Electrocardiography in Ischaemic Heart Disease

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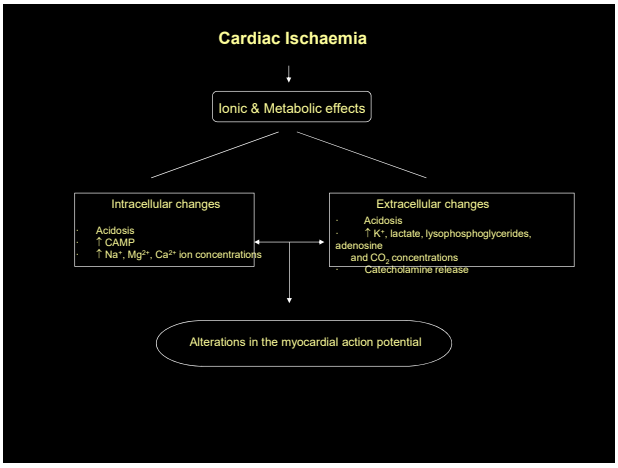
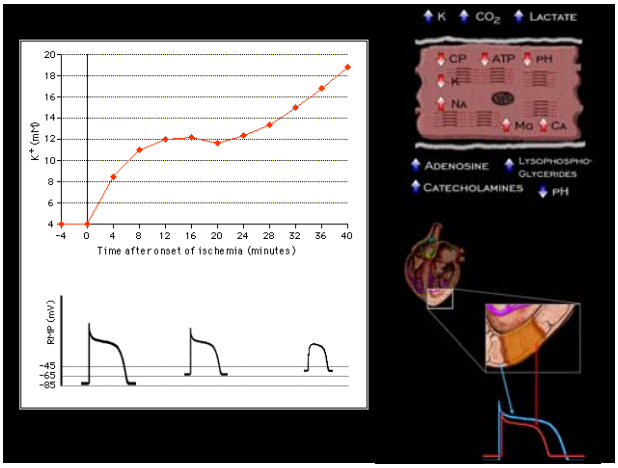
Myocardial Ischaemia

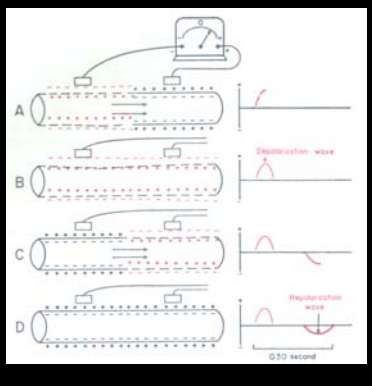
Myocardial ischaemia is defined as the reduction in the supply of oxygen to less than the amount required by myocardial cells to maintain aerobic metabolism.

Myocardial ischaemia is the term used to describe the sequence of events that occur when blood supply to the myocardium is inadequate to meet its metabolic demands

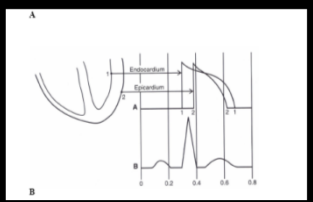




Recording the depolarisation and repolarisation waves from a single cardiac muscle fiber



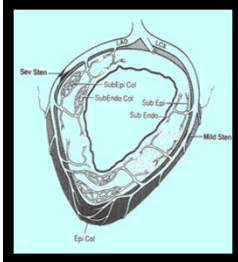
Ventricular Myocardial Depolarisation and Repolarisation

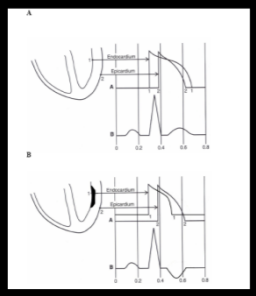


Depolarise from endocardium to epicardium

Repolarise from epicardium to endocardium

Myocardial Ischaemia and the T wave

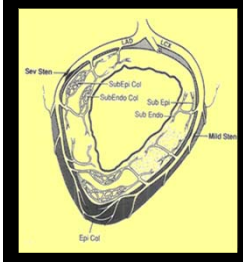


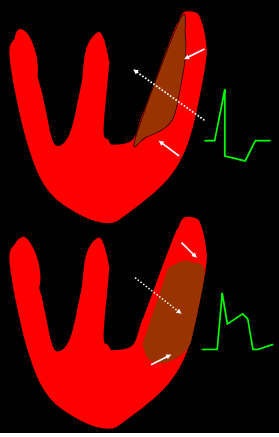


Depolarise from endocardium to epicardium

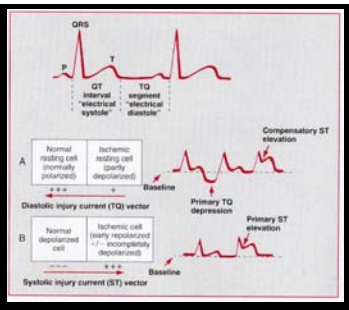
Repolarise from endocardium to epicardium

Myocardial Ischaemia and the ST Segment





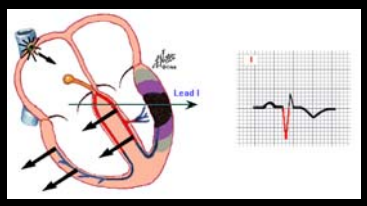
Pathophysiology of ST Elevation



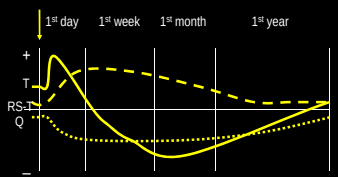
A. Diastolic current of injury. In this case (first QRS-T complex), the ST vector will be directed away from the relatively negative, partly depolarized, ischemic region during electrical diastole (TQ interval), and the result will be primary TQ depression. Conventional alternating-current electrocardiograms compensate for the baseline shift, and an apparent ST elevation (second QRS-T complex) results. ***B, Systolic current of injury***. In this case, the ischemic zone will be relatively positive during electrical systole because the cells are repolarized early and the amplitude and upstroke velocity of their action potentials may be decreased. This injury current vector will be oriented toward the electropositive zone, and the result will be primary ST elevation.

Myocardial Ischaemia and the Q wave

Pathological Q wave: ≥ 25% of R wave, ≥ 0.04 seconds



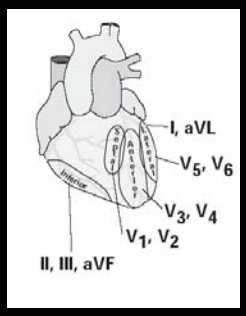
Myocardial Ischaemia/infarction – T, ST and Q waves

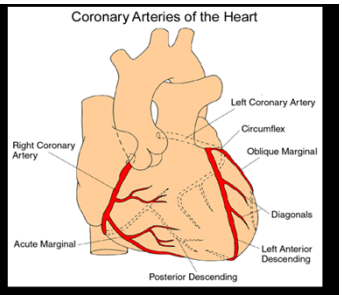


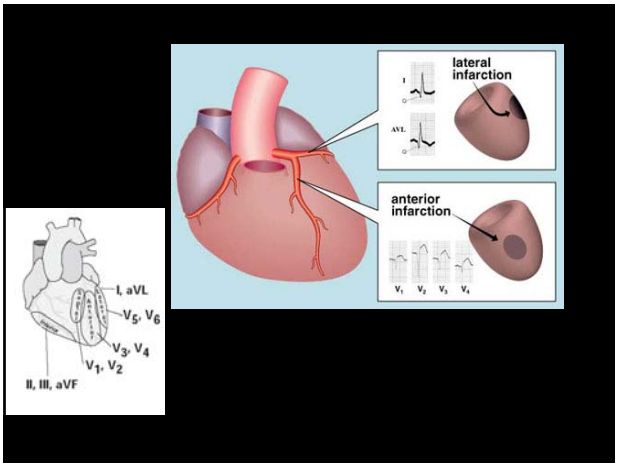
Location of Myocardial infarction

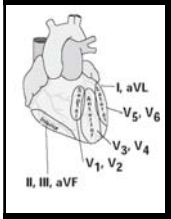
Type of MI	Artery	Affected areas	Leads
Anterior	Proximal LAD	Anterior wall of LV; anterior 2/3 of ventricular septum	V1-V4
Anteroseptal	Midsection of LAD	Portion of anterior wall of LV; anterior ventricular septum	V1/V2
Anterolateral	LAD diagonal	Anterolateral wall of LV	I,aVL,V5,V6
Lateral	Left circumflex artery	Lateral wall of LV	I, aVl
Inferior	RCA	Inferior (or diaphragmatic) wall of LV	II,III,aVF
Posterior	Left ventricular branches of RAD or distal LCxA	Posterior wall of LV	Tall R waves V1,V2 Tall T wave V1,V2 ST ↓ V1,V2

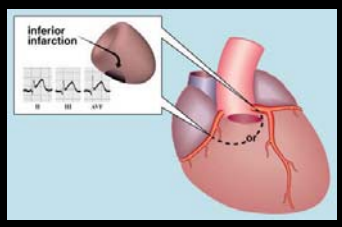
Myocardial infarction by site:



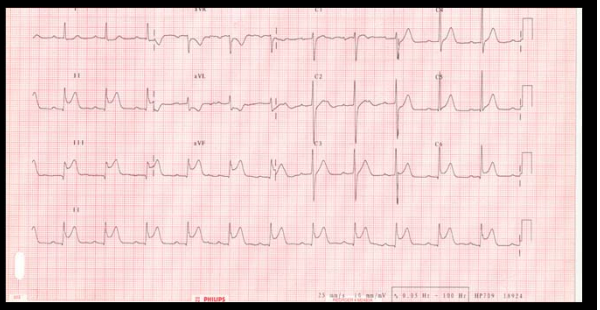




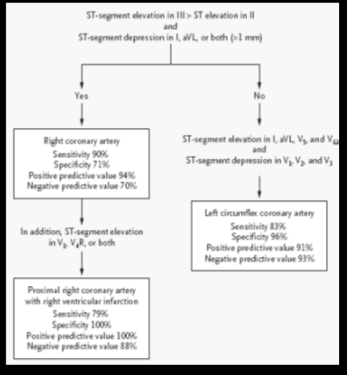


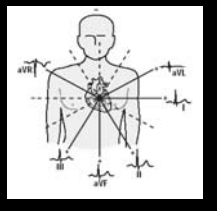


Inferior MI



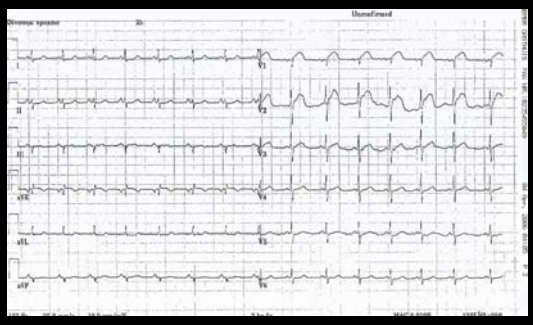
Inferior MI



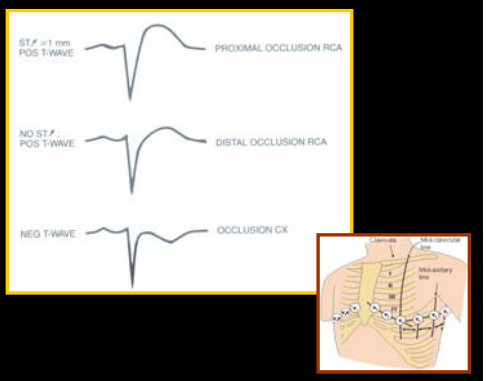


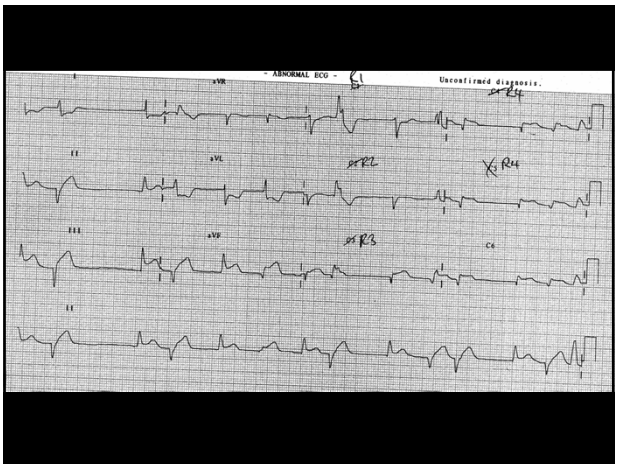
N Engl J Med 2003;348:933-40.

RV MI –occlusion of non dom RCA

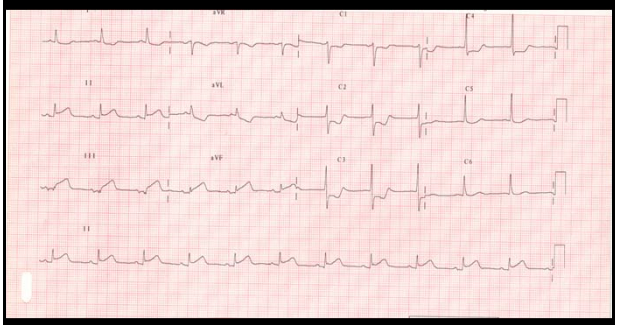


Value of RV4 lead

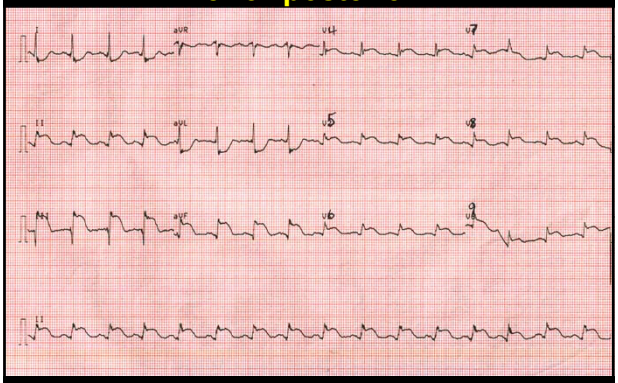




Inferior-posterior MI

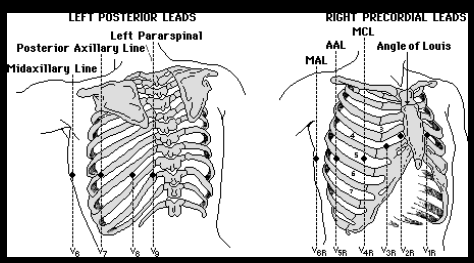


Inferior-posterior MI

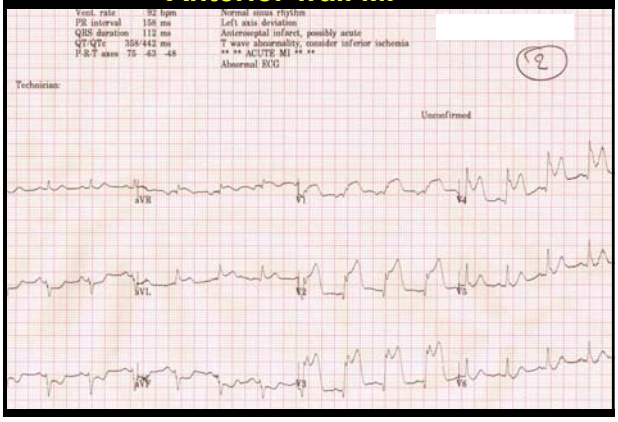


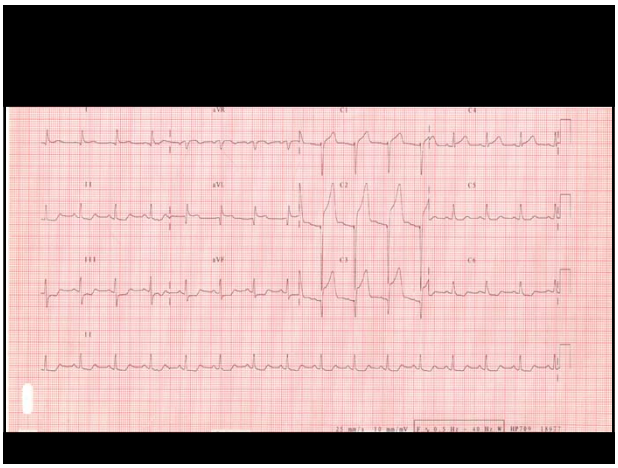
Posterior leads

Right and Posterior Leads Anatomical Positioning

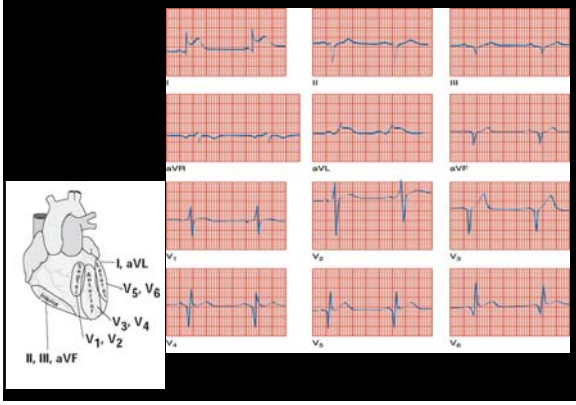


Anterior wall MI

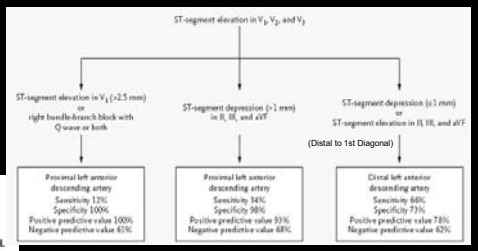


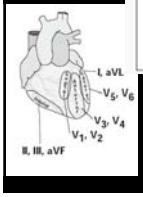


Lateral Infarction



Algorithm for Electrocardiographic Identification of the Infarct-Related Artery in Anterior Myocardial Infarction.

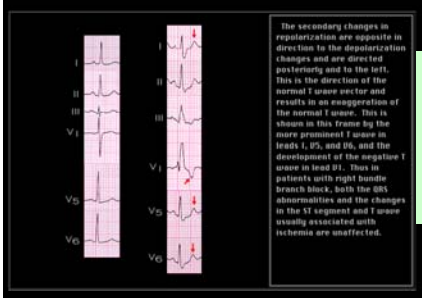




N Engl J Med 2003;348:933-40.

Myocardial Infarction and Bundle Branch Block

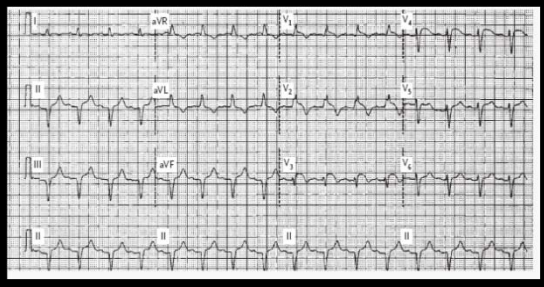
MI and RBBB



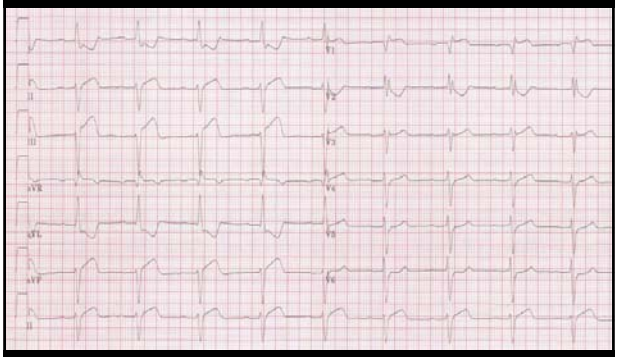
Criteria for RBBB

- V1 = Late intrinsicoid, rSR', sometimes wide R or qR
- V6 = Early intrinsicoid, wide S
- I = Wide S

Anterior Myocardial Infarction Associated with Right Bundle-Branch Block with a QR Pattern and ST-Segment Elevation.

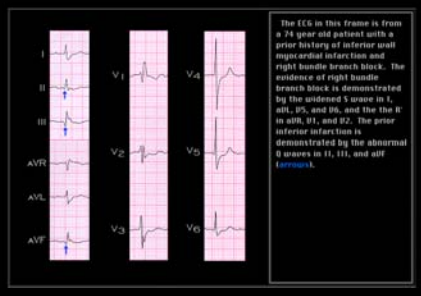


MI and RBBB

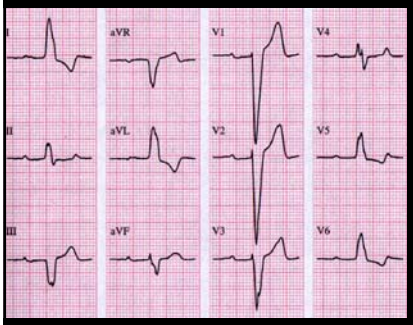


RBBB and inferior MI

MI and RBBB



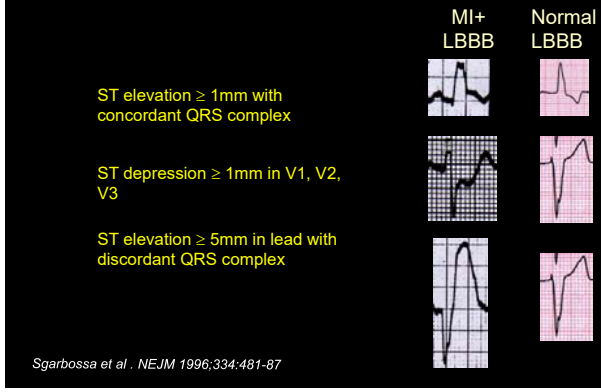
MI and LBBB



Criteria for LBBB

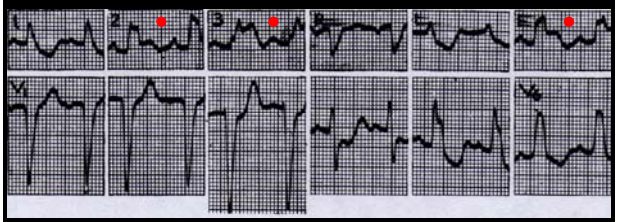
- V1 = QS or rS
- V6 = Late intrinsicoid, no Q waves, monophasic R
- I = Monophasic R wave, no Q

Electrocardiographic Diagnosis of Evolving Acute MI in The Presence of LBBB

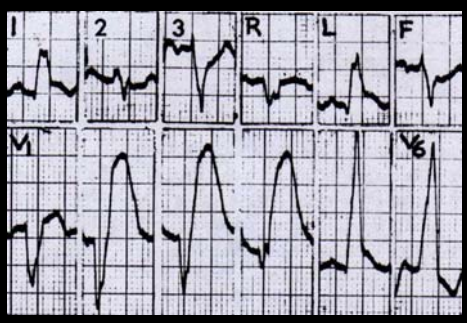


Sgarbossa et al . NEJM 1996;334:481-87

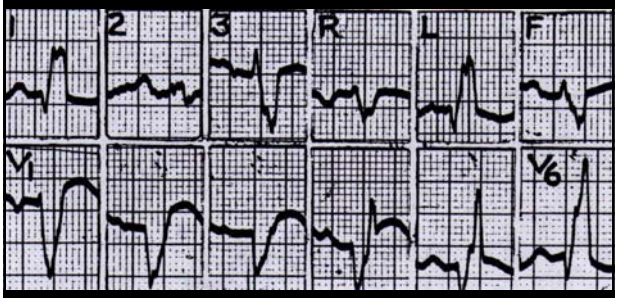
LBBB and AMI



LBBB and AMI

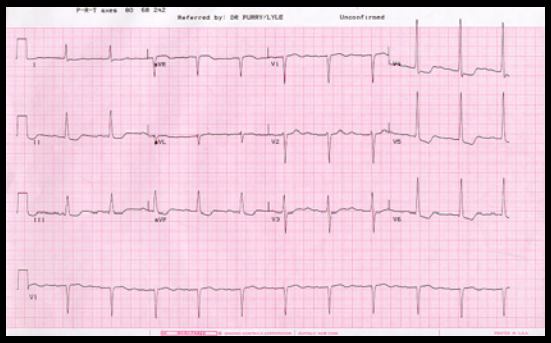


LBBB and AMI



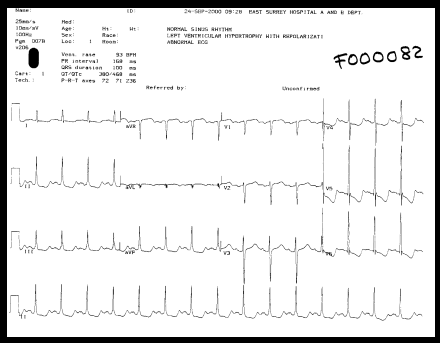
Other causes of ST-T wave changes simulating Ischaemia or Infarction

Left Ventricular Hypertrophy



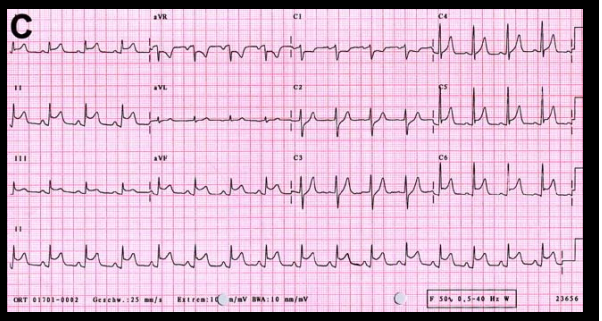
Aortic stenosis, hypertrophic cardiomyopathy, hypertension

60 year old man involved in RTA with severe head injury



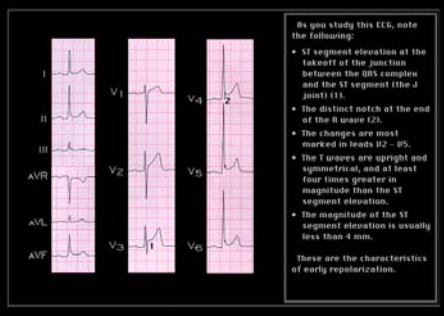
Intracranial bleed, CVA, raised intracranial pressure, Guillain Barre Syndrome, multiple sclerosis

Myocarditis/pericarditis

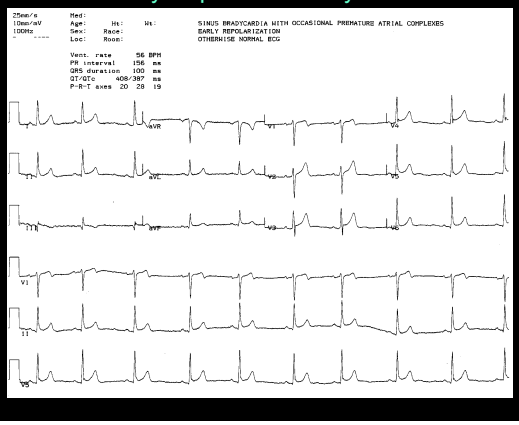


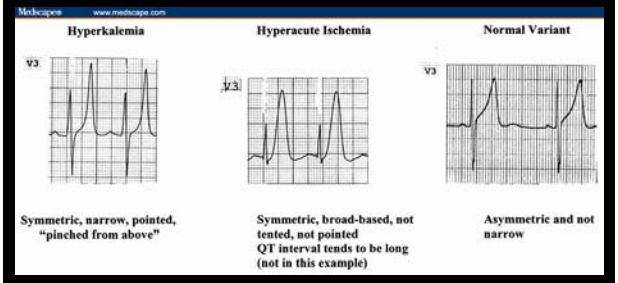
Global concave ST elevation, PR depression, aVr, V1 and sometimes V2 have ST depression

Early Repolarisation Syndrome



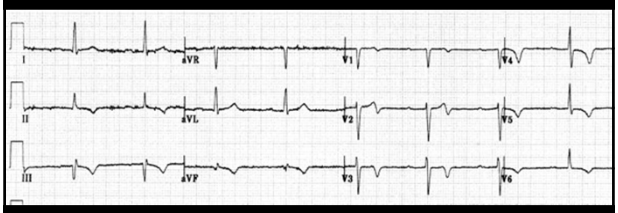
Early Repolarisation Syndrome



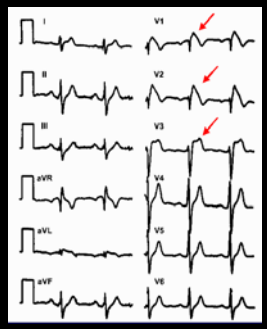


T wave Memory

68-year-old man with OSA, type 1 2nd degree AV block, pauses of 4 seconds with a DDDR PPM.



The Brugada Syndrome



- Elevated downsloping ST segment
- Negative T
- Normal or short QT interval
- Prominent changes V1-V3

Abnormalities leading to ST Segment elevation in Right Precordial leads

- RBBB
- LVH
- Acute myocardial infarction
- Left ventricular aneurysm
- Athletes
- Acute myocarditis, pericarditis
- Dissecting aortic aneurysm
- Acute pulmonary thromboemboli
- Various central and autonomic nervous system abnormalities
- Heterocylic antidepressant overdose
- Duchenne muscular dystrophy
- Friedreich ataxia
- Thiamine deficiency
- Hypercalcemia
- Hyperkalemia
- Compression of the right ventricular outflow tract by metastatic tumor
- Cocaine intoxication
- Chagas disease
- Eideken phenomenon (lead misplacement)
- Pectus excavatum
- Hypothermia

Other causes of ST-T wave changes simulating Ischaemia or Infarction

Persistent juvenile pattern
Drugs e.g. digoxin
Post tachycardia changes
Tako Tsubo cardiomyopathy, acute stress cardiomyopathy
Acute or chronic cor pulmonal/pulmonary emboli
Wolf-Parkinson-White syndrome
Infiltrative disease e.g. amyloidosis, sarcoidosis, etc.
Myocardial fibrosis
Pancreatitis and gallbladder disease
Cardiac tumour
After mital vavuloplasty
After DC cardioversion
Anaphylactic reactions

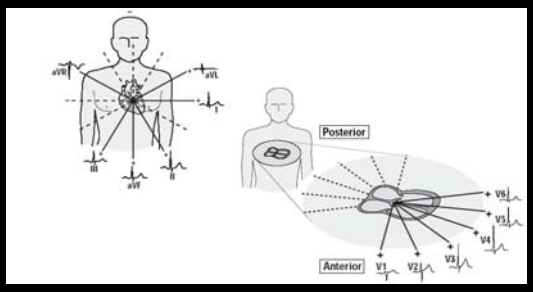
ETT and myocardial Ischaemia

Conclusion

Myocardial ischaemia and infarction cause a series of metabolic, ionic, neurohumeral and anatomical changes that influence the electrophysiological properties of the myocardial cells. This in turn cause changes in the QRS, ST and T waves on the surface ECG.

ECG changes of myocardial ischaemia and infarction are neither uniquely sensitive nor specific and can occur in a variety of other clinical settings.

ECG "seeing" the Heart



Myocardial infarction by site:

Location	QRS abnormalities	Vessel affected
Inferior	II, III, AVF	Terminal branches of LAD and or Right posterior descending
Anteroseptal	V1, V2	Distal LAD
Anterior	V1, V2, V3, V4	Proximal LAD
Anterolateral	I, AVL, V4-V6	Distal circumflex
High lateral wall	Lead 1 and AVL	Proximal circumflex branch of LCA
True posterior	No Q waves. Leads showing reciprocal changes only: Tall R waves, tall T waves and ST depression in V1, V2	Posterior descending

ECG predictors of AMI in The presence of LBBB

Feature	Comment	Reference
ST elevation ≥ 1mm with concordant QRS complex	5 points	Sgarbossa et al, 1996
ST depression ≥ 1mm in V1, V2, V3	3 points	Sgarbossa et al, 1996
ST elevation ≥ 5mm in lead with discordant QRS complex	2 points	Sgarbossa et al, 1996
Initial (0.04s) QRS notching in II or III or the precordial leads.		Dressler et al, 1950
rS in V5		Horan et al, 1971
Notching of 0.05s duration in the ascending limb of the S wave in V3 or V4	Cabrera's sign	Cabrera e al, 1953
Positive T waves in leads with a dominant upright QRS complex		Luy et al, 1973
Notching of the ascending limb of the R wave in 1, AVL or V6	Chapman's sign	Chapman et al, 1957

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Feature	Sen (95% CI)	Spec (95% CI)	PPA (95% CI)	NPA (95% CI)
ST elevation ≥ 1mm with concordant QRS complex	7 (1-21)	100 (95-100)	100 (16-100)	71 (61-80)
ST depression ≥ 1mm in V1, V2, V3	3 (0-17)	100 (95-100)	100 (2-100)	71 (61-79)
ST elevation ≥ 5mm in lead with discordant QRS complex	19 (7-37)	82 (71-90)	32 (13-57)	70 (59-80)
Initial (0.04s) QRS notching in II or III or the precordial leads.	39 (22-58)	57 (45-69)	28 (15-44)	68 (55-80)
rS in V5	26 (12-45)	79 (68-88)	35 (16-57)	71 (60-81)
Cabrera's sign	7 (1-21)	86 (76-93)	17 (2-48)	68 (58-78)
Positive T waves in leads with a dominant upright QRS complex	3 (0-17)	93 (85-98)	17 (0-64)	69 (59-78)
Chapman's sign	3 (0-17)	92 (83-97)	14 (3-58)	68 (58-78)
Overall ECG algorithm	10 (2-26)	100 (96-100)	100 (29-100)	72 (62-81)

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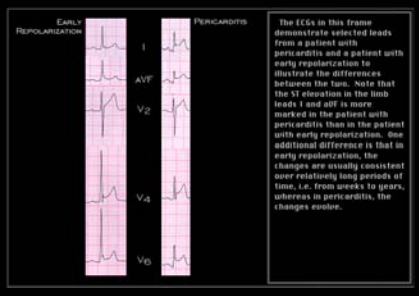
In summary, myocardial ischemia and infarction cause a series of metabolic, ionic, neurohumeral, and anatomic changes which influence the electrophysiologic properties of individual cells and impact on all portions of the ECG waveform. Within the ischemic region, the resting membrane potential becomes less negative, and the rate of rise of the action potential upstroke becomes slower. The action potential amplitude lessens and its duration shortens. As myocardial fibers become inexcitable and irreversibly damaged, conduction within the affected region is slowed and then blocked. These changes result in the creation of currents of injury across the ischemic borders, which cause cause ST segment elevation and depression, changes in the sequence of depolarization leading to changes in the QRS complex, and changes in repolarization, leading to T wave changes.

It is important to recognize that the ECG changes associated with ischemia and infarction are neither uniquely sensitive nor specific. They can occur in a variety of other clinical settings. It is also important to recognize that myocardial ischemia and infarction can occur without causing significant ECG abnormalities, i.e. may be electrically silent. An understanding of these ECG changes and the ability to descriminate between those due to ischemia and those due to other causes requires an appreciation of the mechanisms which underly the changes in the ECG waveform, and always the correlation of the ECG changes to the clinical setting in which they occur.



The ECGs in this frame demonstrate selected leads from a patient with pericarditis and a patient with early repolarization to illustrate the differences between the two. Note that the ST elevation in the limb leads I and aVF is more marked in the patient with pericarditis than in the patient with early repolarization. One additional difference is that in early repolarization, the changes are usually consistent over relatively long periods of time, i.e. from weeks to years, whereas in pericarditis, the changes evolve.

Location of bypass tract	Leads with negative delta wave
left lateral	I and/or aVL
left posterior	III and aVF
posterior septum	III and aVF
right lateral	aVR
right anterior septum	V1 and V2

When the delta wave is negative it may simulate the abnormal Q waves caused by a myocardial infarction. This table illustrates that a bypass tract located on the left lateral wall may lead to a negative delta wave in leads I and aVL, which will simulate a lateral wall infarction. A bypass tract located on the posterior septum will cause negative delta waves in leads III and aVF, which will simulate an inferior wall infarction.