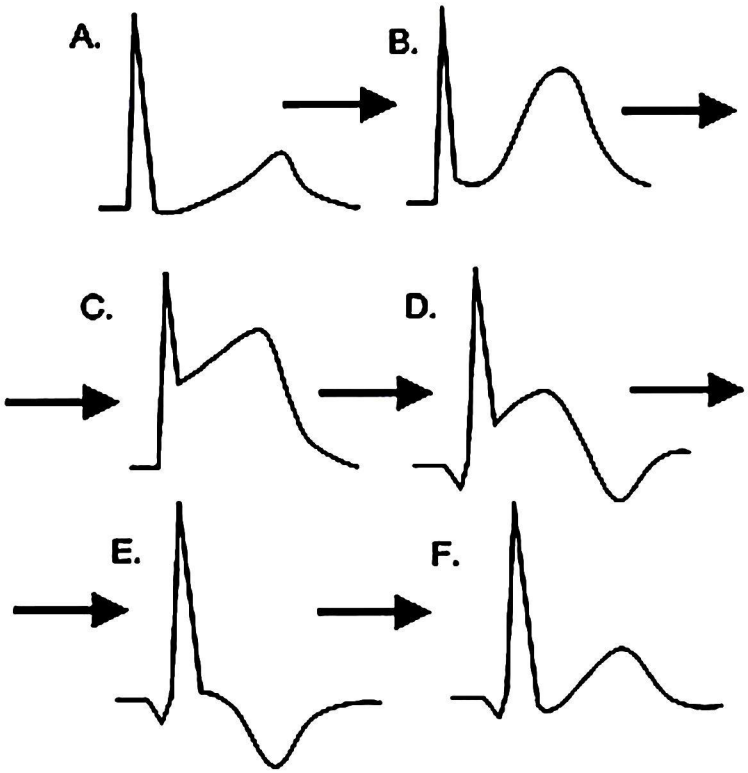
Sequence of changes in acute MI



Evolution of Acute MI

A) Shows the normal QRS complex in a lead.

B & C) Within **hours** of the clinical onset of an MI, there is **ST segment elevation**. At this stage no QRS or T wave changes have occurred. This indicates myocardial damage only, not definitive evidence of infarction.

D) Within **days**, the R wave voltage falls and abnormal Q waves appear. This is sufficient evidence of an infarction. In addition, T wave inversion will also have appeared but the ST segment elevation may be less obvious than before.

E) Within **one or more weeks**, the ST segment changes revert completely to normal. The R wave voltage remains low and the abnormal Q waves persist. Deep, symmetrical T wave inversion may develop at this stage.

F) Months after the MI, the T waves may gradually return to normal. The abnormal Q waves and reduced R wave voltage persist.

Occasionally, all evidence of infarction may be lost with the passing of time; this is due to shrinkage of scar tissue.

Left Circumflex Artery or Right Coronary Artery

An ECG can not only tell you if an MI is present but can also show the approximate location of the heart attack, and often which artery is involved. When the ECG abnormalities mentioned above occur, then the MI can be localized to a certain region of the heart. For example, see the table below:

ECG leads	Location of MI	**Coronary Artery**
II, III, aVF	Inferior MI	Right Coronary Artery
V1-V4	Anterior or Anteroseptal MI	Left Anterior Descending Artery
V5-V6, I,aVL	Lateral MI	Left Circumflex Artery
ST depression in V1, V2	Posterior M	Left Circumflex Artery or Right Coronary Artery

Location of changes in MI

- Because primary ECG changes occur in leads overlying the infarct, the location of an infarct can be derived by looking at the primary changes occurring in such leads. This is depicted in the following table:

Location of infarction	Leads showing primary changes
	Typical changes
Anterior infarction	
Antero-septal	V1, V2, V3
Anterior	Some of V1-V3 plus some of V4-V6
Anterior extensive	V1, V2, V3, V4, V5, V6,I, aVL
Antero-lateral	V4, V5, V6, I, aVL, possibly II
High lateral	aVL and/or I
Inferior infarction	
Inferior	II, III, aVF
Infero-lateral (= apical)	II, III, aVF, V5, V6 & sometimes also I, aVL
Infero-septal	II, III, aVF, V1, V2, V3
	Other changes
Posterior infarction	V1, V2 (inverse of usual changes elsewhere)
Subendocardial infarction	Any lead (usually multiple leads)

Diagnostic criteria for MI

A definitive diagnosis of MI from the ECG can only be made on the basis of abnormalities in the QRS complex. The following changes are seen:

1) q waves which are either 0.04 s or longer in duration (excluding aVR and lead III) or have a depth which is more than 25% of the height of the following R wave (excluding aVR and lead III).
2) qs or QS complexes (excluding aVR and lead III).
3) Local area of inappropriately low R wave voltage.

Additional changes frequently associated with MI are:

a) ST segment elevation (convex upwards) in leads facing the infarcted zone.
b) ST segment depression occurs as a reciprocal change in leads mutually opposite to the primary leads showing evidence of infarction.
c) Horizontal ST segment depression may occur as a primary change in subendocardial infarction.

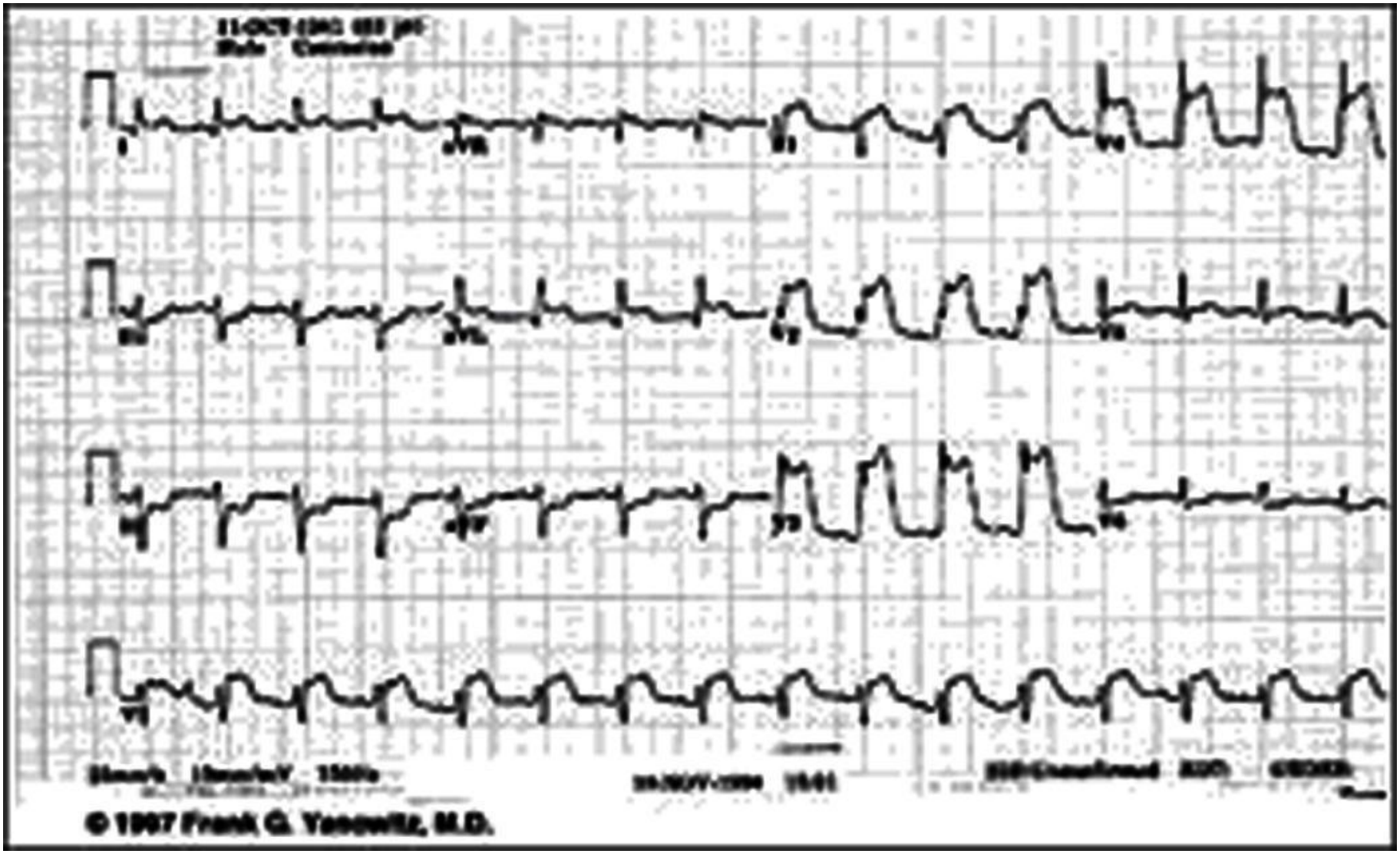
Atypical ECG presentations that deserve promt management in patients with signs and symptoms of ischemia

- LBBB.
- Ventricular paced rhythm.
- Patients without diagnostic ST-segment elevation but with persistent ischaemic symptoms.
- Isolated posterior myocardial infarction.
- ST-segment elevation in lead aVR.

ECG = electrocardiogram; LBBB = left bundle branch block.

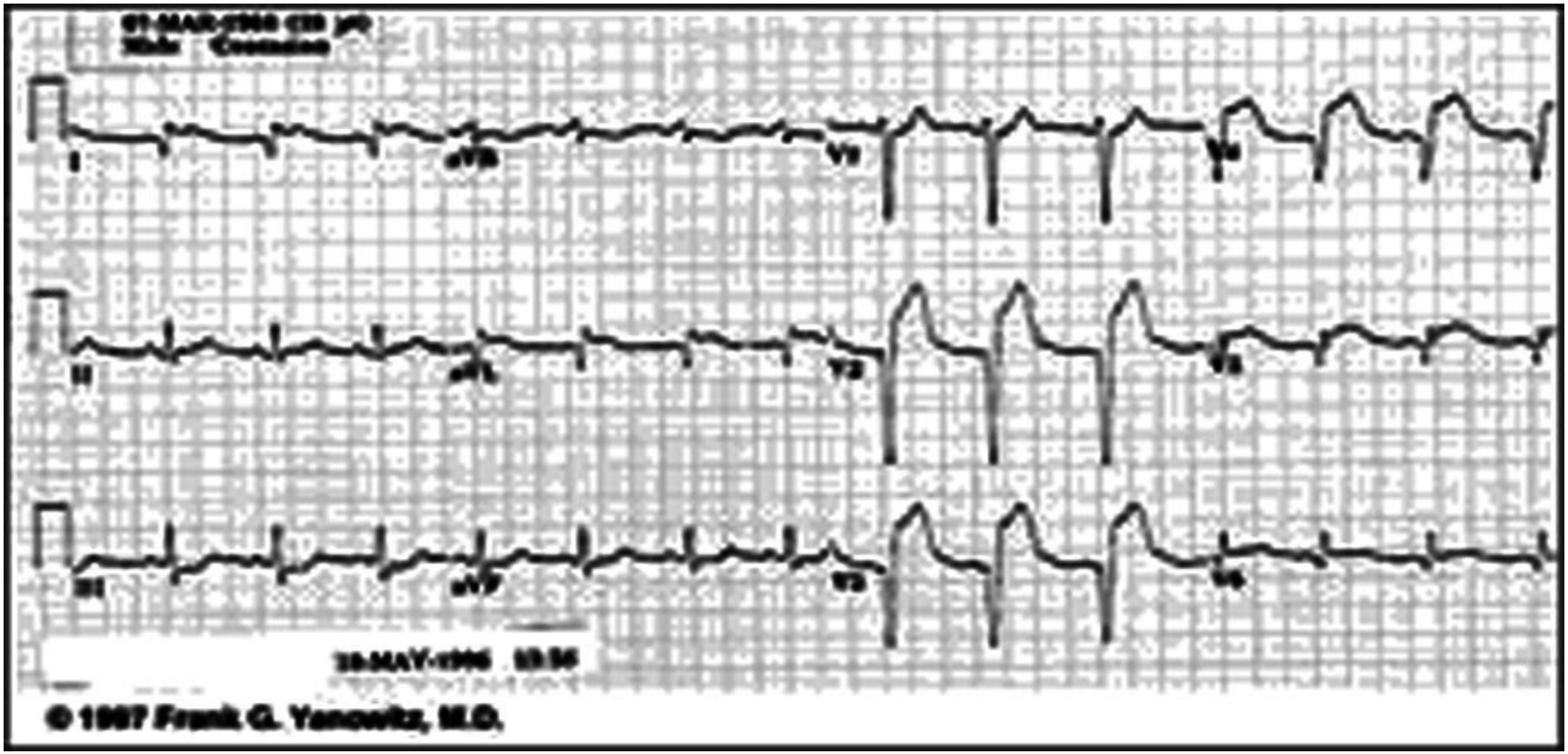
European Heart Journal (2012) 33, 2569–2619
doi:10.1093/eurheartj/ehs215

Acute anterior MI

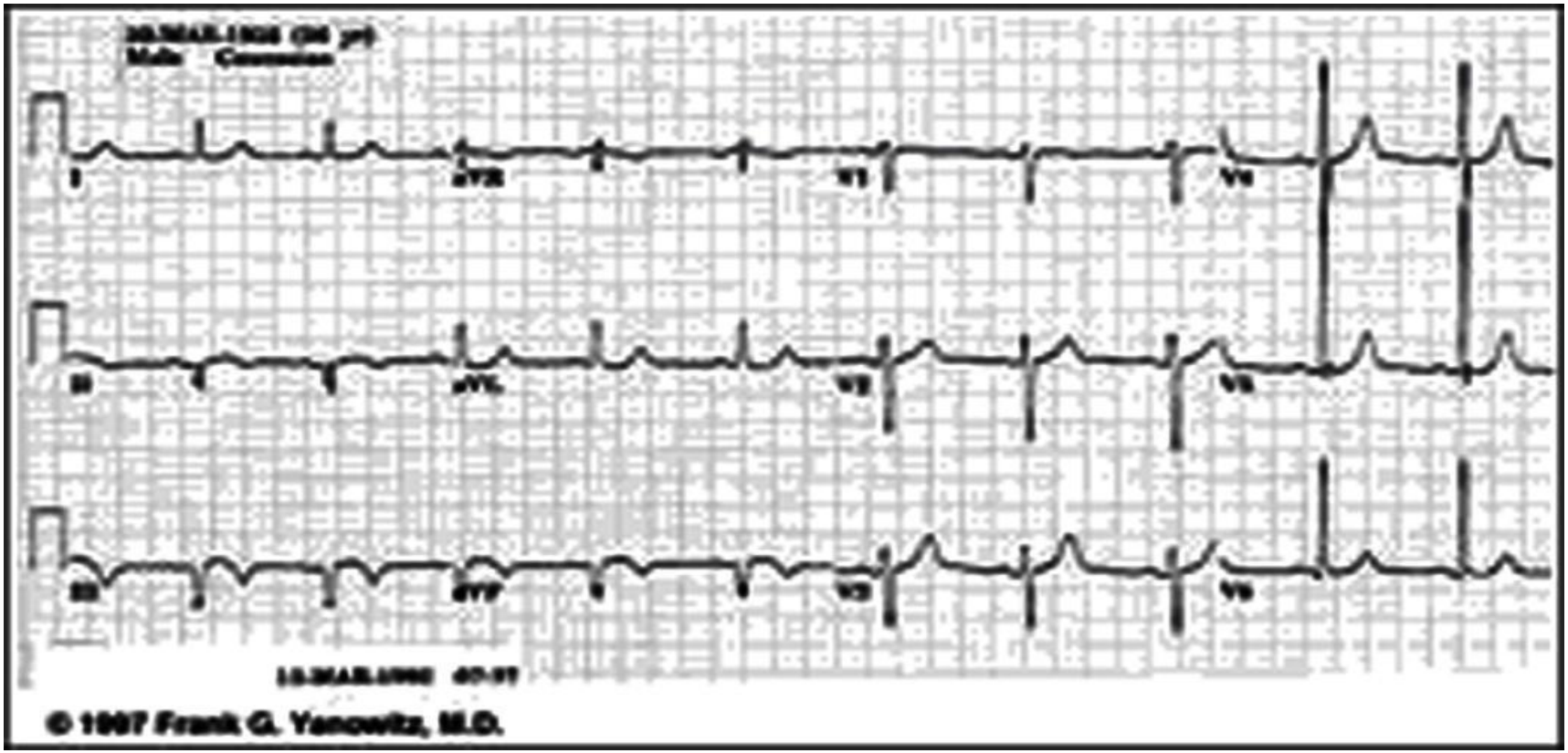


Extensive anterior/antero-lateral MI

Significant pathological Q waves (V2-6, I, aVL) plus marked ST segment elevation are evidence for this large anterior/antero-lateral MI. The exact age of the infarction cannot be determined without clinical correlation and previous ECGs, but this is likely to be a recent MI.



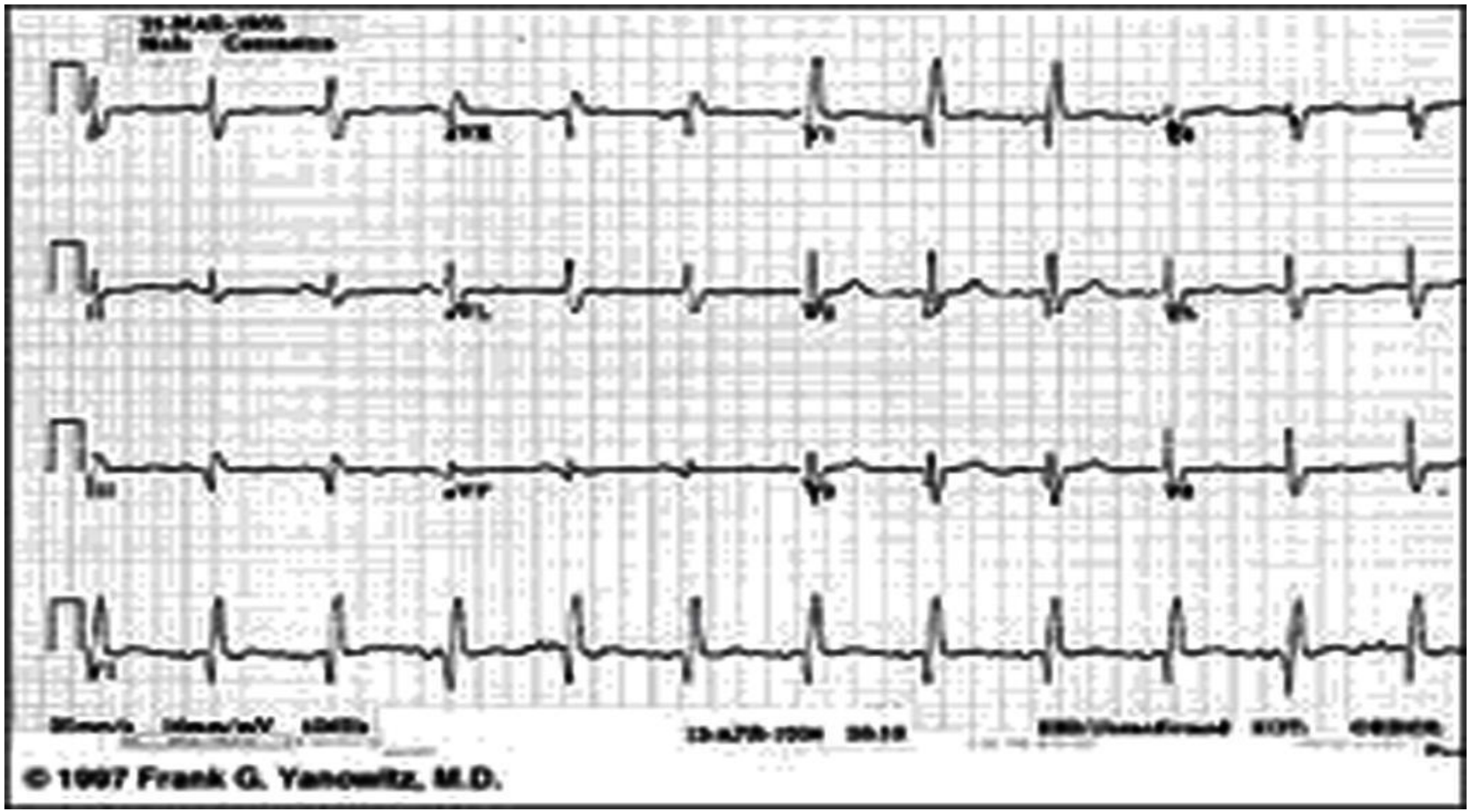
Inferior MI: Fully evolved



Significant pathological Q waves are seen in leads II, III and aVF along with resolving ST segment elevation and symetrical T wave inversion. This is a classic inferior MI.

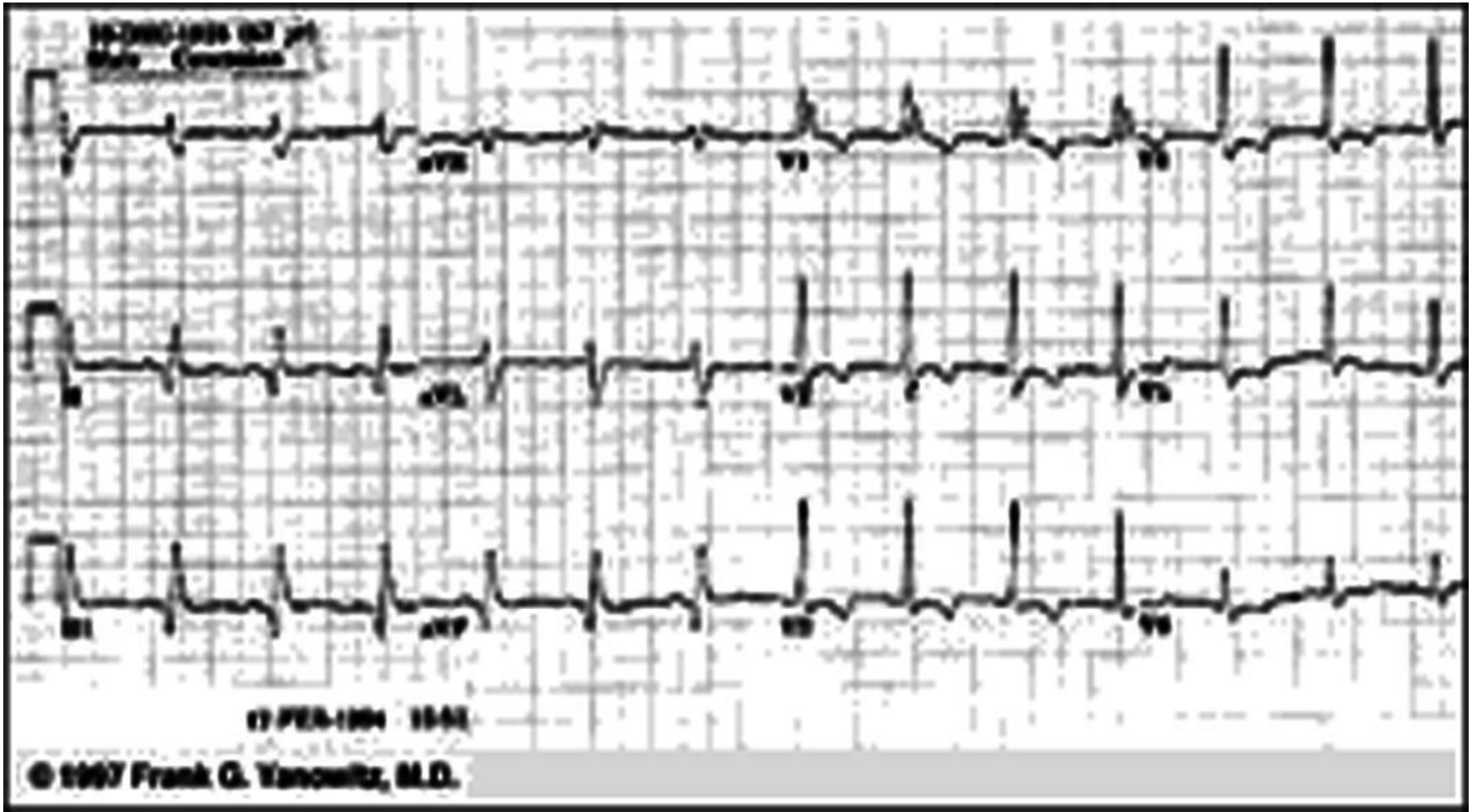
Inferior & antero-septal MI + RBBB

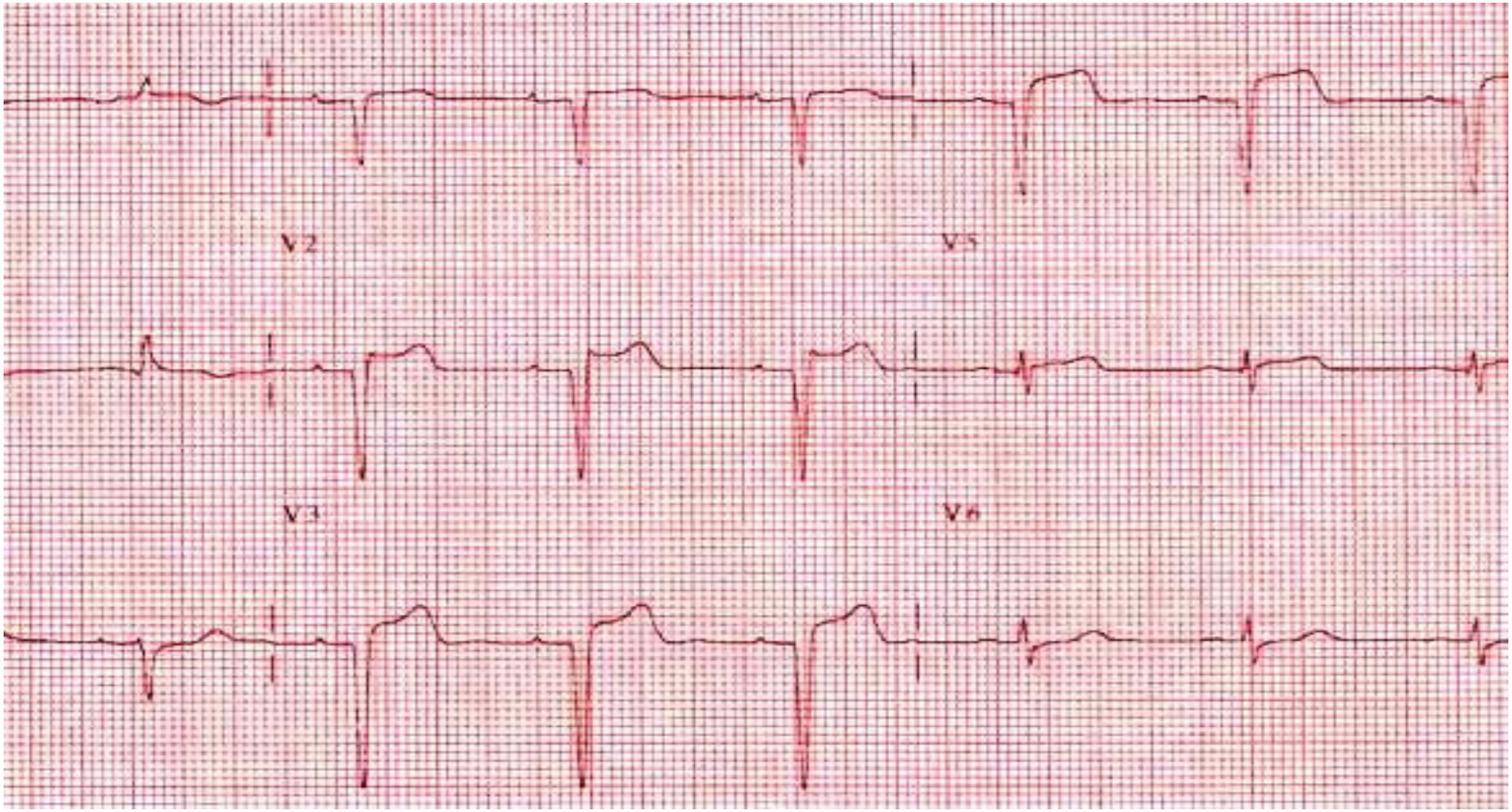
Pathological Q waves are seen in leads II, III, aVF (inferior MI) and in leads V1-3 (antero-septal MI). RBBB is recognised by the wide QRS (>0.12 s) and the anterior/rightwards orientation of terminal QRS forces. When an antero-septal MI complicates RBBB (or vice versa), the rSR' complex in V1 (typical of RBBB) becomes a qR complex.



Infero-posterior MI with RBBB

This is an unusual RBBB because the initial R wave is taller than the R' wave in lead V1. This is the clue for true posterior MI. The tall initial R wave in V1 is a "pathological R" wave analagous to the "pathological Q" wave of an anterior MI.

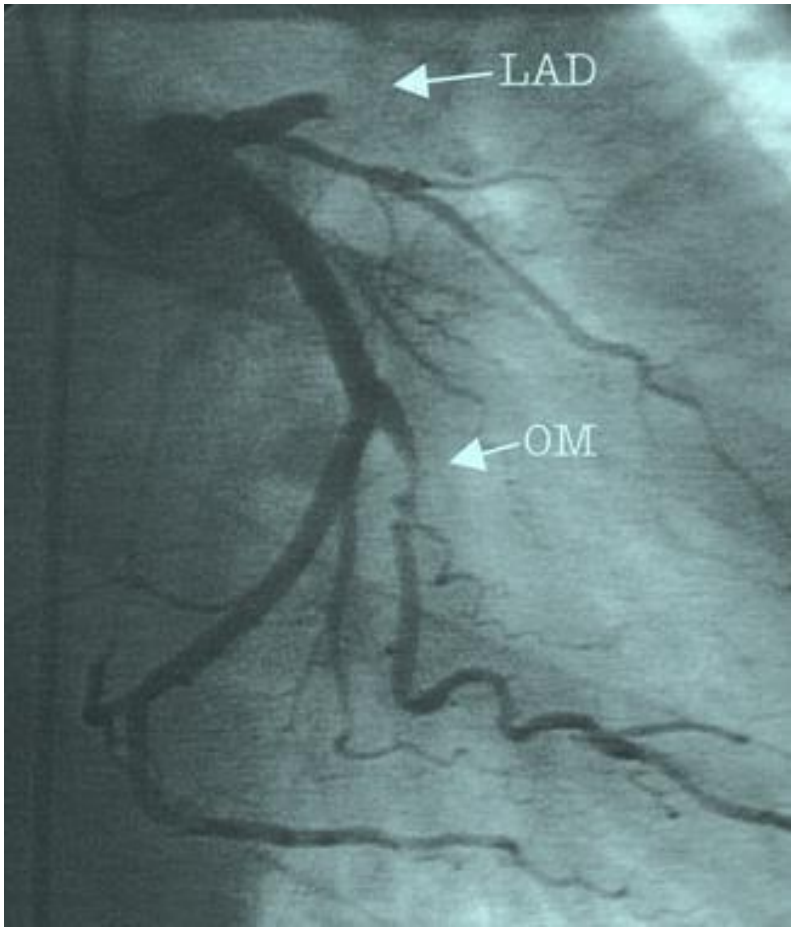




- *Figure : A twelve-lead electrocardiogram (ECG), recorded on admission to Cardiac Care Unit, showing recent extensive anterior wall myocardial infarction.*

  Coronary angiography revealed total occlusion of the proximal segment of left anterior descending artery, and severe disease involving the proximal segment of the obtuse marginal branch (Figure 3). He was treated with direct Percutaneous Coronary Intervention (PCI).

Coronaroangiography



- *Figure : Left coronary artery angiograms showing total occlusion of the left anterior descending artery (LAD) and subtotal occlusion of the obtuse marginal (OM) branch of the left circumflex coronary artery.*

Heart Attack Treatment

- A heart attack is a medical emergency! Hospitalization is required and, possibly, intensive care. Continuous ECG monitoring is started immediately, because life-threatening arrhythmias are the leading cause of death in the first few hours of a heart attack.

  The goals of treatment are to stop the progression of the heart attack, to reduce the demands on the heart so that it can heal, and to prevent complications.

  An intravenous line will be inserted to administer medications and fluids. Various monitoring devices may be necessary. A urinary catheter may be inserted to closely monitor fluid status.

  Oxygen is usually given, even if blood oxygen levels are normal. This makes oxygen readily available to the tissues of the body and reduces the workload of the heart.

Management of Patients with non-ST-elevation AMI in the prehospital setting

- **Calling an ambulance**
- **Clinical death** – cardiopulmonary resuscitation
- **To calm down, to provide fresh air, to seat**
- **Nitroglycerin 0.5 mg** (tablets, spray) 1-3 times every 3-5 min. (check BP and pulse rate!)
- **Aspirin** (160-325 mg a day) to chew
- **Clopidogrel** (Plavix, Reodar) – 300 mg → 75 mg a day

Management of Patients with non-ST-elevation AMI in the prehospital setting

- **Inspection and physical examination**
- **Taking ECG** (whether there are or are not changes in ST, T, pathological Q wave, impaired rhythm and conductibility)
- **Decision as to admission to hospital**
- to the Intensive Care Unit (ICU)
- to the emergency (infarction) department

TREATMENT OF NON-ST-ELEVATION AMI

A list and range of obligatory medical services

- **1. Antithrombotic drugs:** acetylsalicylic acid of 160 – 325 mg as a first dose, then 75 – 100 mg a day; + tienopyridin derivatives: Clopidogrel of 300 – 600 mg as a first dose, then 75 mg a day – DAPT.

TREATMENT OF NON-ST-ELEVATION AMI

A list and range of obligatory medical services

- **2. Anticoagulants**:
- *Unfractionated heparin* 60 IU/kg as intravenous (IV) bolus (up to 4000 IU), then 12 IU/kg/hour as IV infusion (up to 1000 IU per hour) under the control over aPPT (1,5 – 2 times higher than normal)

TREATMENT OF NON-ST-ELEVATION AMI

A list and range of obligatory medical services

- **ANTICOAGULANTS:**
- Low molecular weight heparins or Fondaparinux subcutaneously (SC):
- Fondaparinux 2.5 mg SC once a day;
- Enoxaparin 1 mg/kg SC every 12 hours;
- Dalteparin 120 IU/kg every 12 hours;
- Nadroparin 86 IU/kg every 12 hours;
- Duration of therapy: 2-5 days

TREATMENT OF NON-ST-ELEVATION AMI

A list and range of obligatory medical services

- **3. Antiischemic therapy:**
- β-**adrenoreceptor blockers** without intrinsic sympathomimetic activity (Atenolol, Metoprolol, Bisoprolol, Nebivolol) ;
- **Nitrates** (isosorbide dinitrate, isosorbide mononitrate) or, in their intolerance, sydnonimins (Sydnopharm);
- **Calcium channel blockers** (Verapamil, Diltiazem) if β-blockers are contraindicated or in patients with Prinzmetal's angina.

TREATMENT OF NON-ST-ELEVATION AMI

A list and range of obligatory medical services

- **4. Statins:** Lovastatin, Simvastatin, Atorvastatin, Rosuvastatin.
- **5. ACE inhibitors** or, in case of their intolerance, **angiotensin receptor blockers (ARBs)**

TREATMENT OF NON-ST-ELEVATION AMI

A list and range of obligatory medical services

- **6. Non-narcotic and narcotic analgesics**
- if effect of short-acting nitrates is not sufficient;
- **7. Symptomatic therapy**
- (anti-hypertensive, antiarrhythmic);
- **8. Surgical myocardial revascularization** (indications and choice of a method of revascularization are determined by the character of coronary artery impairment based on coronary ventriculography).

Heart Attack Treatment

- **PAIN CONTROL MEDICATIONS**
  Sublingual (under the tongue) or intravenous (IV) nitrates such as nitroglycerin are given for pain and to reduce the oxygen requirements of the heart. Morphine or morphine derivatives are potent pain killers that may also be given for a heart attack.
- **BLOOD THINNING MEDICATIONS**
  If the ECG recorded during chest pain shows a change called "ST-segment elevation," clot-dissolving (thrombolytic) therapy may be initiated within 6 hours of the chest pain onset. This initial therapy will be administered as an IV infusion of streptokinase or tissue plasminogen activator, and will be followed by an IV infusion of heparin. Heparin therapy will last for 48 to 72 hours. Additionally, warfarin,taken orally, may be prescribed to prevent further development of clots

Basic therapy in ST-elevation AMI

- **1 – Pain relief** (morphine 2-4 mg IV, every 10-15 minutes);
- **2 – Oxygen therapy** (through the mask or nasal catheter, 2- 4 L/min)
- **3 – Reperfusion therapy** (thrombolysis, PCI and/or stenting, ACB);
- **4 – Anticoagulant therapy** (unfractionated heparin, low molecular weight heparins, fondaparinux);
- **5 – Antithrombotic drugs:** (aspirin, clopidogrel);

Basic therapy in ST-elevation AMI

- **6 – β-blockers** to all patients who have no contraindications;
- **7 – Nitrates** – at first nitroglycerin 0.5 mg under the tongue, every 3-5 minutes, then isoket 0.1% -10 mL IV infusion;
- **8 – ACE inhibitors** to all patients who have no contraindications;
- **9 – Statins**

Thrombolysis

- **Streptokinase** – 1500 000 U in 100 ml of saline given as an IV infusion over 1 hour is a widely used regimen .
- Streptokinase is antigenic and occasionally causes serious allergic manifestations. Circula-ting neutralising antibodies may persist for 5 years or more.
- Streptokinase may also cause hypotension

Thrombolysis

- **Alteplase** (human tissue plasminogen activator or t-PA)
- The standart regimen is given over 90 min
- Bolus dose of 15 mg IV
- Followed by 0,75 mg/kg body weight (but not exceeding 50 mg) over 30 min IV
- Followed by 0,5 mg/kg body weight (but not exceeding 35 mg) over 60 min IV

Thrombolysis

- **TENECTEPLASE (TNK)** – is an effective as alteplase at redusing death and MI whilst conferring similar intracerebral bleeding risks.
- Bolus dose of 0,5 mg/kg body weight IV during 10 sec.BUT not exceeding 50 mg.

Heart Attack Treatment

- *Thrombolytic therapy is not appropriate for people who have had:*
- A major surgery, organ biopsy, or major trauma within the past 6 weeks
- Recent neurosurgery
- Head trauma within the past month
- History of GI (gastrointestinal) bleed
- Brain tumor
- Stroke within the past 6 months
- Aortic dissection
- Current severely elevated high blood pressure
- Use of thrombolytic therapy can be complicated by significant bleeding.

Heart Attack Treatment

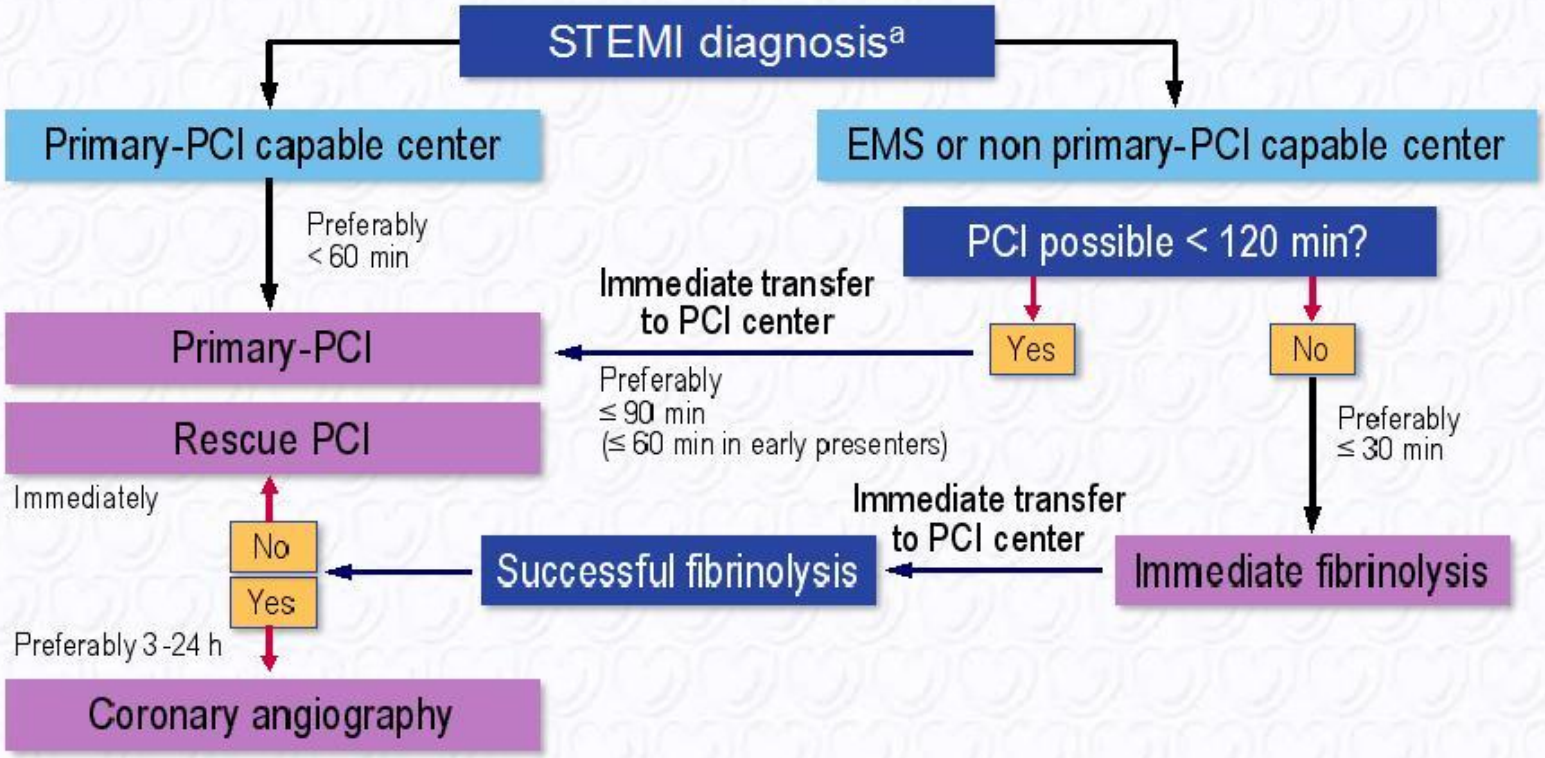
- A cornerstone of therapy for a heart attack is antiplatelet medication. Such medication can prevent the collection of platelets at a site of injury in a blood vessel wall -- like a crack in an atherosclerotic plaque. Platelets collecting and accumulating is the initial event that leads to clot formation. One antiplatelet agent widely used is aspirin. Two other important antiplatelet medications are ticlopidine (Ticlid) and clopidogrel (Plavix).

Heart Attack Treatment

- OTHER MEDICATIONS
- Beta-blockers (like metoprolol, atenolol, and propranolol) are used to reduce the workload of the heart.
- ACE Inhibitors (like ramipril, lisinopril, enalapril, or captopril) to prevent heart failure.

- SURGERY AND OTHER PROCEDURES
  Emergency coronary angioplasty may be required to open blocked coronary arteries. This procedure may be used instead of thrombolytic therapy, or in cases where thrombolytics should not be used. Often the re-opening of the coronary artery after angioplasty is ensured by implantation of a small device called a stent. Emergency coronary artery bypass surgery (CABG) may be required in some cases.

Prehospital and in-hospital management, and reperfusion strategies within 24 h of FMC



[a] The time point the diagnosis is confirmed with patient history and ECG ideally within 10 min from the first medical contact (FMC). All delays are related to FMC (first medical contact).

Cath = catheterization laboratory; EMS = emergency medical system; FMC = first medical contact; PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction.

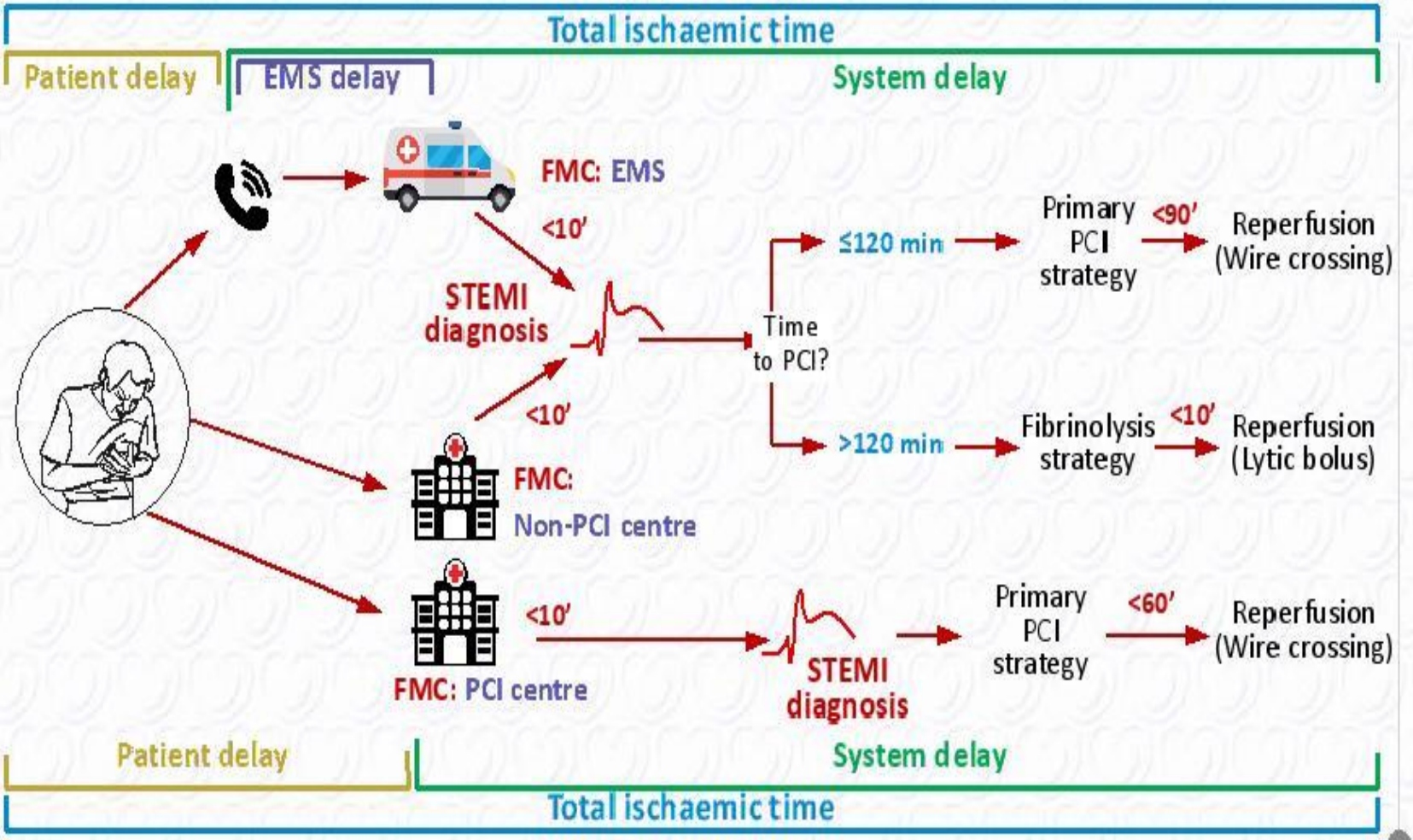
European Heart Journal (2012) 33, 2569–2619
doi:10.1093/eurheartj/ehs215

www.escardio.org/guidelines

EUROPEAN SOCIETY OF CARDIOLOGY®

Modes of patient presentation, components of ischaemic time and flowchart for reperfusion strategy selection

ESC European Society of Cardiology



www.escardio.org/guidelines 2017 ESC Guidelines for the Management of AMI-STEMI (European Heart Journal 2017 - doi:10.1093/eurheartj/ehx095)

Heart Attack Complications

- Arrhythmias such as ventricular tachycardia, ventricular fibrillation, heart blocks
- Congestive heart failure
- Cardiogenic shock
- Infarct extension: extension of the amount of affected heart tissue
- Pericarditis(infection around the lining of the heart)
- Pulmonary embolism (blood clot in the lungs)
- Complications of treatment (For example, thrombolytic agents increases the risk of bleeding.)

Heart Attack Prognosis (Expectations)

- The expected outcome varies with the amount and location of damaged tissue. The outcome is worse if there is damage to the electrical conduction system (the impulses that guide heart contraction).

  Approximately one-third of cases are fatal. If the person is alive 2 hours after an attack, the probable outcome for survival is good, but may include complications.

  Uncomplicated cases may recover fully; heart attacks are not necessarily disabling. Usually the person can gradually resume normal activity and lifestyle, including sexual activity.

Heart Attack Prevention

To prevent a heart attack- control risk factors

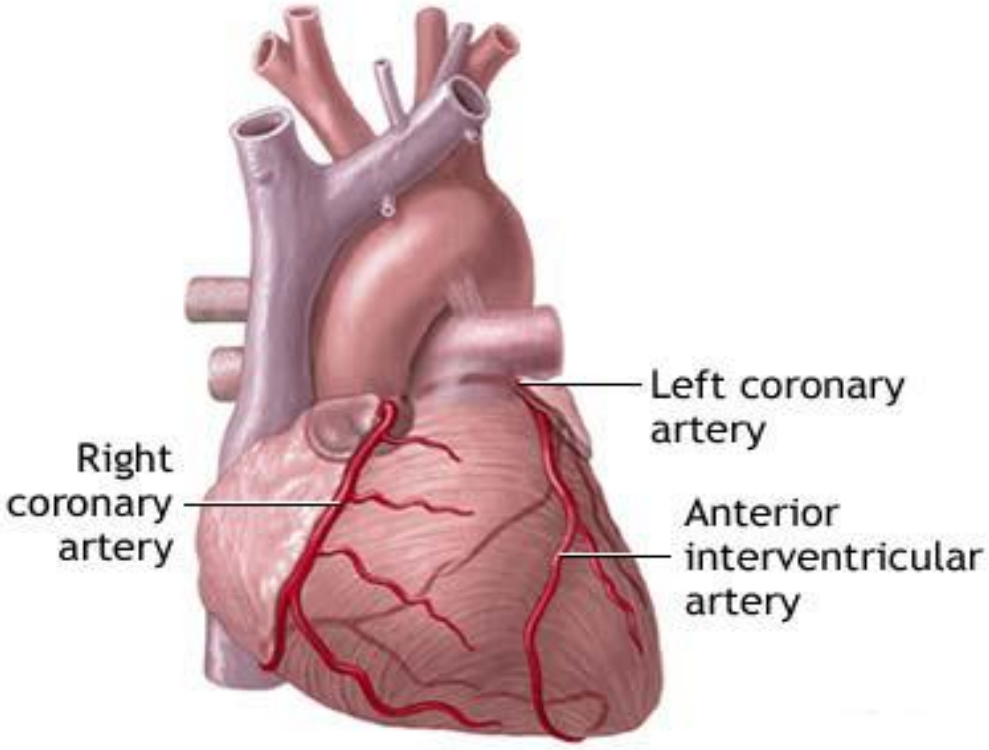
- Control blood pressure.
- Control total cholesterol levels. To help with cholesterol control, doctor may prescribe a medication of the statins group (atorvastatin, simvastatin).
- Stop smoking if patient smoke.
- Eat a low fat diet rich in fruits and vegetables and low in animal fat.
- Control diabetes.

Heart Attack Prevention

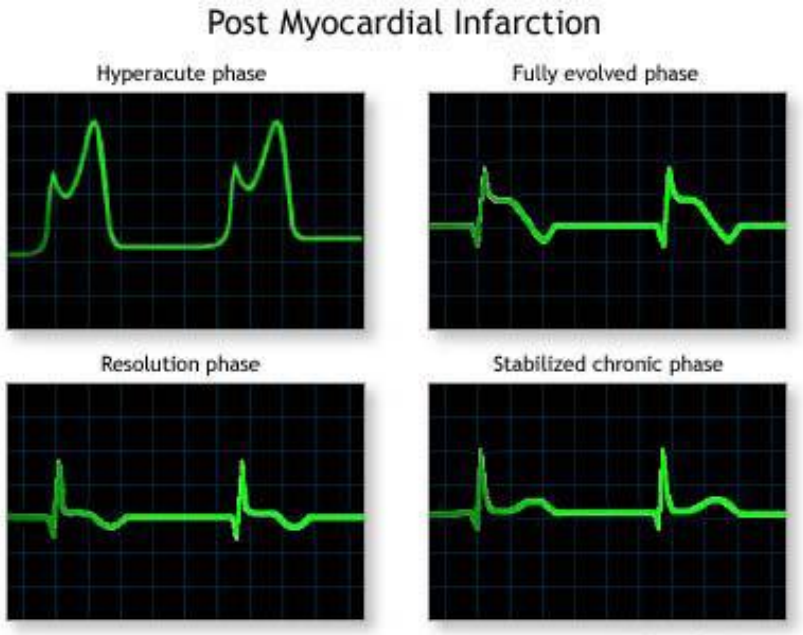
- Lose weight if patient are overweight.
- Exercise daily or several times a week by walking and other exercises to improve heart fitness. (Consult your health care provider first.)
- If patient have one or more risk factors for heart disease, possible taking aspirin to help prevent a heart attack.

After a heart attack, follow-up care is important to reduce the risk of having a second heart attack. Often, a cardiac rehabilitation program is recommended to help you gradually return to a "normal" lifestyle. Follow the exercise, diet, and medication regimen prescribed by your doctor.

- THANK YOU FOR ATTENTION !



- **(Acute MI)**
  A heart attack or acute myocardial infarction (MI) occurs when one of the arteries that supplies the heart muscle becomes blocked. Blockage may be caused by spasm of the artery or by atherosclerosis with acute clot formation. The blockage results in damaged tissue and a permanent loss of contraction of this portion of the heart muscle.



(Post Myocardial Infarction ECG Wave Tracings)

Various phases can be seen through ECG wave tracings following a heart attack:

Hyperacute phase begins immediately after a heart attack

Fully evolved phase starts a few hours to days after a heart attack

Resolution phase appears a few weeks after a heart attack

Stabilized chronic phase is the last phase and typically has permanent pathological changes compared to a normal ECG tracing.

Heart Attack Treatment :

A heart attack is a medical emergency! Hospitalization is required and, possibly, intensive care. Continuous ECG monitoring is started immediately, because life-threatening arrhythmias are the leading cause of death in the first few hours of a heart attack.

The goals of treatment are to stop the progression of the heart attack, to reduce the demands on the heart so that it can heal, and to prevent complications.

An intravenous line will be inserted to administer medications and fluids. Various monitoring devices may be necessary. A urinary catheter may be inserted to closely monitor fluid status.

Oxygen is usually given, even if blood oxygen levels are normal. This makes oxygen readily available to the tissues of the body and reduces the workload of the heart.

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INTRODUCTION

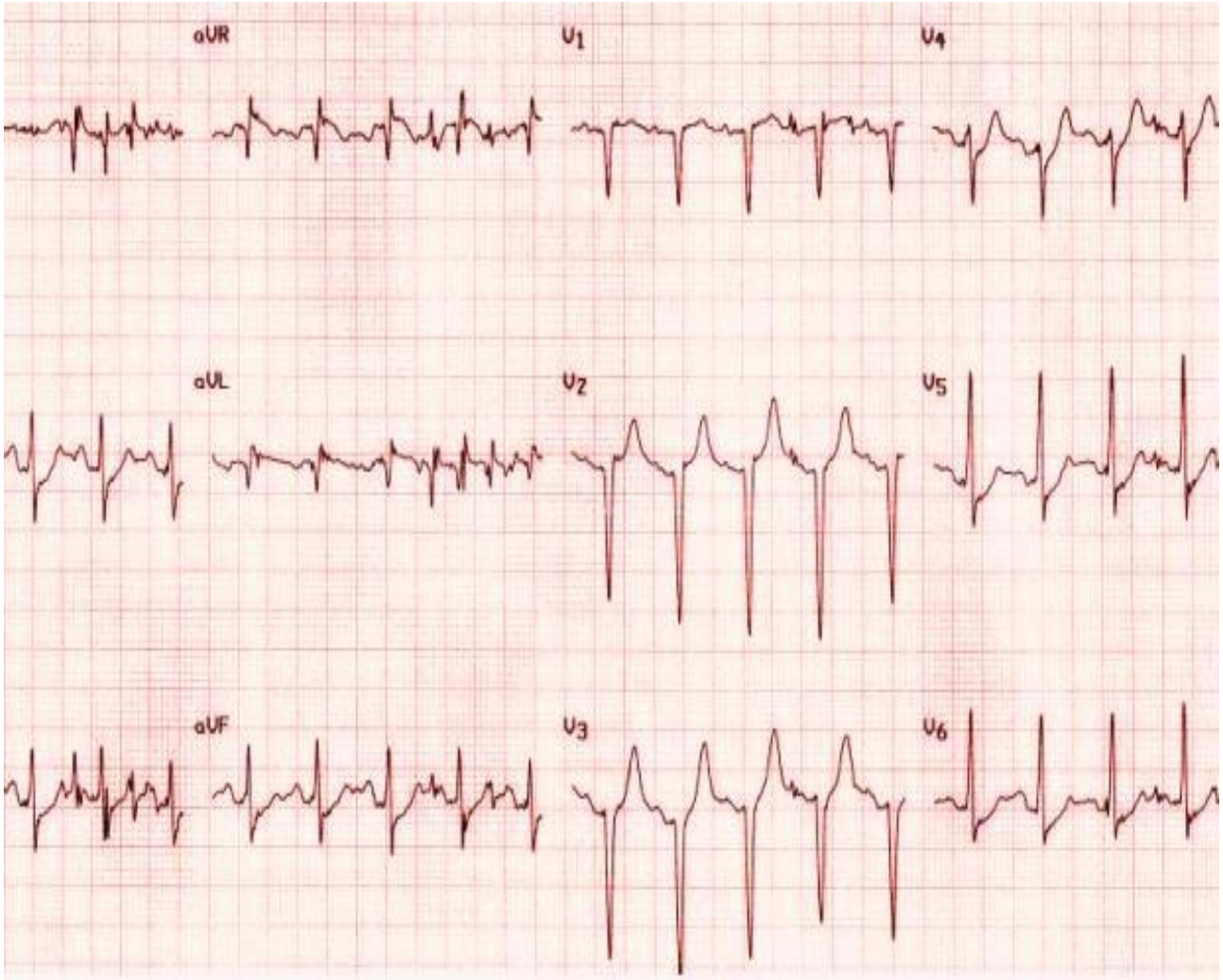
Despite its low sensitivity and specificity (67% and 72%, respectively), **exercise testing** has remained one of the most widely used noninvasive tests to determine the prognosis in patients with suspected or established coronary disease.

As a screening test for coronary artery disease, the exercise stress test is useful in that it is relatively simple and inexpensive. It has been considered particularly helpful in patients with chest pain syndromes who have moderate probability for coronary artery disease, and in whom the resting electrocardiogram (ECG) is normal. The following case presentation and discussion will question the predictive value of a negative stress testing in patients with moderate probability for coronary artery disease.

CASE PRESENTATION

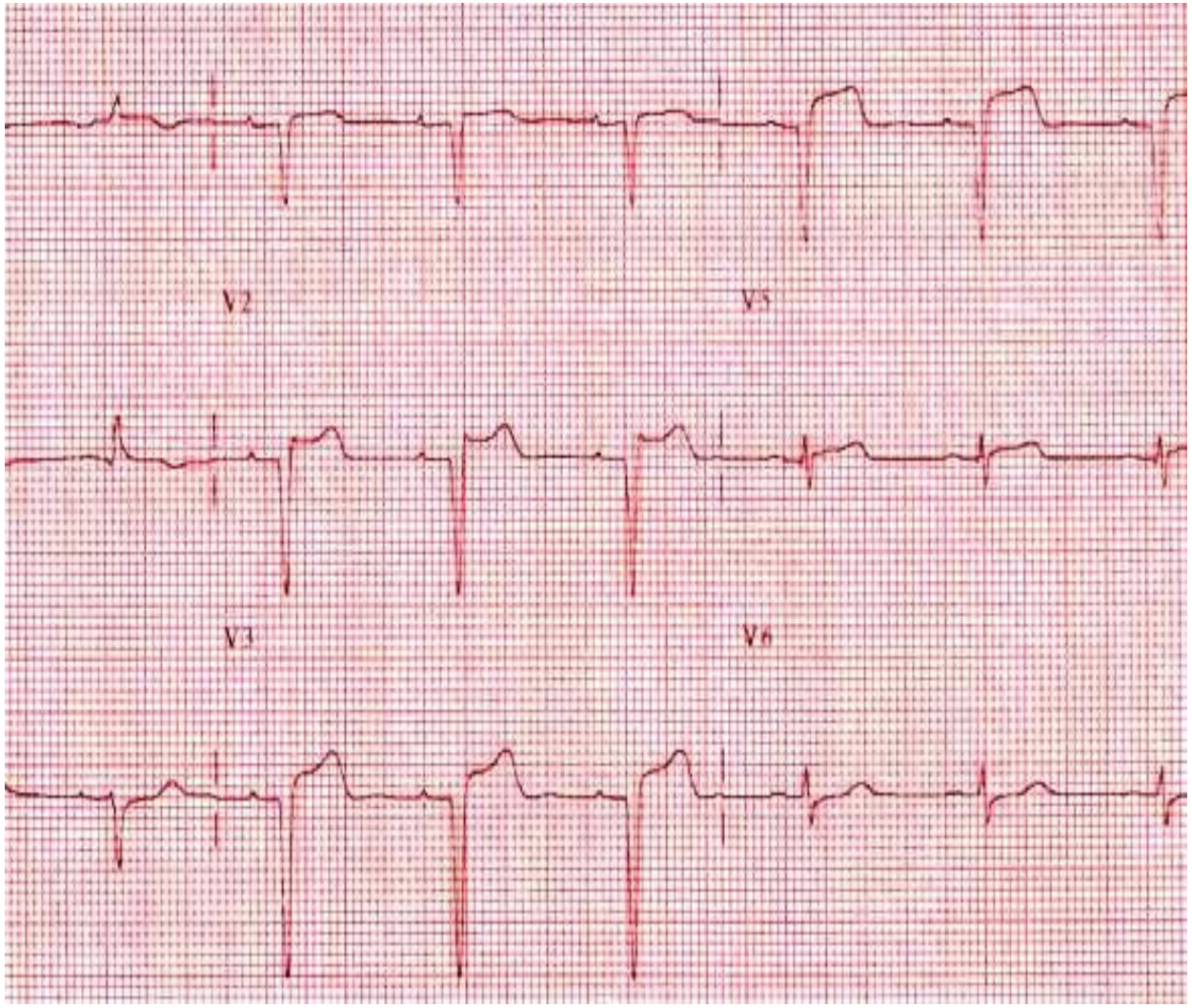
On October 02, 2006, a 56 year-old smoker male presented to our emergency room (ER) with a prolonged episode of epigastric and lower sternal discomfort. His discomfort was relieved with multiple doses of sublingual nitroglycerine and 2 doses of oral antacids. His physical examination, electrocardiogram (ECG),and cardiac markers (including creatine phosphokinase and Troponin I) were unremarkable. His past medical history is significant for mild hyperlipidemia and hypertension. He had a strong family history of premature coronary artery disease; his brother died of myocardial infarction at age 52 years.

Although his chest discomfort was atypical, he was considered as an intermediate-risk patient, based on his multiple cardiac risks. A symptom-limited exercise stress test was carried out. He exercised for 12 minutes on the standard Bruce protocol, achieving a peak heart rate of 144 per minute and a total workload equivalent to 12.1 METS. He reported no chest pain during this test. The exercise ECG revealed no significant ST-segment depression (Figure 1). Therefore, this test was considered as a low-risk negative test, predicting an annual mortality rate of less than 1%.



On November 21, 2006, he presented to our ER again with several hours of mid-sternal chest pain radiating to the left arm. His ECG revealed extensive ST-elevation anterior myocardial infarction (Figure 2).

Figure 1: A twelve-lead exercise stress electrocardiogram (ECG) recorded within the first minute of recovery, showing no significant ST-segment depression in response to exercise.



- *Figure 2: A twelve-lead electrocardiogram (ECG), recorded on admission to Cardiac Care Unit, showing recent extensive anterior wall myocardial infarction.*

Coronary angiography revealed total occlusion of the proximal segment of left anterior descending artery, and severe disease involving the proximal segment of the obtuse marginal branch (Figure 3). He was treated with direct Percutaneous Coronary Intervention (PCI).

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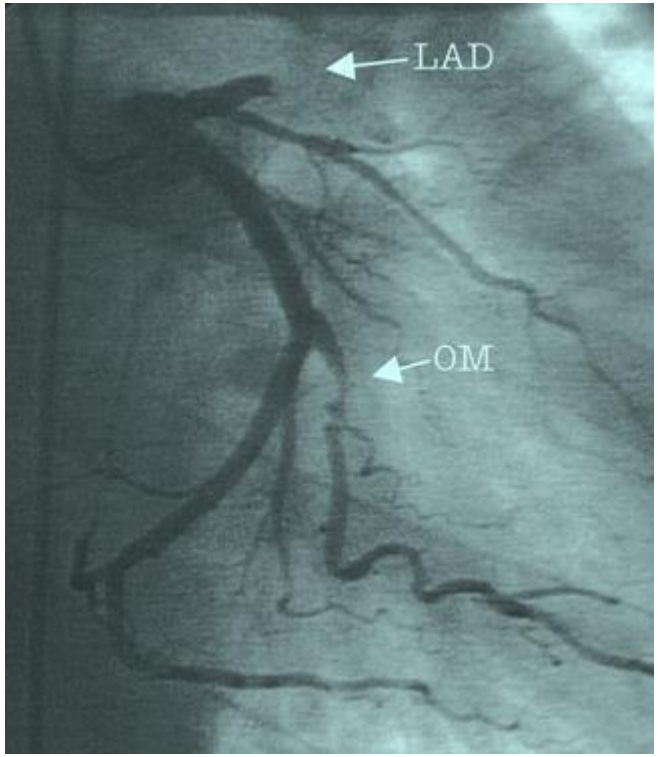


- *Figure 3: Left coronary artery angiograms showing total occlusion of the left anterior descending artery (LAD) and subtotal occlusion of the obtuse marginal (OM) branch of the left circumflex coronary artery.*

DISCUSSION

Exercise stress testing has traditionally served as a noninvasive tool in the diagnosis of coronary artery disease. It complements the medical history and physical examination, and it remains the second most commonly performed cardiologic procedure next to the routine ECG.

Our patient (described above) is also considered an intermediate-risk patient. Atypical chest pain in a 56-year-old man is associated with a 50% probability of CAD. Diagnostic stress testing is most valuable in this intermediate pretest probability category, because the test result has the largest potential effect on diagnostic outcome.

The type of patient being tested and the results of the exercise stress test must be considered together when determining the likelihood of subsequent cardiac event [1].

The estimation of pretest probability of obstructive CAD is based on the patient's history (including age, gender, and chest pain characteristics), physical examination, and initial testing.

Typical or definite angina **(table 1)** makes the pretest probability of obstructive CAD so high that the test result does not dramatically change the probability.

Table 1: Characteristics of anginal chest pain.
A- Typical or definite angina can be defined as a substernal chest pain or discomfort that is provoked by exertion or emotional stress and relieved by rest and/or nitroglycerine.
B- Atypical or probable angina can be defined as chest pain or discomfort that lacks one of the three characteristics of typical angina.

Table 2: High-risk exercise electro-cardiographic variables
1- ST-segment depression ≥ 2.0 mm
2- ST-segment depression in stage I ≥ 1 mm
3- ST-segment depression in multiple leads
4- ST-segment depression persists for greater than 5 minutes during the recovery period
5- Achievement of a workload of less than 4 METS or a low exercise maximal heart rate
6- Abnormal blood pressure response

Exercise-induced typical anginal chest pain can be a valuable indicator of the presences of coronary artery disease. The presence of diagnostic ST-segment depression in association with exercise-induced chest pain is highly predictive of significant coronary artery disease [2] **(table 2).**

Major non-electrocardiographic observations that carry prognostic importance include the maximum work capacity, the peak systolic blood pressure achieved, the presence or absence of angina, and ventricular tachycardia [3]. Exercise capacity has also been considered of prognostic value in patients with coronary artery disease. An exercise capacity of more than 12 METS (Bruce protocol stage 4) is indicative of a good prognosis in patients with coronary artery disease regardless of other responses or whether medical or surgical therapy is selected for management [1,4].

Our patient, described above, was able to exercise for 12 minutes; a workload equivalent to 12.1 METS, without any chest pain or ischemic ST-segment depression. Therefore, his stress test was considered a low-risk test, predictive of an annual mortality rate of less than 1%. Nevertheless, he presented in less than 2 months with an extensive anterior wall myocardial infarction.

The rupture of plaques is now considered to be the common pathophysiological substrate of the acute coronary syndromes. During the natural evolution of the atherosclerotic plaques, an abrupt and catastrophic transition may occur, characterized by plaque rupture and exposure of substances that promote platelets activation and thrombin generation [5].

These changes may lead to the conversion of previously stable and non-obstructive plaques to unstable and occlusive ones. This transition, from an asymptomatic or a minimally symptomatic chronic stable state to acute unstable coronary heart disease, may take place in few hours.

This means that, coronary artery disease that has not resulted in sufficient luminal occlusion to cause ischemia during stress testing can still lead to ischemic events through spasm, plaque rupture, and thrombosis. These non-obstructive lesions explain some of the events that may occur after a negative exercise stress test. This dynamic process of plaque rupture may evolve to a completely occlusive thrombus, typically producing ST elevation on the ECG.

Therefore, we should not be surprised if an asymptomatic patient with underlying insignificant coronary disease, who had a negative stress test just few weeks ago, develops an acute coronary syndrome as result of this dynamic process of plaque rupture.

A negative exercise or even pharmacological radionuclide stress may not mean very much if we consider the dynamic nature of this disease. Therefore, a negative result should not exclude the diagnosis of significant coronary artery disease.

The above-described clinical case provides an example to this view.

More recently, other noninvasive modalities, including coronary CT-angiography and whole-heart coronary magnetic resonance angiography, showed moderate sensitivity and high specificity in detecting coronary artery disease [6-8]. These noninvasive imaging modalities are able to detect the location of the coronary atherosclerotic plaque and to estimate the degree of lumen reduction. It is likely that these relatively new imaging modalities will replace stress testing, as a screening test for coronary artery disease, in future.

ECG Basics

The electrocardiogram (ECG) is a diagnostic tool that measures and records the electrical activity of the heart in detail. Being able to interpretate these details allows diagnosis of a wide range of heart problems.

ECG Electrodes

Skin Preparation:

Clean with an alcohol wipe if necessary. If the patients are very hairy – shave the electrode areas.

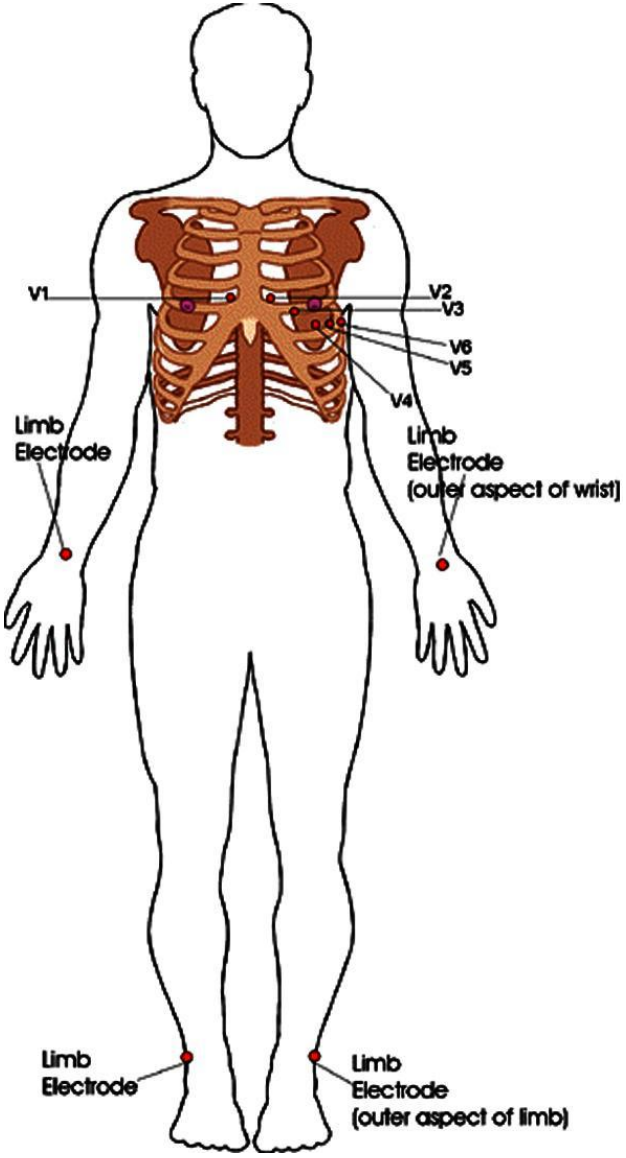
ECG standard leads

There are three of these leads, I, II and III.

Lead I: is between the right arm and left arm electrodes, the left arm being positive.
Lead II: is between the right arm and left leg electrodes, the left leg being positive.
Lead III: is between the left arm and left leg electrodes, the left leg again being positive.

Chest Electrode Placement

V1: Fourth intercostal space to the right of the sternum.
V2: Fourth intercostal space to the Left of the sternum.
V3: Directly between leads V2 and V4.
V4: Fifth intercostal space at midclavicular line.
V5: Level with V4 at left anterior axillary line.
V6: Level with V5 at left midaxillary line. (Directly under the midpoint of the armpit)

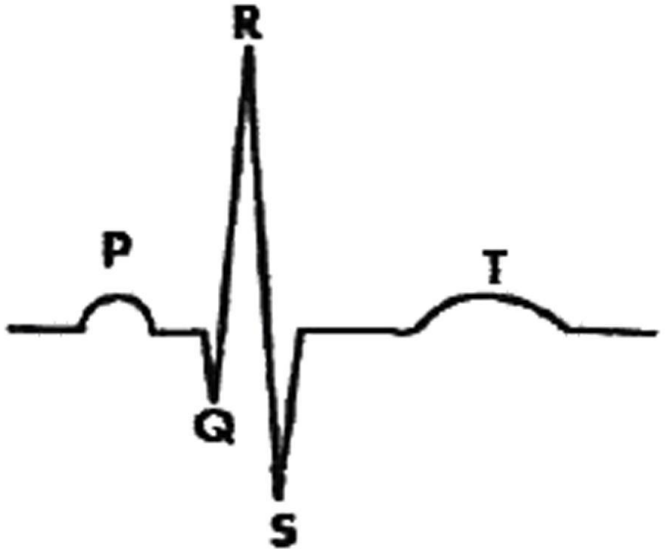


ECG Leads - Views of the Heart

Chest Leads	View
V1 & V2	Right Ventricle
V3 & V4	Septum/Lateral Left Ventricle
V5 & V6	Anterior/Lateral Left Ventricle

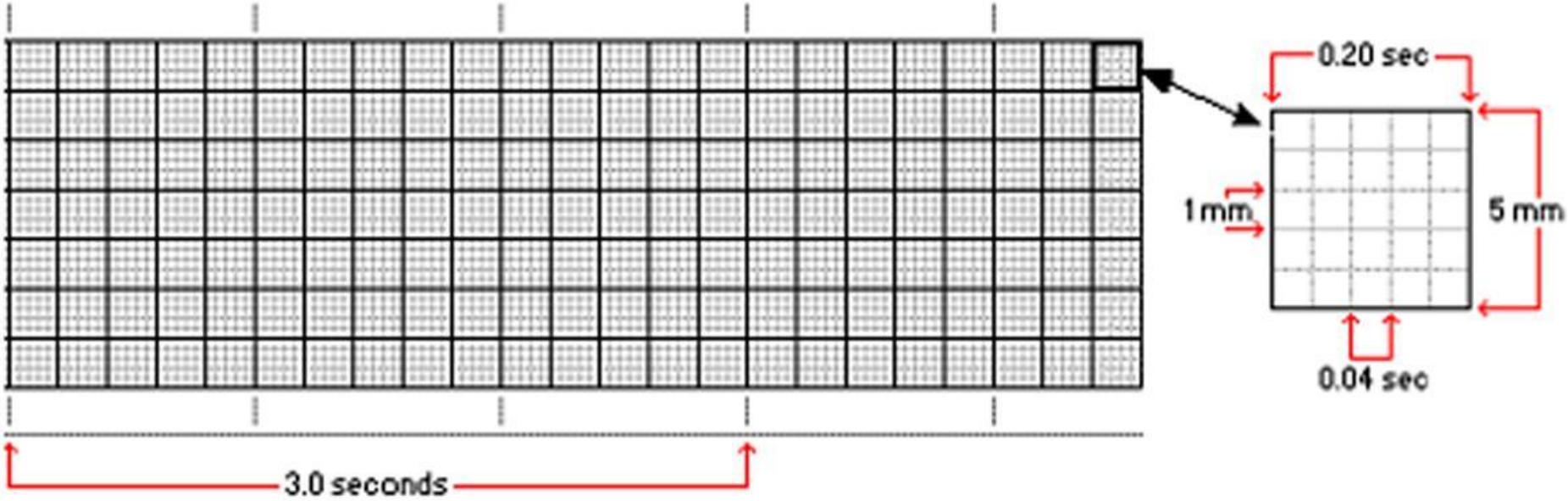
The ECG records the electrical activity that results when the heart muscle cells in the atria and ventricles contract.

- Atrial contractions show up as the P wave.
- Ventricular contractions show as a series known as the QRS complex.
- The third and last common wave in an ECG is the T wave. This is the electrical activity produced when the ventricles are recharging for the next contraction (repolarizing).
- Interestingly, the letters P, Q, R, S, and T are not abbreviations for any actual words but were chosen many years ago for their position in the middle of the alphabet.
- The electrical activity results in P, QRS, and T waves that are of different sizes and shapes. When viewed from different leads, these waves can show a wide range of abnormalities of both the electrical conduction system and the muscle tissue of the hearts 4 pumping chambers.



ECG Interpretation

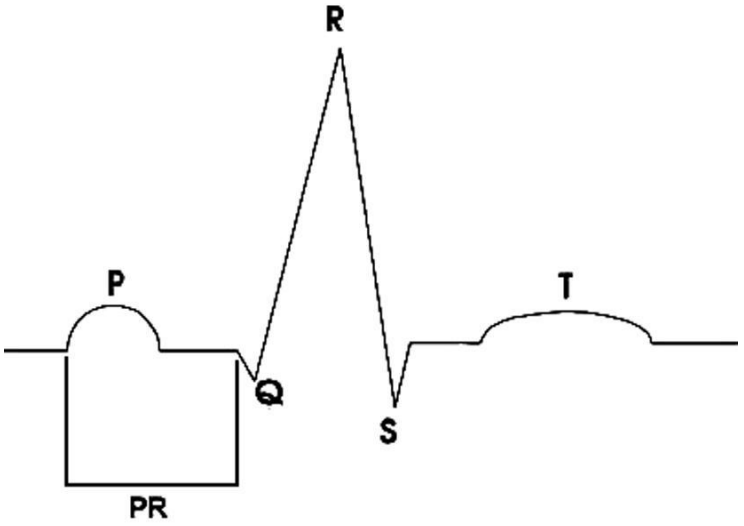
The graph paper that the ECG records on is standardised to run at 25mm/second, and is marked at 1 second intervals on the top and bottom. The horizontal axis correlates the length of each electrical event with its duration in time. Each small block (defined by lighter lines) on the horizontal axis represents 0.04 seconds Five small blocks (shown by heavy lines) is a large block, and represents 0.20 seconds.



Duration of a waveform, segment, or interval is determined by counting the blocks from the beginning to the end of the wave, segment, or interval.

P-Wave: represents atrial depolarization - the time necessary for an electrical impulse from the sinoatrial (SA) node to spread throughout the atrial musculature.

- Location: Precedes QRS complex Amplitude: Should not exceed 2 to 2.5 mm in height Duration: 0.06 to 0.11 seconds
- P-R Interval: represents the time it takes an impulse to travel from the atria through the AV node, bundle of His, and bundle branches to the Purkinje fibres.
- Location: Extends from the beginning of the P wave to the beginning of the QRS complex
  Duration: 0.12 to 0.20 seconds.



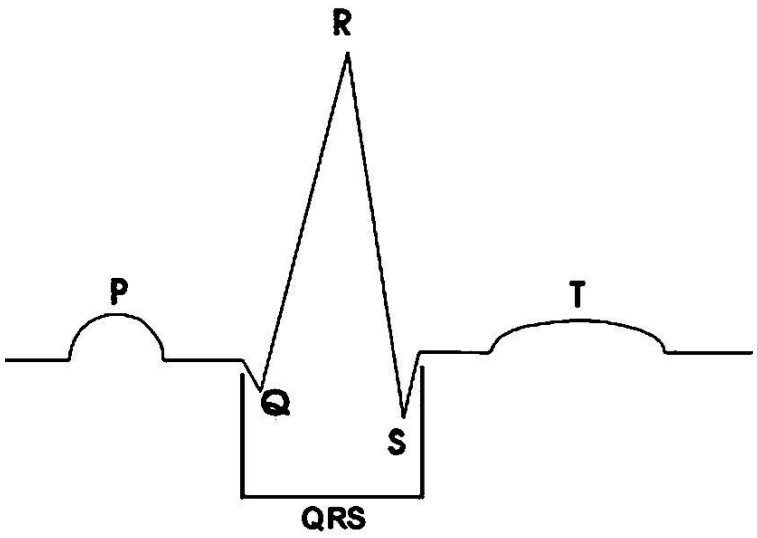
QRS Complex: represents ventricular depolarisation. The QRS complex consists of 3 waves: the Q wave, the R wave, and the S wave.

- The Q wave is always located at the beginning of the QRS complex.
  It may or may not always be present.

  The R wave is always the first positive deflection.

  The S wave, the negative deflection, follows the R wave
- Location: Follows the P-R interval
  Amplitude: Normal values vary with age and sex

  Duration: No longer than 0.10 seconds

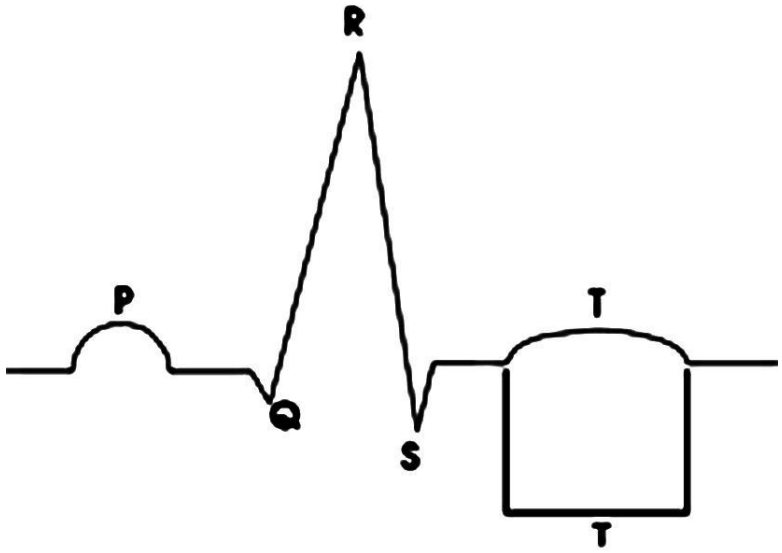


Q-T Interval: represents the time necessary for ventricular depolarization and repolarization.

- **Location: Extends from the beginning of the QRS complex to the end of the T wave (includes the QRS complex, S-T segment, and the T wave)**

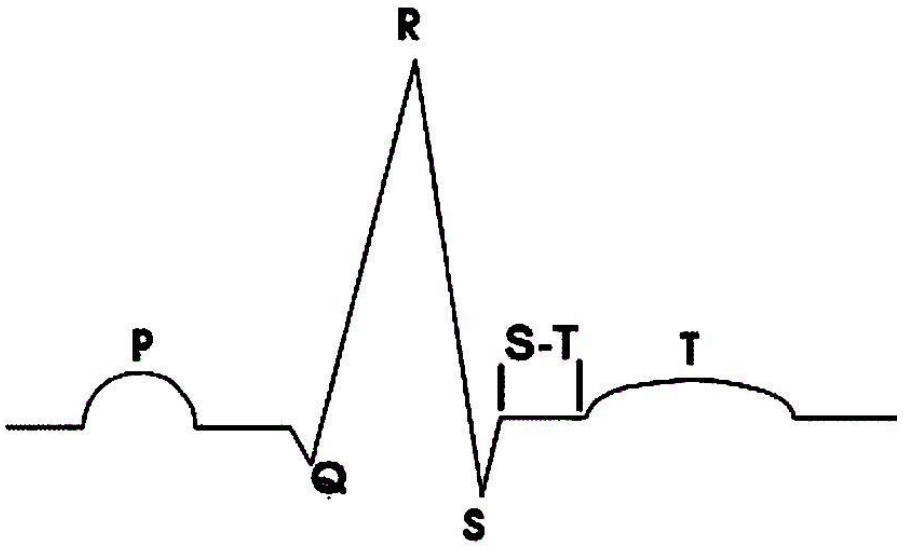
  Duration: Varies according to age, sex, and heart rate
- **T Wave: represents the repolarization of the ventricles. On rare occasions, a U wave can be seen following the T wave. The U wave reflects the repolarization of the His-Purkinje fibres.**
- **Location: Follows the S wave and the S-T segment**
  Amplitude: 5mm or less in standard leads I, II, and III; 10mm or less in precordial leads V1-V6.

  Duration: Not usually measured



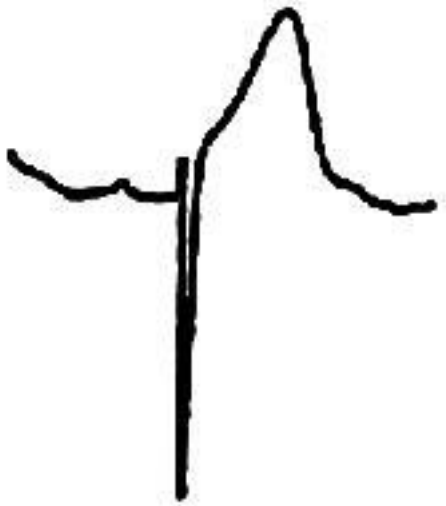
S-T Segment: represents the end of the ventricular depolarization and the beginning of ventricular repolarization.

- Location: Extends from the end of the S wave to the beginning of the T wave
  Duration: Not usually measured



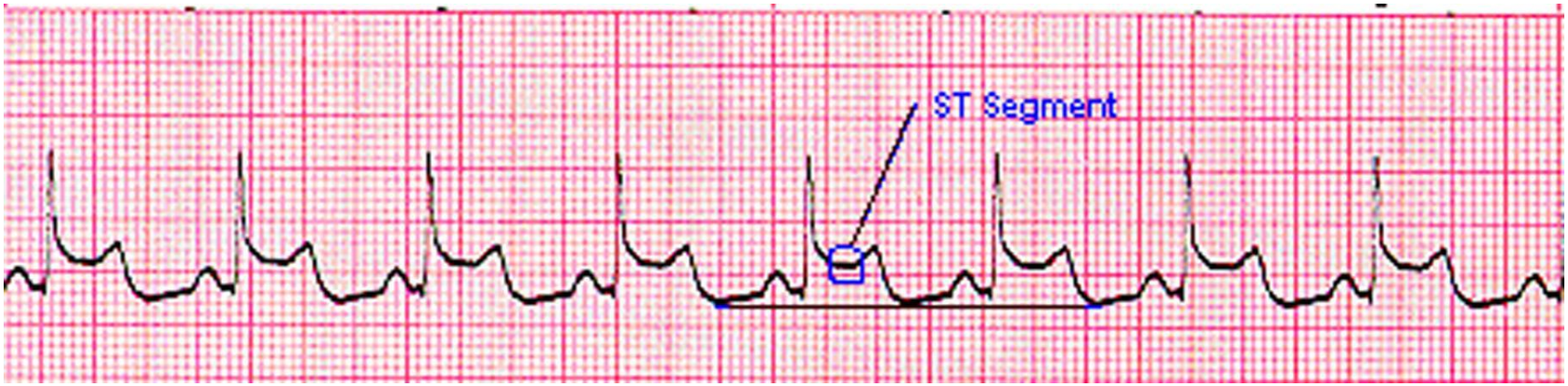
The ECG and Myocardial Infarction

- During an MI, the ECG goes through a series of abnormalities. The initial abnormality is called a **hyperacute T wave**. This is a T wave that is taller and more pointed than the normal T wave.



Hyperacute T Wave

The abnormality lasts for a very short time, and then elevation of the ST segment occurs. This is the hallmark abnormality of an acute MI. It occurs when the heart muscle is being injured by a lack of blood flow and oxygen and is also called a *current of injury*.

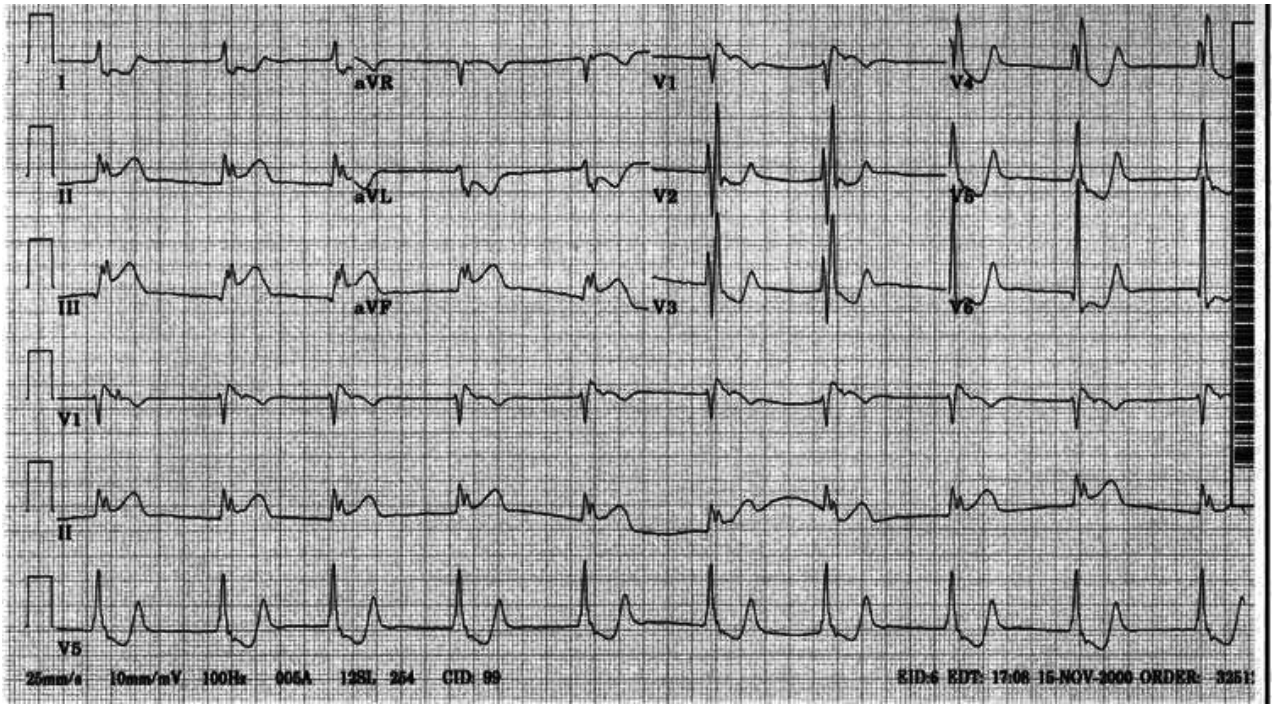


Left Anterior Descending Artery Right Coronary Artery

An ECG can not only tell you if an MI is present but can also show the approximate location of the heart attack, and often which artery is involved. When the ECG abnormalities mentioned above occur, then the MI can be localized to a certain region of the heart. For example, see the table below:

ECG leads	Location of MI	**Coronary Artery**
II, III, aVF	Inferior MI	Right Coronary Artery
V1-V4	Anterior or Anteroseptal MI	Left Anterior Descending Artery
V5-V6, I,aVL	Lateral MI	Left Circumflex Artery
ST depression in V1, V2	Posterior M	Left Circumflex Artery or Right Coronary Artery

Right Ventricular Myocardial Infarction EKG



Characteristics

This EKG shows an Acute Inferior Myocardial Infarction which is often associated with a **Right Ventricular Myocardial Infarction**. If there is ST elevation in V1 and V2, the RV infarction should be considered.

ECG Rounds

A 76-year-old retired physician came to the clinic for a medical check-up. He had never experienced any serious medical problem and had no history of heart disease. His physical examination was unremarkable. A chest x-ray was ordered, revealing prominence of the right heart border in the area of the ascending aorta, which was not seen on an old x-ray taken 7 years earlier. His electrocardiogram (ECG) revealed some irregularities (Figure 1).

Questions: **Does the ECG show any specific heart disease? How do you explain the loss of anterolateral R-wave forces? Is it a "Q-wave equivalent" and a marker of previous silent myocardial infarction (MI) in this patient?**

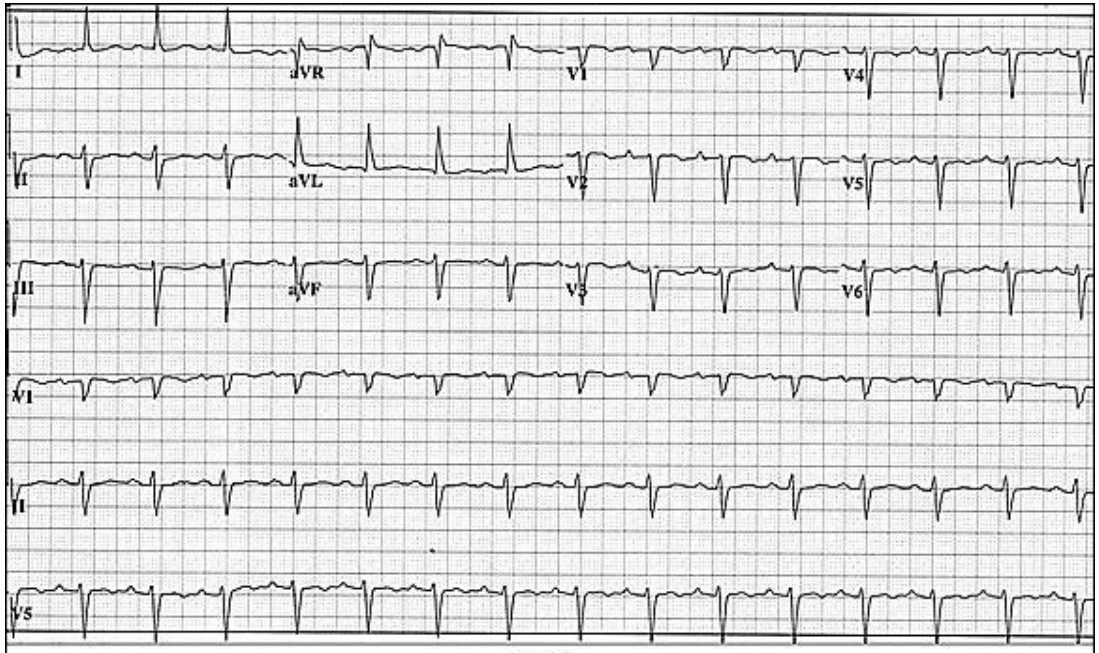
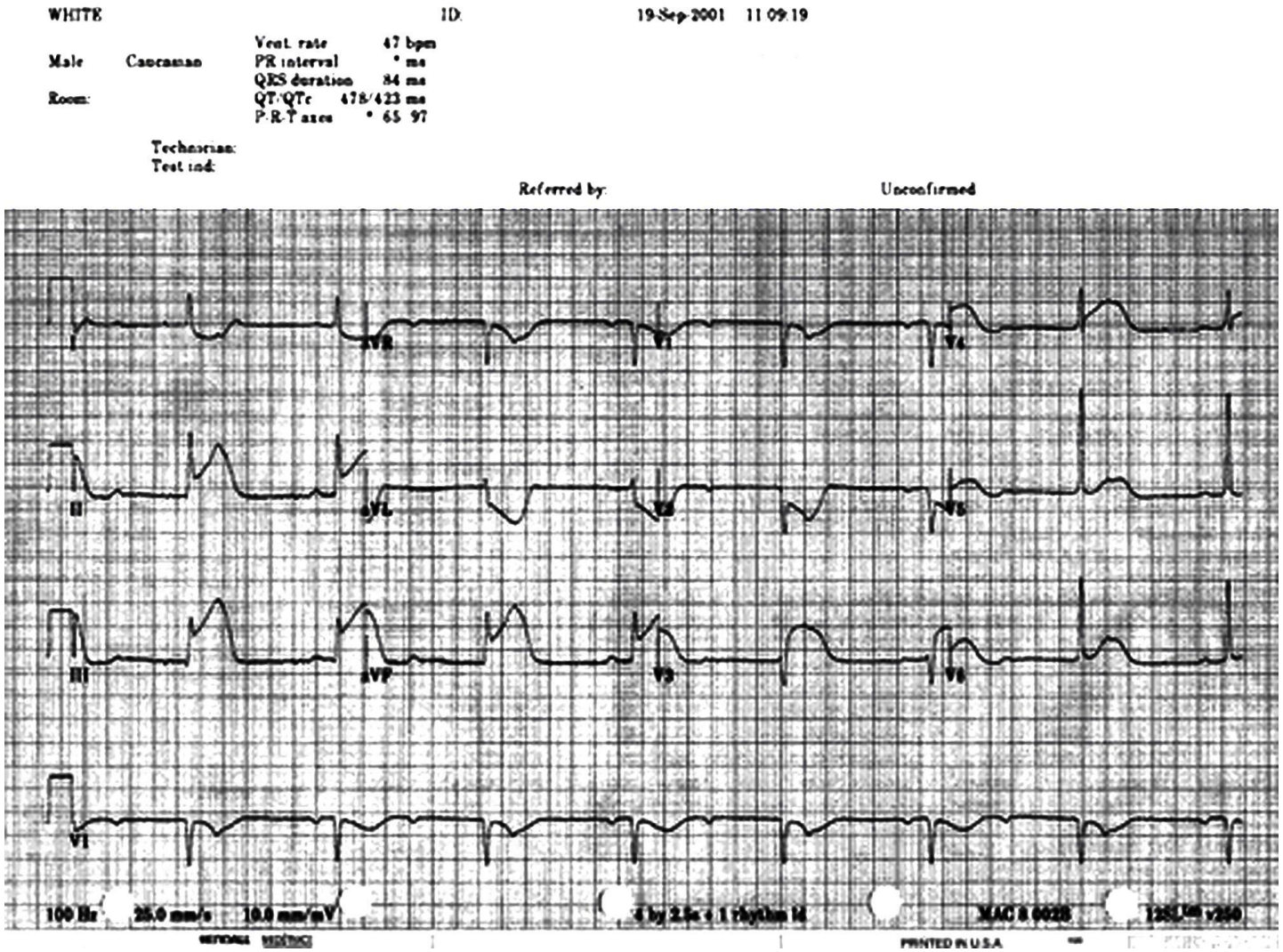


Figure 1

Inferior Myocardial Infarction with AV Block



Characteristics

Both bradyarrhythmias and conduction disturbances can be seen with myocardial infarctions and are generally related to ischemia or autonomic disturbance. The clinical features and management of bradyarrhythmias and conduction block depends on the location of the infarction. The right coronary artery supplies the SA node in 60 percent of people and the left circumflex the remaining. In over 90 percent of people, the RCA feeds the AV node and proximal His. The terminal portion of the His and main left bundle and right bundle branch are supplied by septal perforators of the LAD. Sinus bradycardia, prolonged PR conduction with Wenkebach and complete heart block are common in inferior myocardial infarctions (IMI). Complete AV block occurs in approximately 10 percent of patients with IMI. This rarely occurs suddenly, most often seen with prolonged PR conduction gradually progressing to complete AV block. AV block occurs within the node in over 90 percent of cases and typically results in a transient block. The escape complex is usually narrow and infrequently requires pacing. Bradyarrhythmias occurring in the setting of inferior infarctions are generally responsive to atropine.

The ECG in acute myocardial infarction (MI)

Acute MI may cause changes in the QRS complex, ST segment or the T wave. However, the only definitive diagnostic changes of myocardial infarction are changes in the QRS complex.

The QRS complex in infarction

Two types of QRS abnormalities may indicate infarction:
1) Inappropriately low R wave voltage in a local area and
2) Abnormal Q waves

The above two abnormalities are actually part of the same process - i.e. the development of a negative Q wave and the reduction in size of the positive wave.

The loss of positivity is the result of myocardial necrosis beneath the exploring electrode. The size of the positive wave in each precordial lead is related to the thickness of viable myocardium underneath that electrode.

Abnormal Q waves and QS complexes

In a **transmural infarction** (endocardium to epicardium), there will be **total loss of R waves** in leads overlying the infracted zone. This gives rise to entirely negative waves - i.e. **QS complexes**. These negative waves are the result of depolarisation of the posterior wall of the ventricle travelling from endocardium to epicardium (i.e. away from the anterior leads).

The reduction in R wave voltage can only be confirmed if either a previous ECG shows a significantly greater R wave height in the appropriate leads before the infarction occurred, or the leads involved are two or more of the leads V2 to V5.

Therefore, the four possible QRS changes indicative of infarction are:
1) Reduced R wave voltage (confirmed by previous ECGs)
2) Abnormal Q waves without any conclusive evidence of R wave reduction
3) Reduced R wave voltage in association with abnormal Q waves and
4) QS complexes.

These four changes represent increasing thickness of infarction as part of a common process. A combination of these findings is seen in an infarction of non-uniform thickness.

Abnormal Q waves

Q waves may be recognised to be abnormal because of:
1) Abnormal width (duration) - i.e. Q wave = 0.04 s or
2) Abnormal depth (relative to the following R wave) - i.e. depth of Q wave >25% of the height of the following R wave is abnormal.

ST segment changes in myocardial infarction

Dramatic ST segment changes occur in the early stages of myocardial infarction. Such changes indicate **myocardial injury rather than infarction**.

The injury state is unstable, and acute ST segment elevation **always** resolves to some extent and **usually** resolves completely. The resolution of the acute ST elevation is **usually** accompanied by development of the QRS changes of frank infarction, although **occasionally**, it may resolve without the development of diagnostic changes of infarction.

The ST segment shift is produced by myocardial injury, which causes a disturbance in the current flow across the cell membrane.

The essential change of myocardial injury is ST segment elevation above the isoelectric line.

The normal ST segment does not deviate by more than 1 mm above or below the isoelectric line.

Abnormal ST segment elevation occurs in leads facing the infarction, both in transmural and subepicardial infarction. Reciprocal ST segment depression may be seen at the same time as the above primary changes in leads recording from positions opposite to the infarct.

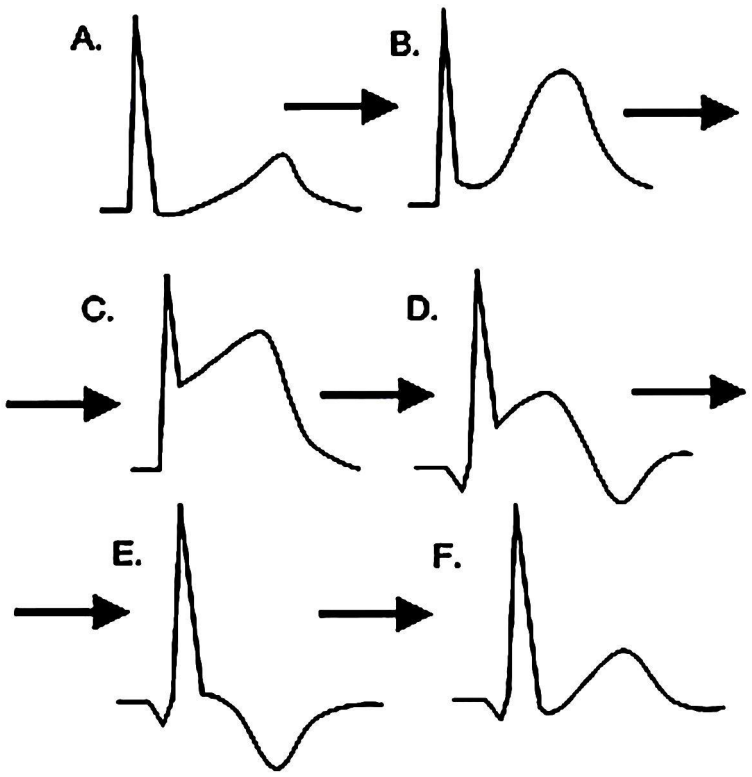
Primary ST segment depression is seen in leads facing the infarct when a ubendocardial infarction occurs.

T wave changes of infarction

The spectrum of changes in the T waves during infarction includes flattening of the T waves, bi-phasic T waves, inverted T waves and abnormally tall T waves.

The most typical T wave change in acute MI is deep, symmetrical T wave inversion.

Sequence of changes in acute MI



Evolution of Acute MI

A) Shows the normal QRS complex in a lead.

B & C) Within **hours** of the clinical onset of an MI, there is **ST segment elevation**. At this stage no QRS or T wave changes have occurred. This indicates myocardial damage only, not definitive evidence of infarction.

D) Within **days**, the R wave voltage falls and abnormal Q waves appear. This is sufficient evidence of an infarction. In addition, T wave inversion will also have appeared but the ST segment elevation may be less obvious than before.

E) Within **one or more weeks**, the ST segment changes revert completely to normal. The R wave voltage remains low and the abnormal Q waves persist. Deep, symmetrical T wave inversion may develop at this stage.

F) Months after the MI, the T waves may gradually return to normal. The abnormal Q waves and reduced R wave voltage persist.

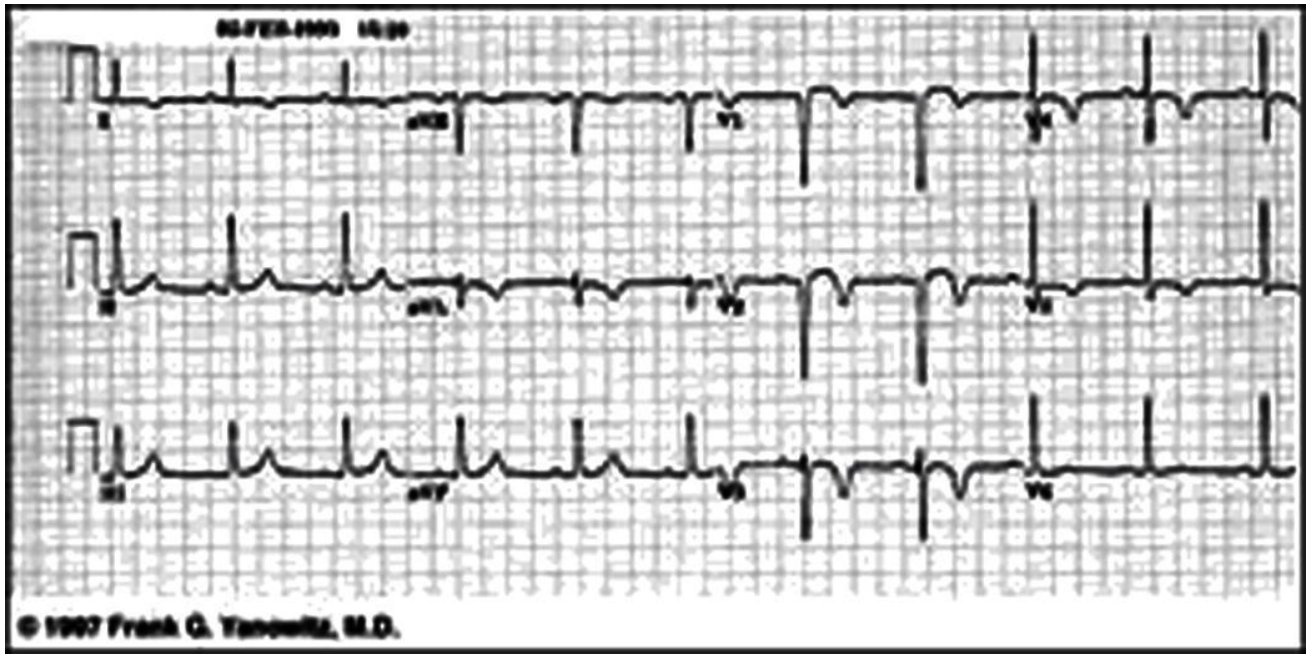
Occasionally, all evidence of infarction may be lost with the passing of time; this is due to shrinkage of scar tissue.

Location of changes in MI

Because primary ECG changes occur in leads overlying the infarct, the location of an infarct can be derived by looking at the primary changes occurring in such leads. This is depicted in the following table:

Location of infarction	Leads showing primary changes
	Typical changes
Anterior infarction	
Antero-septal	V1, V2, V3
Anterior	Some of V1-V3 plus some of V4-V6
Anterior extensive	V1, V2, V3, V4, V5, V6,I, aVL
Antero-lateral	V4, V5, V6, I, aVL, possibly II
High lateral	aVL and/or I
Inferior infarction	
Inferior	II, III, aVF
Infero-lateral (= apical)	II, III, aVF, V5, V6 & sometimes also I, aVL
Infero-septal	II, III, aVF, V1, V2, V3
	Other changes
Posterior infarction	V1, V2 (inverse of usual changes elsewhere)
Subendocardial infarction	Any lead (usually multiple leads)

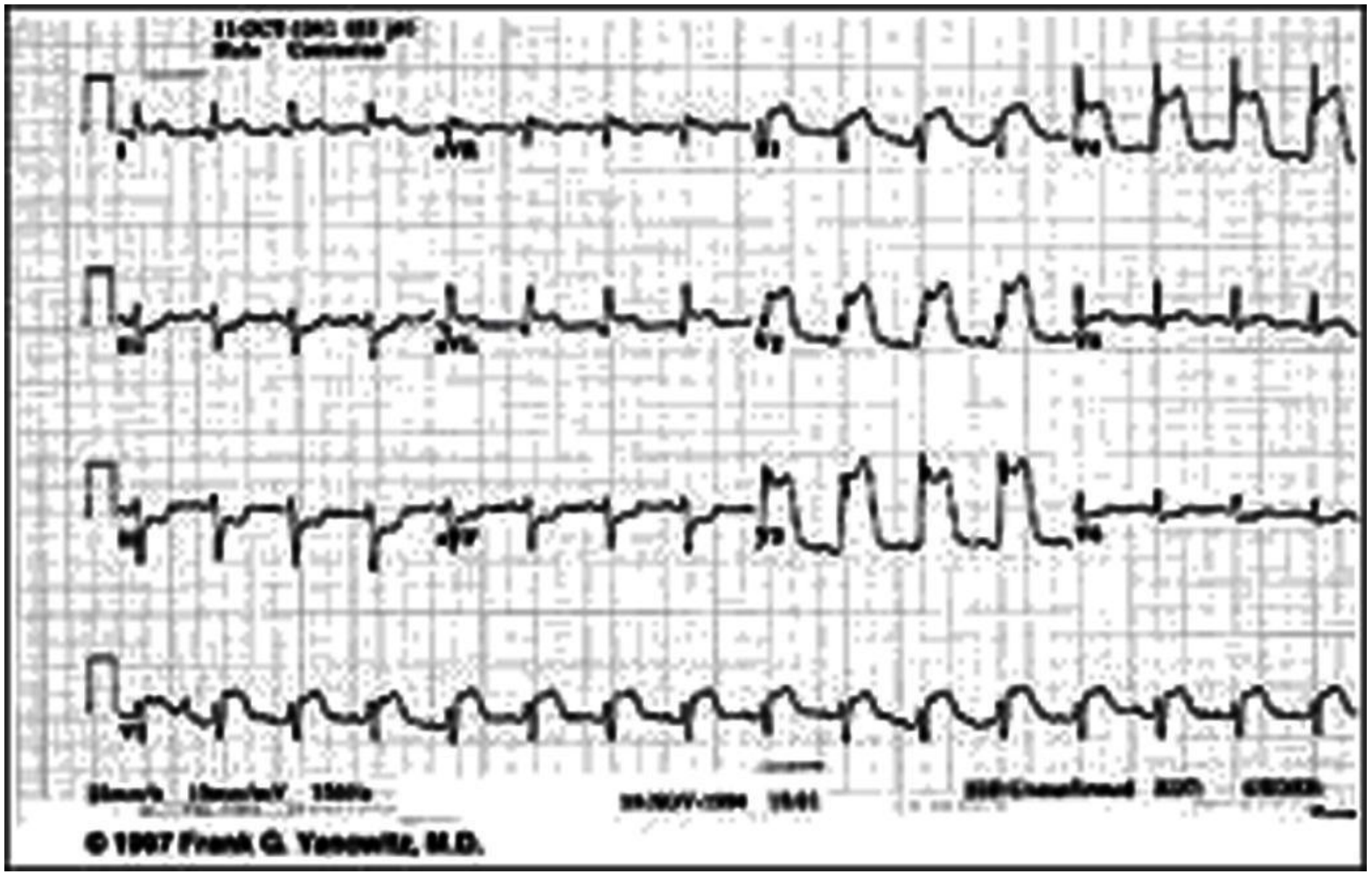
Examples of ECGs depicting MI



Antero-septal MI: Fully evolved

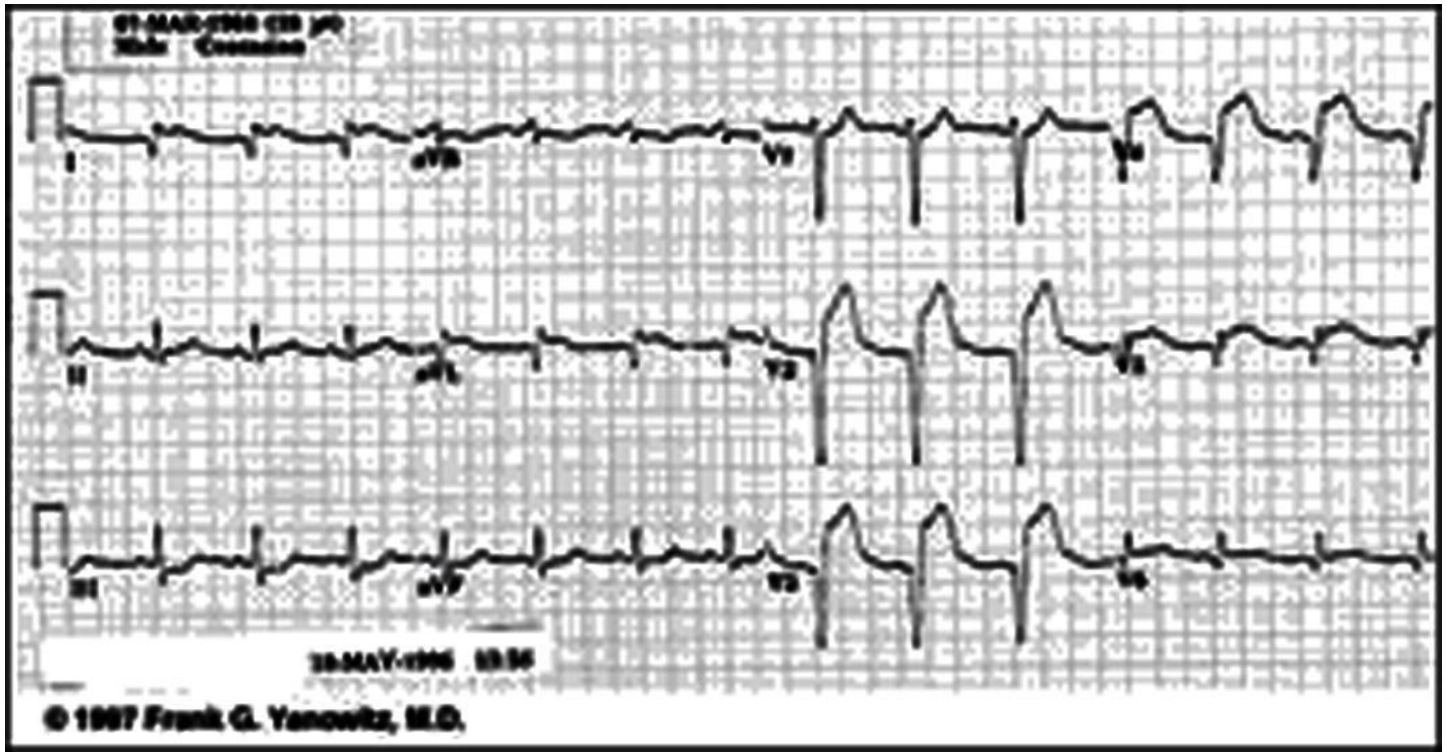
The QS complexes, resolving ST segment elevation and T wave inversions in V1-2 are evidence for a fully evolved antero-septal MI. The inverted T waves in V3-5, I, aVL are also probably related to the MI.

Acute anterior MI

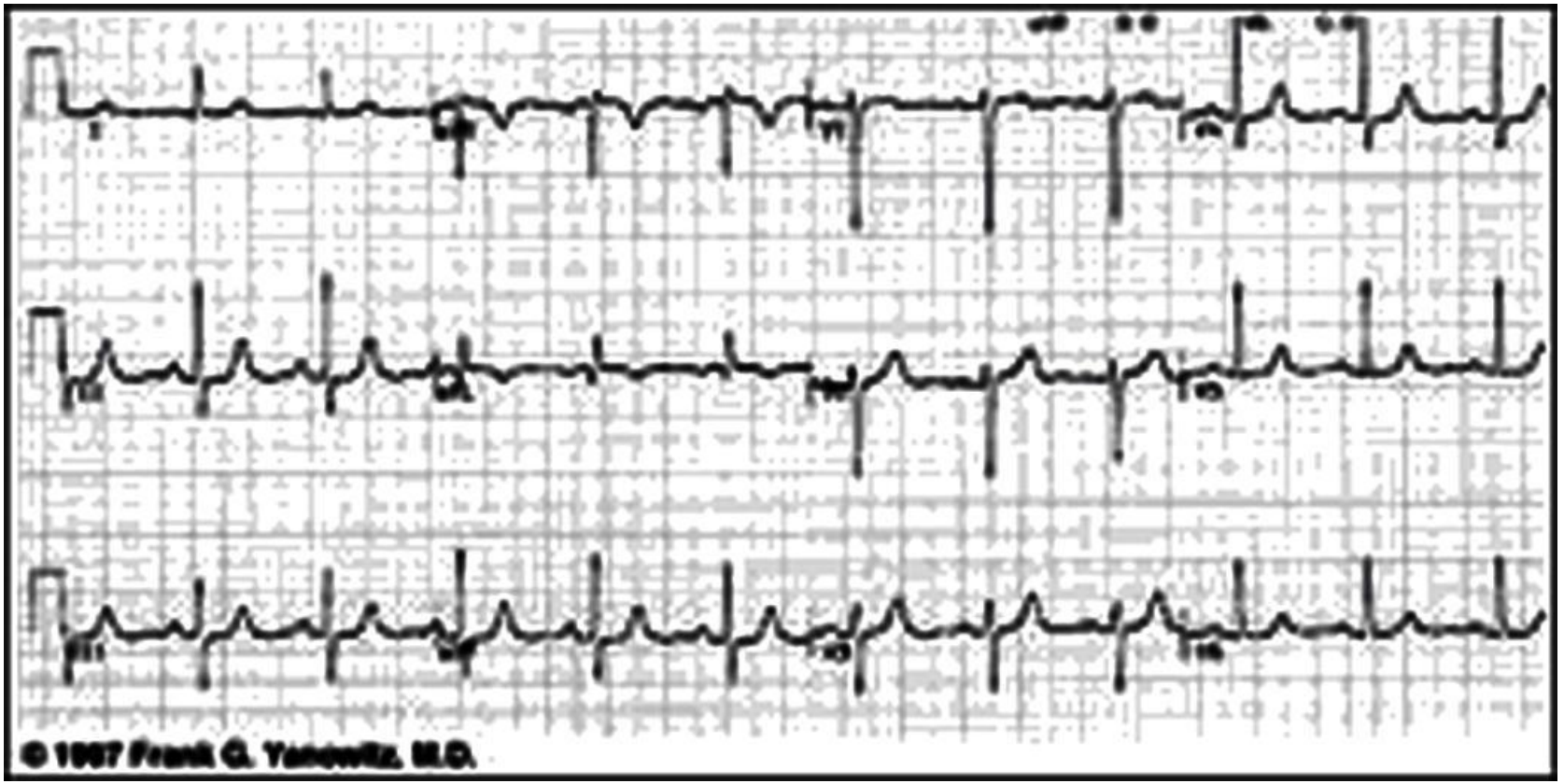


Extensive anterior/antero-lateral MI

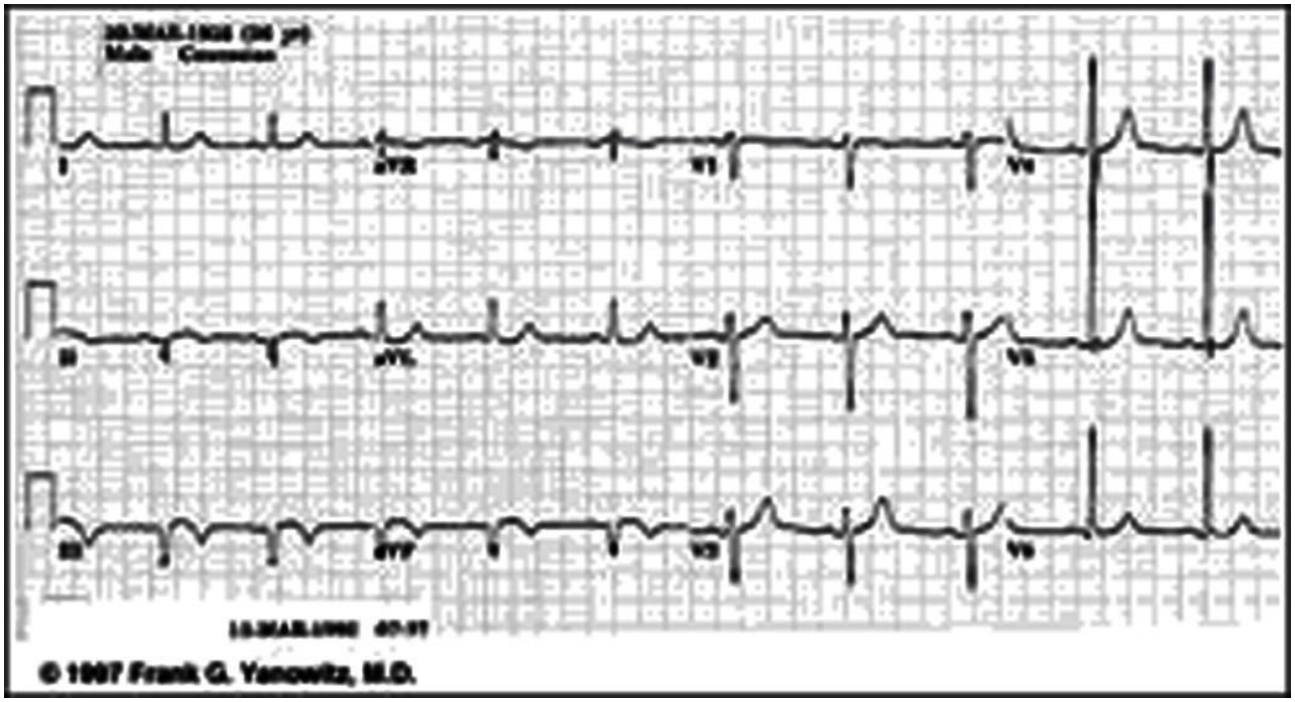
Significant pathological Q waves (V2-6, I, aVL) plus marked ST segment elevation are evidence for this large anterior/antero-lateral MI. The exact age of the infarction cannot be determined without clinical correlation and previous ECGs, but this is likely to be a recent MI.



High lateral wall MI



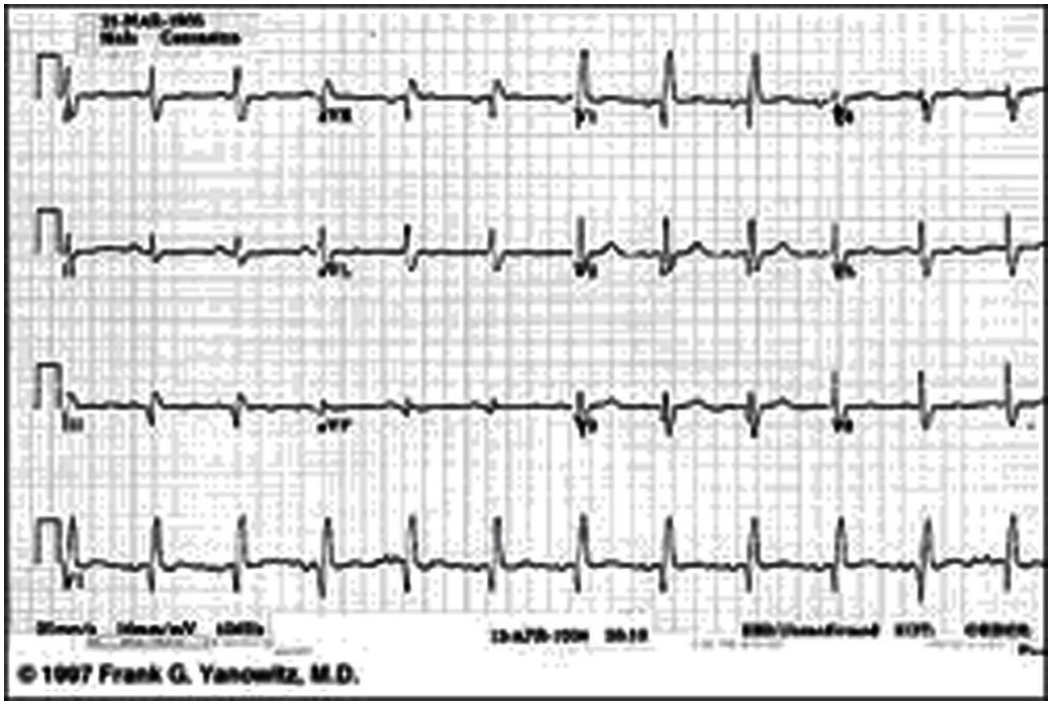
Inferior MI: Fully evolved



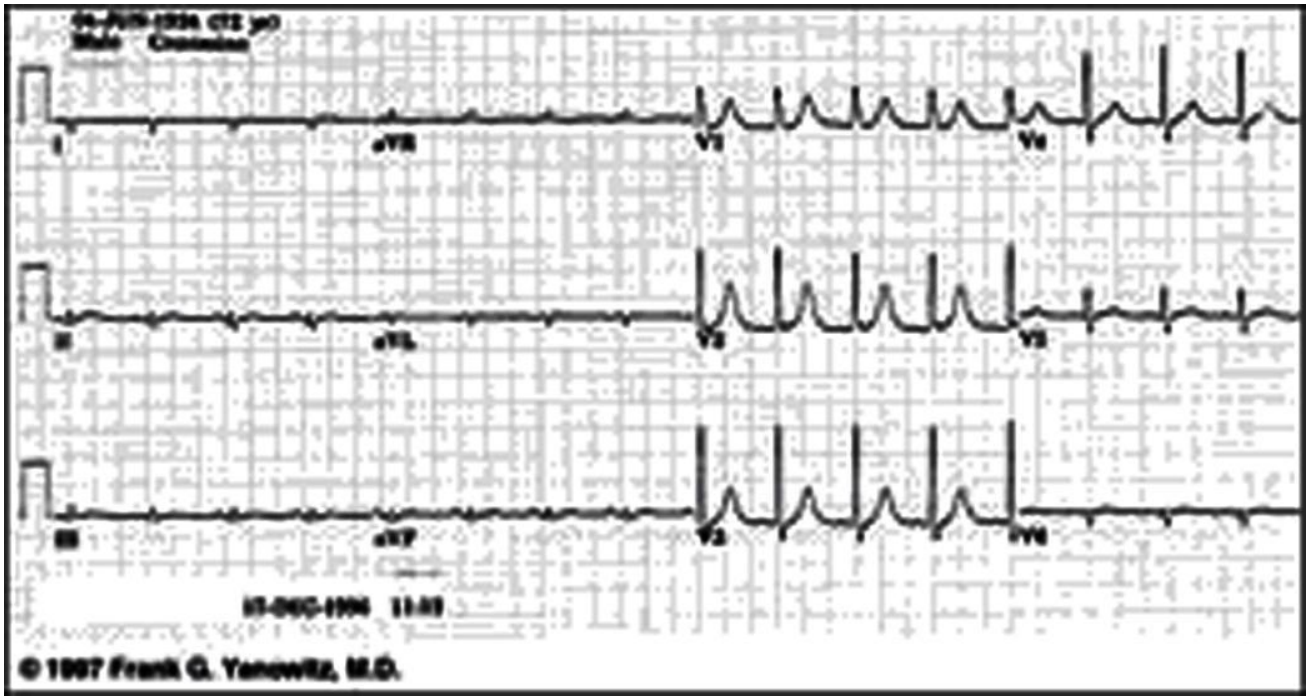
Significant pathological Q waves are seen in leads II, III and aVF along with resolving ST segment elevation and symetrical T wave inversion. This is a classic inferior MI.

Inferior & antero-septal MI + RBBB

Pathological Q waves are seen in leads II, III, aVF (inferior MI) and in leads V1-3 (antero-septal MI). RBBB is recognised by the wide QRS (>0.12 s) and the anterior/rightwards orientation of terminal QRS forces. When an antero-septal MI complicates RBBB (or vice versa), the rSR' complex in V1 (typical of RBBB) becomes a qR complex.



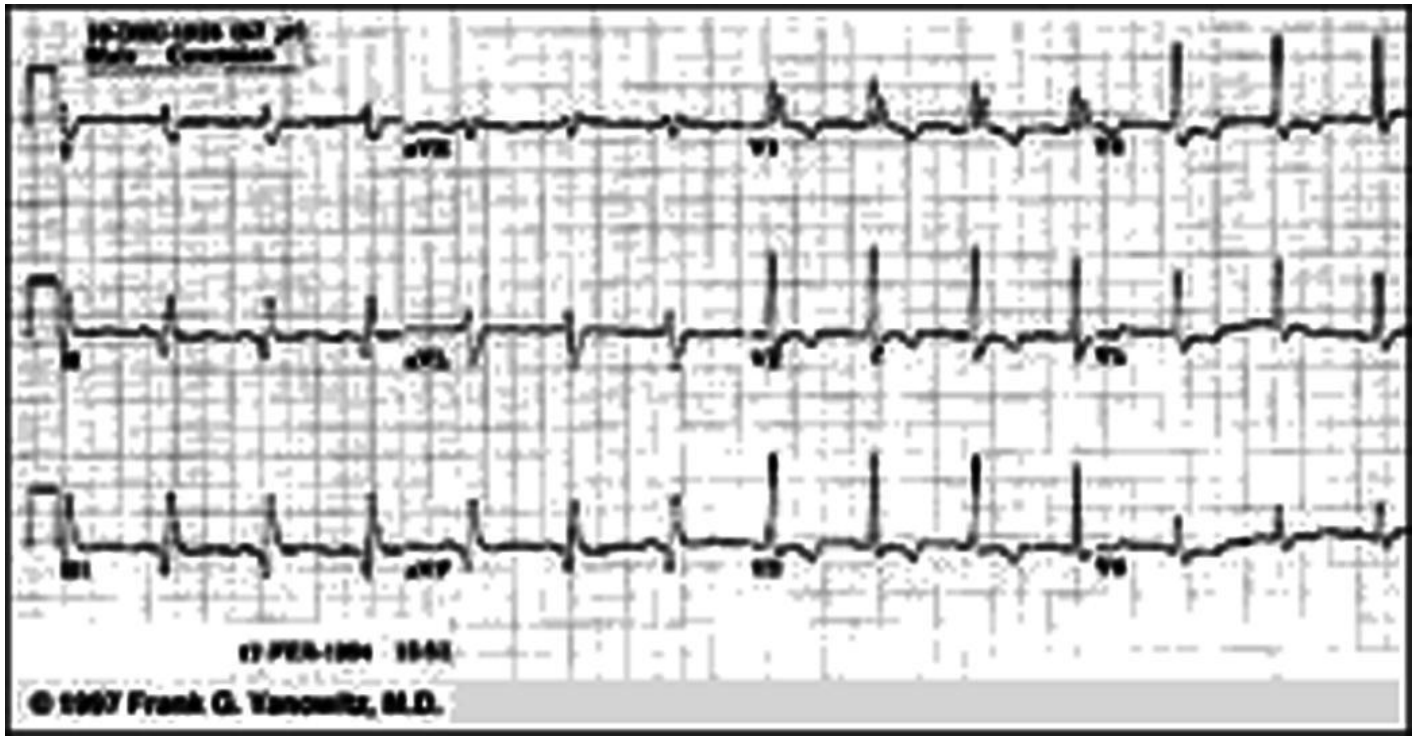
Postero-lateral MI: Fully evolved



The "true" posterior MI is recognised by pathological R waves in leads V1-2. These are the posterior equivalent of pathological Q waves (seen from the perspective of the anterior leads). Tall T waves in these same leads are the posterior equivalent of inverted T waves in this fully evolved MI. The loss of forces in V6, I, aVL suggest a lateral wall extension of this MI.

Infero-posterior MI with RBBB

This is an unusual RBBB because the initial R wave is taller than the R' wave in lead V1. This is the clue for true posterior MI. The tall initial R wave in V1 is a "pathological R" wave analagous to the "pathological Q" wave of an anterior MI.



Diagnostic criteria for MI

A definitive diagnosis of MI from the ECG can only be made on the basis of abnormalities in the QRS complex. The following changes are seen:

1) q waves which are either 0.04 s or longer in duration (excluding aVR and lead III) or have a depth which is more than 25% of the height of the following R wave (excluding aVR and lead III).
2) qs or QS complexes (excluding aVR and lead III).
3) Local area of inappropriately low R wave voltage.

Additional changes frequently associated with MI are:

a) ST segment elevation (convex upwards) in leads facing the infarcted zone.
b) ST segment depression occurs as a reciprocal change in leads mutually opposite to the primary leads showing evidence of infarction.
c) Horizontal ST segment depression may occur as a primary change in subendocardial infarction.

Reciprocal changes

In addition to the primary changes that occur in the ECG leads facing the infarcted myocardium, "reciprocal changes" may occur in leads opposite to the site of infarction. The changes are just the inverse of the primary changes.

Thus, "ST segment elevation and T wave inversion" will appear as "ST segment depression and tall pointed T waves", respectively.

The inferior limb leads on the one hand and the precordial leads, together with leads I and aVL, on the other hand are "mutually opposite". Thus, primary changes in one of the above groups will usually be accompanied by reciprocal changes in the other group.

It will be safe to assume that if on the ECG there is ST segment elevation in one group (as above) and ST segment depression in the other group, the elevation is the primary change and the ST segment depression is the secondary change.

True posterior MI

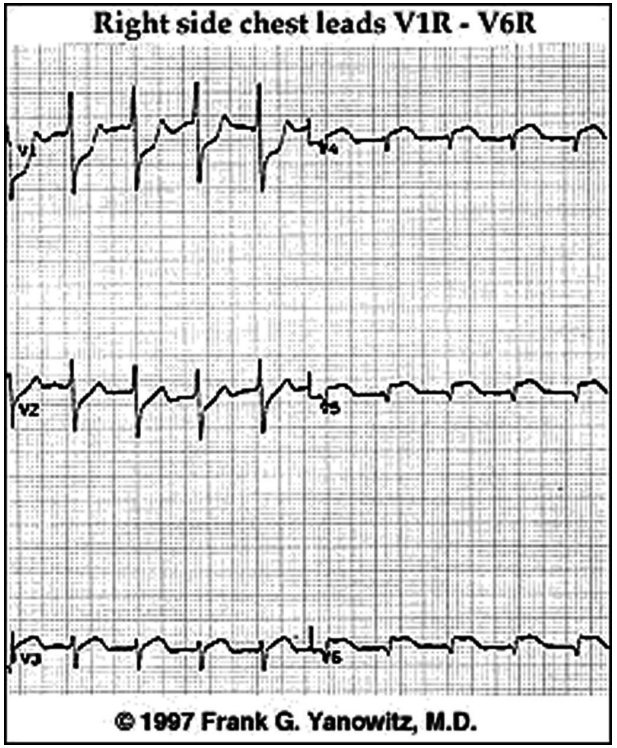
Infarction evident in the inferior leads (II, III and aVF) was previously called posterior infarction (now called inferior infarction).

However, true posterior infarction is quite rare and is not easily recognised, as none of the ECG leads are actually situated posteriorly.

Hence, it is only recognisable by looking for "reciprocal" changes in the anterior leads. Primary changes are not seen, as there are no actual posterior leads.

The changes in the ECG of a true posterior infarction are:

1) Abnormally tall and broad "R" waves in V1 (reciprocal to abnormally deep and wide q waves in a posterior lead, if there were any) and
2) ST segment depression in V1 in recent infarcts; in infarcts of intermediate age, tall T waves may be present in V1, V2 and V3.



Right-sided chest leads, V1R - V6R, are shown. The true posterior MI is evidenced by the marked ST segment elevation in V1R (actual V2) and V2R (actual V1). The RV MI is evidenced by the ST elevation in V3R to V6R.

Subendocardial infarction

Infarcts are most commonly intramural infarcts (transmural or subepicardial). Subendocardial infarcts are relatively rare and may encircle the interior of the left ventricle.

The ECG shows primary ST segment depression or deep symmetrical T wave inversion without any changes in the QRS complexes. Since these changes can also be produced by myocardial ischaemia without infarction, the diagnosis of a subendocardial infarction cannot be made with a single ECG (unless correlated with clinical or enzyme evidence of infarction).

When ST depression is the primary change, it will be seen in all or most leads except the cavity leads (aVR - always a cavity lead, aVL - a cavity lead in a vertical heart and aVF - a cavity lead in a horizontal heart). By definition, cavity leads inevitably show QS complexes.

Changes in myocardial ischaemia

Hypoxia of the myocardium may occur in the absence of infarction and necrosis. The changes may occur following stress (physical or emotional) or even spontaneously.
Significant degrees of ischaemia may exist with no evidence of ECG abnormalities. The changes, when present, are confined to the ST segment and T waves. There will be **no change in the QRS complexes**.

The following ECG changes may accompany myocardial ischaemia:
1) Flattening of T waves
2) Inverted T waves
3) Abnormally tall T waves
4) "Normalisation" of primarily abnormal T waves
5) Sloping ST segment depression
6) Horizontal ST segment depression
7) ST segment elevation
8) Any combination of the above changes