UNIT 6 INTERPRETATION OF ECG

Structure

6.0 OBJECTIVES

After reading this unit, you should be able to:

- describe the diagnostic criteria of left atrial, right atrial, left ventricular and right ventricular hypertrophy;
- know the various ECG changes in myocardial infraction;
- identify the ECG changes in anterior and lateral wall infraction, posterior wall infraction, inferior wall infraction and right ventricular infraction; and
- know the ECG changes in electrolyte disturbances.

6.1 INTRODUCTION

The ECG is the graphical representation of the electrical potentials generated in the heart. The signals are detected by electrodes and then amplified and recorded by the electrocardiograph. This ECG is inexpensive, very easy to use and non-invasive test. It can detect the conduction disturbances, myocardial infection, infraction, atrial and ventricular hypertrophy of the heart. In addition to that ECG can also detect the electrolyte disturbance and effects of drugs on the heart.

6.2 ECG IN PULMONARY EMBOLISM (PE)

The ECG is of limited diagnostic value in patients with suspected Pulmonary Embolism (PE). Many of the classically described ECG changes in patients with suspected PE are equally

common in patients suspected of having PE but in whom the diagnosis is ultimately excluded. Even the two ECG changes noted commonly in pulmonary embolism, namely tachycardia and incomplete right bundle branch block, are infrequently observed and are only slightly more frequent in patients with PE. The ECG is thus a poor diagnostic test for pulmonary embolism. Changes have low sensitivity and low specificity. The greatest utility of the ECG in the patient with suspected PE is ruling out other potential life threatening diagnoses such as myocardial infarction.

Findings

a) ECG shows non-specific changes in 80 per cent

b) Classic Findings – S1 Q3 T3 (seen in under 20 per cent of cases)

   i) S-Wave in Lead I

   ii) Q-Wave in Lead III

   iii) T-wave inversion in Lead III

c) Common Findings

   i) Sinus tachycardia

   ii) Right sided strain pattern

      — Right bundle branch block

      — Right axis deviation

   iii) Findings that mimic myocardial infarction

      — ST segment changes

      — T-wave changes

   iv) Atrial fibrillation(new onset)

The S1Q3T3 was first described by McGinn and White in *JAMA* in 1935.

The S1Q3T3 sign is a manifestation of combined right heart pressure and volume overload of acute onset leading to repolarization abnormalities. In other words, this is a reflection of acute cor pulmonale. An S-wave in lead I signifies a complete or more often incomplete RBBB. In lead III, look for a Q-wave, slight ST elevation, and an inverted T-wave. *Any cause of acute cor pulmonale can cause the S1Q3T3 finding on the ECG.* This includes PE, acute bronchospasm, pneumothorax, and other acute lung disorders. In addition, transient LPFB may cause this finding as well.

Check Your Progress 1

What are the ECG changes in pulmonary embolism?

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6.3 ECG IN ACUTE PERICARDITIS

Acute pericarditis typically presents as sharp central chest pain that worsens with recumbency and deep breathing and is relieved by leaning forward. The pathognomonic physical finding is the pericardial friction rub, which is usually auscultated along the lower left sternal border. The ECG is useful in the diagnosis of acute pericarditis, with abnormalities found in approximately 90 per cent of cases. Changes on ECG classically occur in four stages. All cases of acute pericarditis do not show all the 4 stages. In fact, all four stages are present in only 50 per cent of patients or less. The stage I occurs during the first few days of pericardial inflammation and is characterized by diffuse ST-segment elevation. This stage may last up to two weeks. Stage II is characterized by return of the ST-segments to baseline and flattening of the T-wave and may last from days to several weeks. Stage III usually begins at the end of the second or third week and is characterized by inversion of the T-waves in the opposite direction of the ST-segment; this stage may last several weeks. Stage IV represents the gradual resolution of the T-wave changes and may last up to three months. The most sensitive ECG change characteristic of acute pericarditis is ST-segment elevation, which reflects the abnormal repolarization that develops secondary to pericardial inflammation. The following features differentiate the pericarditis and acute myocardial ST elevations.

1) The ST-segment elevation that occurs during acute pericarditis is usually “concave”, compared with the “convex” appearance of the ST-segment that occurs during the acute injury stage of a myocardial infarction.

2) In acute pericarditis there is widespread ST-segment elevation not corresponding with any specific arterial territory, which usually occurs in association with acute myocardial infarction.

3) Reciprocal changes are absent in acute pericarditis, while they are frequent with acute myocardial infarction.

4) The ST-segments in acute pericarditis return to baseline in a few days and are followed by diffuse T-wave inversion, in conjunction with the ST-segment at baseline. In contrast, T inversion in infarction occurs before ST-segment returns to baseline.

5) Another feature that may aid in differentiating acute pericarditis from acute myocardial infarction is the absence of Q-waves.

6) Loss of R-wave progression may occur with acute myocardial infarction, but this feature is not present with acute pericarditis.

There may also be ST-segment depression in leads aVR and V1. In the absence of underlying cardiac disease, the P-wave and QRS complexes are normal. Depression of the PR-segment is very specific of acute pericarditis and is attributed to subepicardial atrial injury and occurs in all leads except aVR and V1. These leads may exhibit PR-segment elevation. Symptoms subside within two weeks and ECG changes resolve over a period of weeks to months. Development of atrial and ventricular arrhythmias, heart blocks and fascicular blocks does not occur in acute pericarditis. Presence of these changes in the setting of acute pericarditis indicates the presence widespread underlying myocardial involvement.

Check Your Progress 2

What are the differences between the ST segment changes in acute myocardial infarction and acute pericarditis?

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6.4 ATRIAL HYPERTROPHY

The P-wave represents the wave of depolarization that spreads from the sino atrial node, throughout the atria, and is usually 0.08 to 0.1 seconds (80-100 ms) in duration. Right atrial depolarization forms the initial portion of the P-wave. The left atrial depolarization forms the terminal portion of the P-wave. The normal P-wave axis falls between + 45° and + 60°. P-wave in normal individuals is upright in I, II, aVl and aVf. In lead III, it could be upright or negative. In the right precordial leads (V1 and sometimes V2) the P-wave is biphasic with an initial positive deflection followed by a later negative one. In the remaining chest leads, normal P-waves are always upright. Atrial repolarisation (Ta-wave) produces low amplitude signals and coincides with normal QRS wave; hence atrial repolarisation normally does not manifest on a surface ECG. However, in complete heart block Ta waves may be seen on PR-segment as short deflections just beyond the P-waves with polarity opposite to that of P-wave. P-wave abnormalities occur in atrial enlargement and arrhythmias. The following cartoon shows patterns of atrial enlargement in lead II and lead V1.

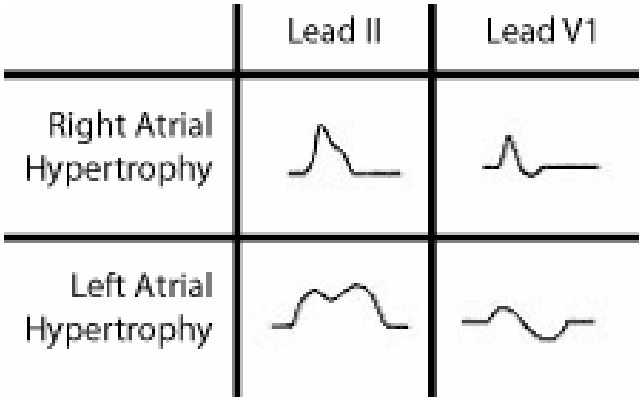

Fig. 6.1: Right and left atrial hypertrophy in lead II and VI

Left Atrial Enlargement (LAE)

Diagnostic Criteria

- The terminal portion of the P-wave in lead V1 must be one small box wide by one small box (0.04second x 0.04mv) deep or larger to qualify as left atrial enlargement.
- This force can be calculated by multiplying the time in seconds by the depth in millimeters. If this product is more negative than -0.04, LAE is present.
- A notched broad based P-wave in leads I and II with a duration of 0.12 milliseconds or more is called “P mitrale”.
- LAE can shift the P-wave axis to +15^{o} or less.
- Comparision of ECG abnormalities to echocardiographic criteria for left atrial enlargement demonstrates limited sensitivity but high specificity for the ECG criteria.

Common Causes of LA Enlargement

- Valvular Disease
  - Mitral stenosis
  - Mitral regurgitation
- Decreased Left Ventricular Compliance
  - Longstanding hypertension
  - Hypertrophic cardiomyopathy
  - Aortic stenosis
  - Aortic regurgitation
  - Infiltrative heart disease
  - Restrictive Cardiomyopathy
- All of these conditions increase either pressure or volume loading on the atria leading to enlargement and/or hypertrophy.

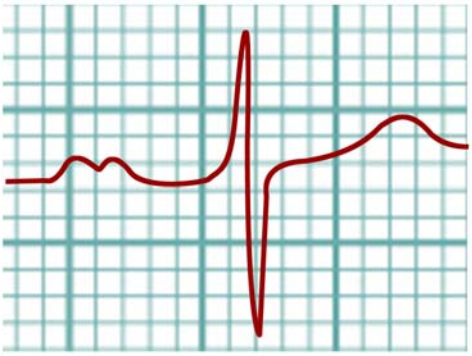

Fig. 6.2: Cartoon showing the pattern of P-wave in lead II in left atrial enlargement

Right Atrial Enlargement (RAE)

Diagnostic Criteria

- The P-wave in leads II, II and aVF is peaked with a height greater than 2.5mm. “P pulmonale”.
- The P-wave axis is +75 or greater.
- The positive aspect of the P-wave in lead V1 or V2 is >1.5mm in height.

Differential Diagnosis

- Valvular Disease
  - — Tricuspid stenosis
  - — Tricuspid regurgitation
- Pulmonary Hypertension
  - — COPD
  - — Pulmonary emboli
  - — Interstitial lung disease
  - — Sleep apnea
  - — Mitral valve disease with pulmonary hypertension
  - — Restrictive cardiomyopathy
- Congenital Heart Disease
  - — Ebstein’s anomal
  - — Tricuspid atresia
  - — Pulmonary atresia

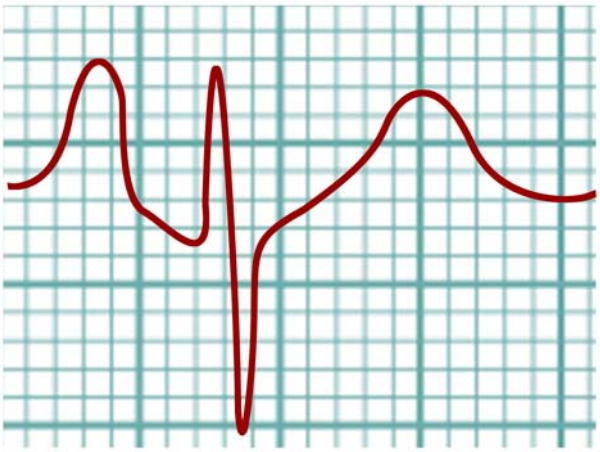

Fig. 6.3: Cartoon showing the pattern of P-wave in right atrial enlargement

Biatrial Enlargement

Diagnostic Criteria

Because the P-wave is composed of distinct right and left atrial components, the diagnosis of biatrial enlargement is simply made by looking for the criteria for both right and left atrial enlargement.

- A large biphasic P-wave in lead V1 with the initial component greater than 1.5mm in height and the terminal component at least 1mm in depth and 0.04 sec in duration.
- A P-wave amplitude of >2.5mm and duration of >0.12 seconds in the limb leads II.

Check Your Progress 3

1) What are the ECG criteria for left atrial enlargement?

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2) What are the criteria for right atrial enlargement?

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6.5 VENTRICULAR HYPERTROPHY

The ECG criteria for diagnosing right or left ventricular hypertrophy are highly specific but carry a low sensitivity. In other words, if the criteria are present it is very likely that ventricular hypertrophy is present. It also means that ECG may miss the presence of hypertrophy.

Left Ventricular Hypertrophy (LVH)

ECG features include:

1) ≥ QRS amplitude (voltage criteria; i.e., tall R-waves in LV leads, deep S-waves in RV leads)

2) Delayed intrinsicoid deflection in V6 (i.e., time from QRS onset to peak R is ≥ 0.05 sec)

3) Widened QRS/T angle (i.e., *left ventricular strain pattern,* or ST-T oriented opposite to QRS direction)

4) Leftward shift in frontal plane QRS axis

5) Evidence for left atrial enlargement

6) ESTES Criteria for LVH ("diagnostic", ≥ 5 points; "probable", 4 points)

+ ECG Criteria	**Points**
Voltage Criteria (any of): • R or S in limb leads ≥ 20 mm • S in V1 or V2 ≥ 30 mm • R in V5 or V6 ≥ 30 mm	3 points
ST-T Abnormalities: Without digitalis With digitalis	3 points 1 point
Left Atrial Enlargement in V1	3 points
Left axis deviation	2 points
QRS duration 0.09 sec	1 point
Delayed intrinsicoid deflection in V5 or V6 (> 0.05 sec)	1 point

7) CORNELL Voltage Criteria for LVH (sensitivity = 22 per cent, specificity = 95 per cent)

-S in V3 + R in aVL > 24 mm (men)

-S in V3 + R in aVL > 20 mm (women)

8) Other Voltage Criteria for LVH

a) Limb-lead voltage criteria:

-R in aVL ≥11 mm *or*, if *left axis deviation*, R in aVL ≥13 mm *plus* S in III ≥15 mm

-R in I + S in III >25 mm

b) Chest-lead voltage criteria:

-S in V1 + R in V5 or V6 ≥ 35 mm

Right Ventricular Hypertrophy

ECG features include:

1) Right axis deviation (> 90 degrees)
2) Tall R-waves in RV leads; deep S-waves in LV leads
3) Slight increase in QRS duration
4) ST-T changes directed opposite to QRS direction (i.e., wide QRS/T angle)
5) May see incomplete RBBB pattern or qR pattern in V1
6) Evidence of right atrial enlargement

Specific ECG features (assumes normal calibration of 1 mV = 10 mm):

Any one or more of the following (if QRS duration < 0.12 sec):

a) Right axis deviation (>90 degrees) in presence of disease capable of causing RVH
b) R in aVR $\geq$ 5 mm, or
c) R in aVR > Q in aVR

Any one of the following in lead V1:

a) R/S ratio > 1 *and* negative T-wave qR pattern
b) R > 6 mm, *or* S < 2mm, *or* rSR' with R' >10 mm

Other Chest Lead Criteria

a) R in V1 + S in V5 (or V6) 10 mm
b) R/S ratio in V5 or V6 < 1
c) R in V5 or V6 < 5 mm
d) S in V5 or V6 > 7 mm

ST-segment depression and T-wave inversion in right precordial leads is usually seen in severe RVH such as in pulmonary stenosis and pulmonary hypertension.

Check Your Progress 4

1) What are ECG criteria for left ventricular hypertrophy?

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2) What are the ECG changes in right ventricular infarction?

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6.6 ECG IN PATIENTS WITH CHEST PAIN

There are many causes of chest pain. Many non-cardiac conditions can mimic a myocardial infarction, and so the ECG can be extremely useful when making a diagnosis. However, the ECG is less important than the history and physical examination, because the ECG can be normal in the first few hours of a myocardial infarction.

Acute chest pain can be caused by:

- Myocardial infarction
- Pulmonary embolism
- Pneumothorax
- Pleurisy
- Pericarditis
- Aortic dissection
- Ruptured oesophagus
- Oesophagitis
- Collapsed vertebra
- Herpes zoster

Chronic or *recurrent* chest pain may be:

- Exertional angina
- Radiculopathy
- Muscular pain
- Oesophageal reflux
- Nonspecific pain

Acute Chest Pain

The typical pain of *myocardial infarction* is easy to recognize, the features being:

- central
- radiates to neck, jaw, teeth, arm(s) or back
- severe
- associated with nausea, vomiting and sweating.

Unfortunately not all patients have typical pain, and pain can even be absent. The diagnosis of a myocardial infarction depends on the history and examination, on the measurement of biochemical markers of cardiac muscle damage (especially the troponins) and on the ECG. A rise in troponin I or troponin T levels in patients with a history suggestive of a myocardial infarction is now taken to mean that infarction has occurred, but treatment still depends on the ECG.

The term ‘acute coronary syndrome’ is now used to include:

1) Myocardial infarction with ST-segment elevation on the ECG.
2) Myocardial infarction (as shown by a troponin rise) with only T-wave inversion or ST-segment depression.
3) Chest pain with ischaemic ST-segment depression but no troponin rise (what used to be called ‘unstable angina’).
4) Sudden death due to coronary disease.

Stable angina and ‘chest pain of unknown cause’ remain entirely proper diagnostic labels for those patients who are admitted to hospital with chest pain, but for whom the term ‘acute coronary syndrome’ is inappropriate.

The Development of ECG Changes in Myocardial Infarction

The sequence of features characteristic of ‘full thickness’, or ‘ST-segment elevation’, myocardial infarction is:

- Normal ECG
- Delayed intrinsicoid deflection
- Tall peaked T-waves (so called hyperacute T-waves)
- ST-segment elevation
- Development of Q-waves
- ST-segment returns to the baseline
- T-waves inversion
- Loss of R-wave voltage in anterior chest leads.

The ECG leads that show the changes typical of a myocardial infarction depend on the part of the heart affected.

Infarct

Accurate ECG interpretation in a patient with chest pain is critical. Basically, there can be three types of problems — *ischaemia* is a relative lack of blood supply (not yet an infarct), *injury* is acute damage occurring right now, and finally, *infarct* is an area of dead myocardium. It is important to realize that certain leads represent certain areas of the left ventricle; by noting which leads are involved, you can localize the process. The prognosis often varies depending on which area of the left ventricle is involved (i.e., anterior wall myocardial infarct generally has a worse prognosis than an inferior wall infarct).

Anterior and Lateral Infarction

The changes of anterior infarction are seen in leads V2–V5. Lead V1, which lies over the right ventricle, is seldom affected. The lateral wall of the left ventricle is often damaged at the same time as the anterior wall, and then leads I, VL and V6 show infarction changes. Persistent ST-segment elevation is quite common after an anterior infarction: it sometimes indicates the development of a left ventricular aneurysm, but it is not reliable evidence of this. The following ECG shows acute anterolateral infraction.

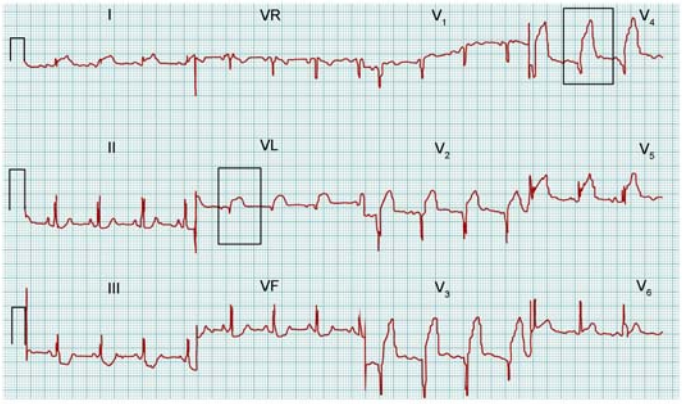

Fig. 6.4: Acute anterolateral infraction

An old anterior infarction often causes only what is called poor R-wave progression. A normal ECG would show progressive increase in the size of the R-wave from lead V1 to V5 or V6. In this case the R-wave remains very small in leads V3 and V4, but becomes a normal size in V5. This loss of 'progression' indicates the old infarction. The ECG shows old anterior infarction.

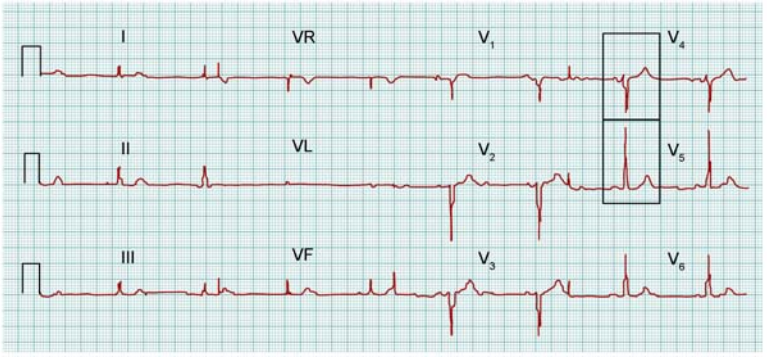

Fig. 6.5: Old anterior infraction

The time taken for the various ECG changes of infarction to occur is extremely variable, and the ECG is an unreliable way of deciding when an infarction occurred. Serial records showing progressive changes are the only way of timing the infarction from the ECG. Two other caveats: One is that normally the R-wave gets larger as one moves from V1 to V6. If there is no R-wave "progression" from V1 to V6 this can also mean infarct. The second caveat is that, with a left bundle branch block, it is difficult to diagnoseMI. In a patient with chest pain and left bundle branch block, one must rely on cardiac enzymes (blood tests) and the history.

Posterior Infarction

It is possible to 'look at' the back of the heart by placing the V lead on the back of the left side of the chest, but this is not done routinely because it is inconvenient and the complexes recorded are often small. An infarction of the posterior wall of the left ventricle can, however, be detected in the ordinary 12-lead ECG because it causes a dominant R-wave in lead V1. The shape of the QRS complex recorded from lead V1 depends on the balance of electrical forces reaching the ECG electrode. Normally the right ventricle is being depolarized towards lead V1, so causing an upward movement (an R-wave) on the record; at the same time the posterior wall of the left ventricle is being depolarized, with the wave of excitation moving away from the electrode and so causing a downward movement (an S-wave) on the record. The left ventricle is more muscular than the right and therefore, exerts a greater influence on the ECG, so in lead V1 the QRS complex is normally predominantly downward, i.e. there is a small R-wave and a deep S-wave. In a posterior infarction, the rearward-moving electrical forces are lost so lead V1 'sees' the unopposed forward moving depolarization of the right ventricle and records a predominantly upright QRS complex.

Inferior Infarction

There is ST elevation in lead II,III and aVF with reciprocal ST depression in V1-V3. There is also ST depression in lead I and aVL. The following two ECGs show acute and evolved inferior infarction.

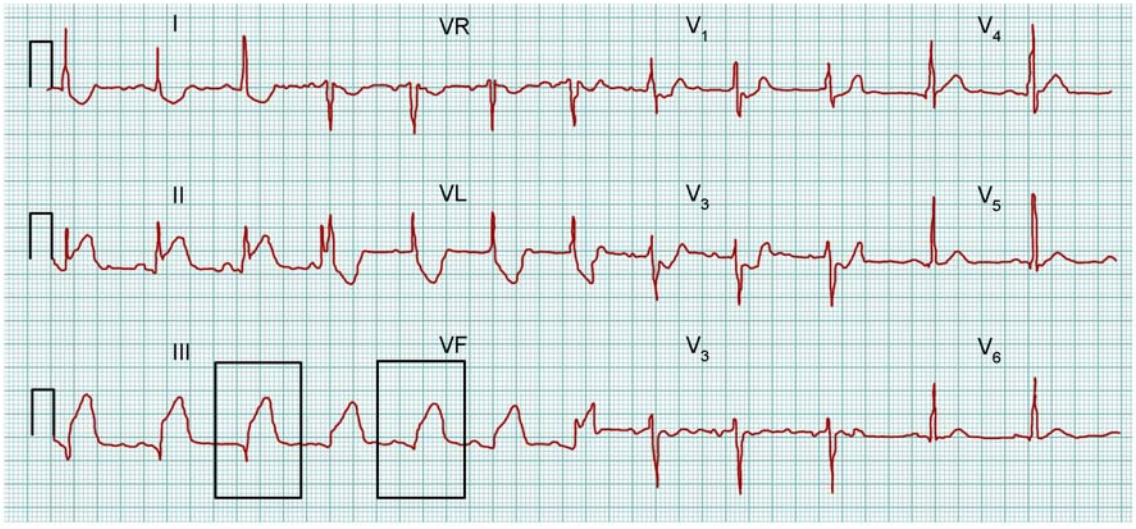

Fig. 6.6: Acute and Evolve Inferior Infraction

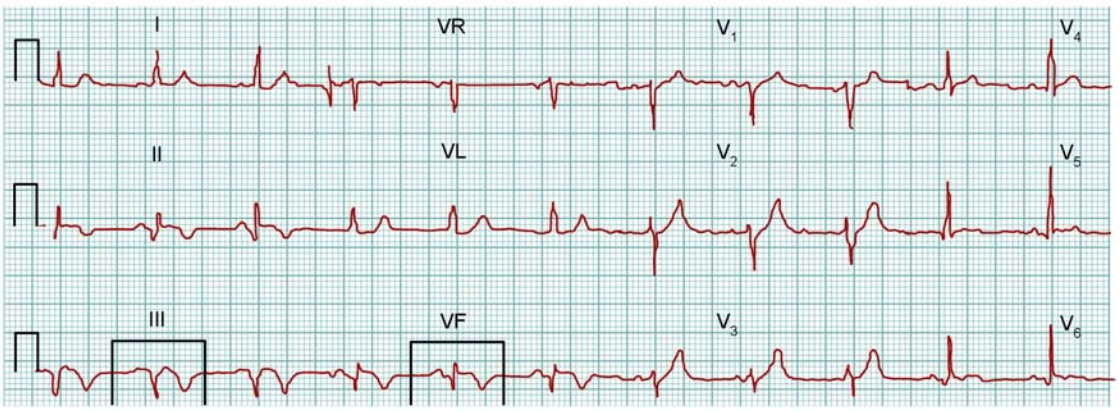

Fig. 6.7: Acute and Evolve Inferior Infraction

Right Ventricular Infarction

Inferior infarction is sometimes associated with infarction of the right ventricle. Clinically, this is suspected in a patient with an inferior infarction when the lungs are clear but the jugular venous pressure is elevated. The ECG will show a raised ST-segment in leads recorded from the right side of the heart. The positions of the leads correspond to those on the left side as follows: V1R is in the normal V2 position; V2R is in the normal V1 position; V3R etc. are on the right side, in positions corresponding to V3 etc. on the left side.

Check Your Progress 5

1) What are the ECG changes in inferior infarction?

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2) What are the ECG changes in posterior infarction?

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3) What are the ECG changes in anterior infarction?

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4) What is the sequence of ECG changes in an ST segment elevation infarction?

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6.7 DRUG AND ELECTROLYTE EFFECTS ON ECG

Hyperkalemia and ECG Results

Hyperkalemia is a commonly encountered metabolic abnormality in clinical practice. Several different types of drugs-most notably potassium-sparing diuretics and ACE inhibitors-can cause hyperkalemia. Renal failure is another common cause for elevated potassium levels. Characteristic ECG changes occur at various levels of hyperkalemia.

ECG is essential and may be instrumental in diagnosing hyperkalemia in the appropriate clinical setting. ECG changes have a sequential progression of effects, which roughly correlate with the potassium level. ECG findings may be observed as follows:

1) Early changes of hyperkalemia include peaked T-waves, shortened QT interval, and ST-segment depression.
2) These changes are followed by bundle branch block causing a uniform widening of the QRS complex. Increase in the PR interval, and decreased amplitude of the P-wave follow QRS widening.
3) These changes reverse with appropriate treatment.
4) In the absence of treatment, the P-wave eventually disappears and the QRS morphology widens to resemble a sine wave. Ventricular fibrillation or asystole follows.
5) ECG findings generally correlate with the potassium level, but potentially life-threatening arrhythmias can occur without warning at almost any level of hyperkalemia.

The QRS complexes begin to widen when the patient's serum potassium level reaches about 6-6.5 mEq/L, becoming markedly slurred and abnormally winded at 10 Me/qL. The QRS complexes may widen so that they merge with the T-waves, resulting in a "sine wave" appearance. The S-segments disappear when the serum potassium level reaches 6 mEq/L and the T-waves typically become tall and peaked at this same range. The P-waves begin to flatten out and widen when a patient's serum potassium level reaches about 6.5 mEq/L; this effect tends to disappear when levels reach 7-9 mEq/L. Sinus arrest may occur when the serum potassium level reaches about 7.5 mEq/L, and cardiac standstill or ventricular fibrillation may occur when serum levels reach 10 to 12 mEq/L. The following ECG shows tall peaked T-waves with narrow based best seen in midprecordial leads.

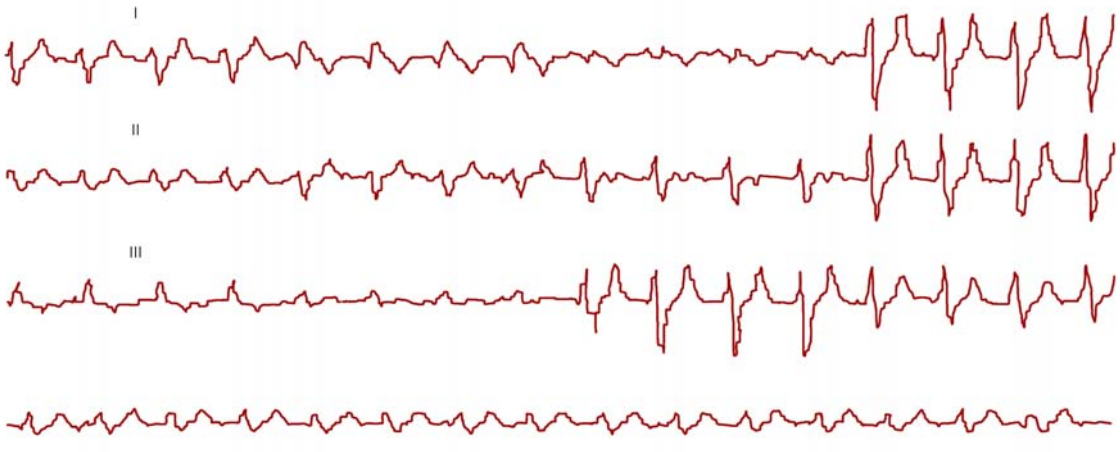


Fig. 6.8: ECG changes in hyperkalemia

Changes Caused by Hypokalemia

Hypokalemia can commonly result from the loss of potassium through dehydration, vomiting, gastric suction, or excessive diuretic use. The thiazide and loop diuretics are most commonly implicated in the development of hypokalemia.

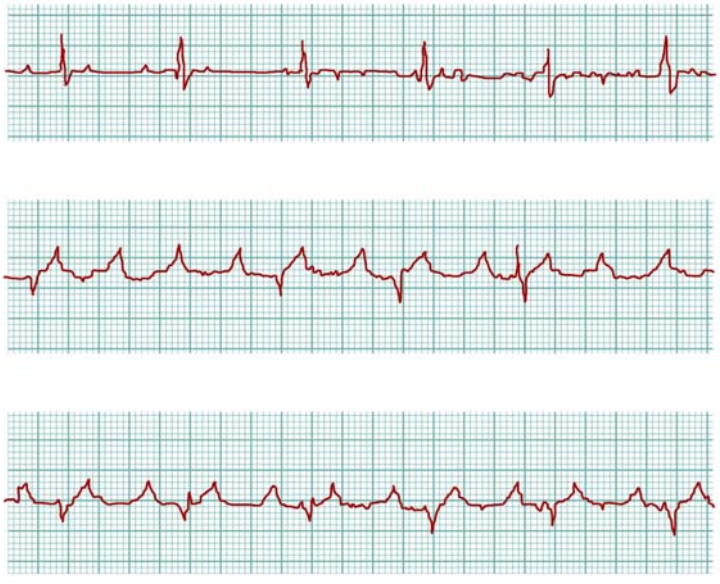
The QRS complexes begin to widen when the serum potassium drops to about 3 mEq/L, the ST-segments may become depressed, and the T-waves may begin to flatten. The U-waves also begin to increase in size, becoming as tall as the T-waves. The U-waves reach "giant" size and fuse with the T-waves when the level drops to 1 mEq/L.

Calcium

Alterations in serum calcium levels may also produce serious arrhythmias, leading to alterations in ECG results. Hypocalcemia, which may be caused by loop diuretics, osteomalacia, hypoparathyroidism, or respiratory alkalosis, may produce prolongation of the ST-segment and QT interval. Hypercalcemia, caused by adrenal insufficiency, hypoparathyroidism, kidney failure, or malignancy, may also cause serious arrhythmias, especially in the presence of digitalis.

Digitalis Effect

The administration of digitalis can cause ECG changes, even when the dosage is within the recommended therapeutic range. In cases of digitalis toxicity, excitatory or inhibitory effects on the heart and its electrical conduction system may occur. Excitatory effects include various types of ventricular and supraventricular ectopy, ventricular tachycardia, and ventricular fibrillation. Inhibitory effects include sinus bradycardia and heart block. The digitalis effect produces prolonged PR intervals, depressed (scooped) ST-segments, and alterations in T-wave morphology. The following ECG shows PAT with block,one of the classic arrhythmias seen in digitalis toxicity.



1. The P-wave axis is normal
2. The non-conducted P-wave hides in the T wave
3. The conducted P-wave often has a long PR interval
4. The P-P interval may not be exactly regular

Fig. 6.9: Atrial Tachycardia, 2:1AV block

Phenothiazines

The electrophysiological properties of phenothiazines are comparable to those of the Class I a antiarrhythmic-quinidine. Numerous ECG aberrations may be induced by these agents, including changes in the morphology of the T-wave, prolongation of the QT interval, and accentuation of the U-wave.

"These repolarization abnormalities occur more frequently with thioridizine than with chlorpromazine, and even less so with trifluoperazine," wrote Symanski and Gettes in the February 1993 issue of Drugs. "Supraventricular and ventricular tachycardias have been reported in patients receiving high doses of phenothiazines. Even with standard clinical dosages (100-400 mg/day), thioridazine causes minimal prolongation of the QT interval, reduction of T-wave amplitude, and prominent U-waves in nearly 50 per cent of patients."

Antidepressants

At either therapeutic or toxic dosages, tricyclic and tetracyclic antidepressants can produce a number of effects on the ECG. ECG Changes produced by TCAs occur in about 20 per cent of patients receiving therapeutic dosages and include an increase in heart rate, prolongation of the PR interval, intraventricular conduction disturbances, increase in QTc interval, and flattening of T-waves. Factors such as the specific agent, plasma drug concentration, duration of therapy, age of the patient, and degree of underlying cardiovascular disease all play a role in the severity and frequency of these effects. While the risk of ventricular arrhythmia has been shown to correlate poorly with serum TCA concentrations, the electrocardiogram may still be a useful tool in detecting patients with suspected TCA overdose.

Antihistamines

Stimulation of histamine receptors (H_1 and H_2), which are located in both the atrial and ventricular myocardium and on epicardial coronary arteries, may also produce changes on the ECG. Stimulation of the H_2 receptors in the atrial and ventricular myocardium raises intracellular concentrations of cAMP by activating adenylate cyclase and phosphorylase, resulting in enhanced inotropic and chronotropic effects. Thus, the blocking of H_2 receptors by the H_2 antagonists may result in bradyarrhythmias, as noted in several case reports in which cimetidine and ranitidine have been implicated as rare causes of sinus bradycardia and heart block. The risk of these complications appears to be greater with long-term therapy and among elderly individuals.

The newer, long-acting, nonsedating antihistamines such as terfenadine and astemizole can pose a threat of cardiotoxicity. Rare reactions have occurred when blood levels of these agents become elevated due to either overdose or inhibition of their metabolism when given concomitantly with other agents (erythromycin, ketoconazole, or, as has been recently reported, grapefruit juice) affecting the same CP450 isozyme.

The disturbance in cardiac conduction is thought to be caused by these agents' ability to block the potassium channel in the myocardial cell membrane, which affects cardiac repolarization. Thus, the effect on the ECG is prolongation of the QT interval, which may lead to torsade de pointes (see glossary). Studies to date have found no similar threat of cardiotoxicity with loratidine and cetirizine.

Other Drug Influences

The electrocardiographic effects of catecholamines, including agents such as dopamine and epinephrine, can be problematic to predict, since these agents have numerous effects on the heart. Catecholamines affect the currents that regulate repolarization of individual cells and fibers, and also can affect the heart rate, blood pressure, and serum potassium levels.

ECG changes caused by catecholamines are influenced by the route and rate of administration, as well as the dosage. For example, subcutaneous administration of epinephrine produces only sinus tachycardia and occasional premature beats. However, when administered intravenously, epinephrine may cause a variety of repolarization abnormalities, including ST changes. Intravenous infusion of isoprenaline (isoproterenol), to give another example, may cause inversion of T-waves.

As more and more elderly nursing home residents are treated with a variety of pharmacological agents that affect membrane function and myocardial cells, the ECG is becoming a useful tool for monitoring drug effects and toxicities-not only for the physician, but for the entire interdisciplinary care team, including the consultant pharmacist. An appreciation of the information contained in the ECG printout is an essential component in the continuing effort to provide the best in comprehensive patient care.

Check Your Progress 6

What are the ECG changes in hyperkalemia?

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6.8 LET US SUM UP

In this unit, you have learnt the changes ECG in various diseases i.e. acute pericarditis, pulmonary embolism etc. ECG changes in lead II and Lead VI helps to identify the left and right atrial hypertrophy. Now you can enlist the causes and ECG changes of atrial hypertrophy, ventricular hypertrophy and myocardial infraction. You have also learnt the effects of various drugs and electrolyte disturbances on the ECG.

6.9 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progres 1

ECG is a poor diagnostic test for pulmonary embolism.

I) Findings

a) ECG shows non-specific changes in 80 per cent

b) Classic Findings: S1 Q3 T3 (seen in under 20 per cent of cases)

i) S Wave in Lead I

ii) Q Wave in Lead III

iii) T wave inversion in Lead III

c) Common Findings

i) Sinus tachycardia

ii) Right sided strain pattern

- Right bundle branch block
- Right axis deviation

iii) Findings that mimic myocardial infarction

- ST segment changes
- T wave changes

iv) Atrial fibrillation(new onset)

Check Your Progres 2

The following features differentiate the pericarditis and acute myocardial ST elevations.

1) The ST-segment elevation that occurs during acute pericarditis is usually "concave," compared with the "convex" appearance of the ST segment that occurs during the acute injury stage of a myocardial infarction.

2) In acute pericarditis there is widespread ST-segment elevation not corresponding with any specific arterial territory, which usually occurs in association with acute myocardial infarction.

3) Reciprocal changes are absent in acute pericarditis, while they are frequent with acute myocardial infarction.

4) The ST segments in acute pericarditis return to baseline in a few days and are followed by diffuse T-wave inversion, in conjunction with the ST segment at baseline. In contrast, T inversion in infarction occurs before ST segment returns to baseline.

5) Another feature that may aid in differentiating acute pericarditis from acute myocardial infarction is the absence of Q waves.

6) Loss of R-wave progression may occur with acute myocardial infarction, but this feature is not present with acute pericarditis.

There may also be ST-segment depression in leads aVR and V1. In the absence of underlying cardiac disease, the P wave and QRS complexes are normal. Depression of the PR segment is very specific of acute pericarditis and is attributed to subepicardial atrial injury and occurs in all leads except aVR and V1. These leads may exhibit PR-segment elevation. Symptoms subside within two weeks and ECG changes resolve over a period of weeks to months. Development of atrial & ventricular arrhythmias, heart blocks and fascicular blocks does not occur in acute pericarditis. Presence of these changes in the setting of acute pericarditis indicates the presence widespread underlying myocardial involvement.

Check Your Progres 3

1)

- The terminal portion of the P wave in lead V1 must be one small box wide by one small box (0.04second x 0.04mv) deep or larger to qualify as left atrial enlargement.
- This force can be calculated by multiplying the time in seconds by the depth in millimeters. If this product is more negative than -0.04, LAE is present.
- A notched broad based P wave in leads I and II with a duration of 0.12 milliseconds or more is called "P mitrale".
- LAE can shift the P wave axis to $+15^{o}$ or less.
- Comparision of ECG abnormalities to echocardiographic criteria for left atrial enlargement demonstrates limited sensitivity but high specificity for the ECG criteria.

2)

- The P wave in leads II, II and aVF is peaked with a height greater than 2.5mm. "P pulmonale".
- The P-wave axis is +75 or greater.

`The positive aspect of the P-wave in lead V1 or V2 is >1.5mm in height.

Check Your Progres 4

1) ECG features include:

   i) ≥ QRS amplitude (voltage criteria; i.e., tall R-waves in LV leads, deep S-waves in RV leads)

   ii) Other Voltage Criteria for LVH

      a) Limb-lead voltage criteria:

      -R in aVL ≥11 mm or, if left axis deviation, R in aVL ≥13 mm plus S in III ≥15 mm

      -R in I + S in III >25 mm

      b) Chest-lead voltage criteria:

      -S in V1 + R in V5 or V6 ≥ 35 mm

   iii) CORNELL Voltage Criteria for LVH (sensitivity = 22 per cent, specificity = 95 per cent)

   -S in V3 + R in aVL > 24 mm (men)

   -S in V3 + R in aVL > 20 mm (women)

2) Inferior infarction is sometimes associated with infarction of the right ventricle. The ECG will show a raised ST segment in leads recorded from the right side of the heart. The positions of the leads correspond to those on the left side as follows: V1R is in the normal V2 position; V2R is in the normal V1 position; V3R etc. are on the right side, in positions corresponding to V3 etc. on the left side.

Check Your Progres 5

1) In inferior infarction there is ST elevation in leadII, III and aVF often with reciprocal ST depression in V1-V3. There may also be ST depression in lead I and aVL.

2) An infarction of the posterior wall of the left ventricle can be detected in the ordinary 12-lead ECG because it causes a dominant R wave in lead V1. Normally there is a small R wave and a deep S wave in V1.. In a posterior infarction, the rearward-moving electrical forces are lost so lead V1 'sees' the unopposed forward moving depolarization of the right ventricle and records a predominantly upright QRS complex.

3) The changes of anterior infarction are seen in leads V2–V5. Lead V1, which lies over the right ventricle, is seldom affected. The lateral wall of the left ventricle is often damaged at the same time as the anterior wall, and then leads I, VL and V6 show infarction changes. Persistent ST segment elevation is quite common after an anterior infarction: it sometimes indicates the development of a left ventricular aneurysm, but it is not reliable evidence of this.

4) The sequence of features characteristic of 'full thickness', or 'ST segment elevation', myocardial infarction is:

- Normal ECG
- Delayed intrinsicoid deflection
- Tall peaked T waves (so called hyperacute T waves)
- ST segment elevation
- Development of Q waves
- ST segment returns to the baseline
- T waves inversion
- Loss of R wave voltage

Check Your Progres 5

ECG changes have a sequential progression of effects, which roughly correlate with the potassium level. ECG findings may be observed as follows:

1) Early changes of hyperkalemia include peaked T waves, shortened QT interval, and ST segment depression.

2) These changes are followed by bundle branch block causing a uniform widening of the QRS complex. Increase in the PR interval, and decreased amplitude of the P wave follow QRS widening.

3) These changes reverse with appropriate treatment.

4) In the absence of treatment, the P-wave eventually disappears and the QRS morphology widens to resemble a sine wave. Ventricular fibrillation or asystole follows.

5) ECG findings generally correlate with the potassium level, but potentially life-threatening arrhythmias can occur without warning at almost any level of hyperkalemia.

The QRS complexes begin to widen when the patient's serum potassium level reaches about 6-6.5 mEq/L, becoming markedly slurred and abnormally winded at 10 Me/qL. The QRS complexes may widen so that they merge with the T-waves, resulting in a "sine wave" appearance. The S segments disappear when the serum potassium level reaches 6 mEq/L and the T waves typically become tall and peaked at this same range. The P waves begin to flatten out and widen when a patient's serum potassium level reaches about 6.5 mEq/L; this effect tends to disappear when levels reach 7-9 mEq/L. Sinus arrest may occur when the serum potassium level reaches about 7.5 mEq/L, and cardiac standstill or ventricular fibrillation may occur when serum levels reach 10 to 12 mEq/L.