# ECG in Non Cardiac Disorders

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## INTRODUCTION

The electrocardiogram (ECG) is still the basic cardiologic test and is widely applied in patients with suspected or known heart disease as a basic reference. However, ECG abnormalities can be present in a wide variety of noncardiac conditions like electrolyte abnormalities, pulmonary embolism, CNS diseases, hypothermia, drug-related and other conditions. These conditions may provide the characteristic ECG changes which can simulate primary cardiac conditions. Knowledge of these changes may provide an early clue to the diagnosis of these disorders, which can be life-saving.

This review article discusses the ECG changes pertaining to these conditions in detail.

## CENTRAL NERVOUS SYSTEM DISORDERS (CNS)

ECG changes are often seen in disorders of CNS like CNS heamorrhage, neuromuscular disorders and cerebellar ataxias.

### CNS Hemorrhage

ECG changes may be seen in extradural, subdural, parenchymal and subarachnoid heamorrhage (SAH). The most common changes include broad or inverted T waves, prolonged QT interval and a prominent U wave (Fig.1). Rarely ST elevation may also be seen.[1,2] The common rhythm changes are sinus tachycardia and sinus bradycardia

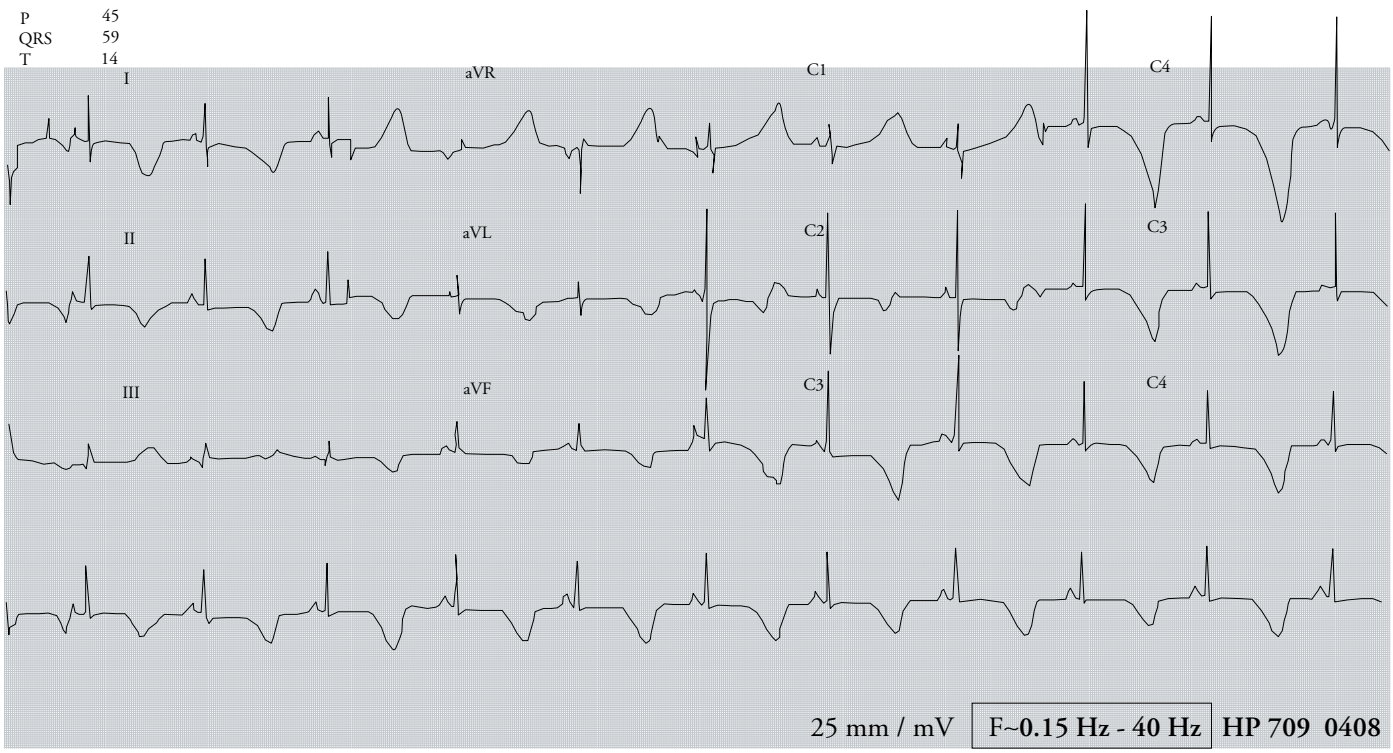


**Fig. 1**: ECG taken in a patient with raised intracranial tension due to subarachnoid haemorrhage; showing QT interval prolongation, symmetrical deep T wave inversion in multiple leads

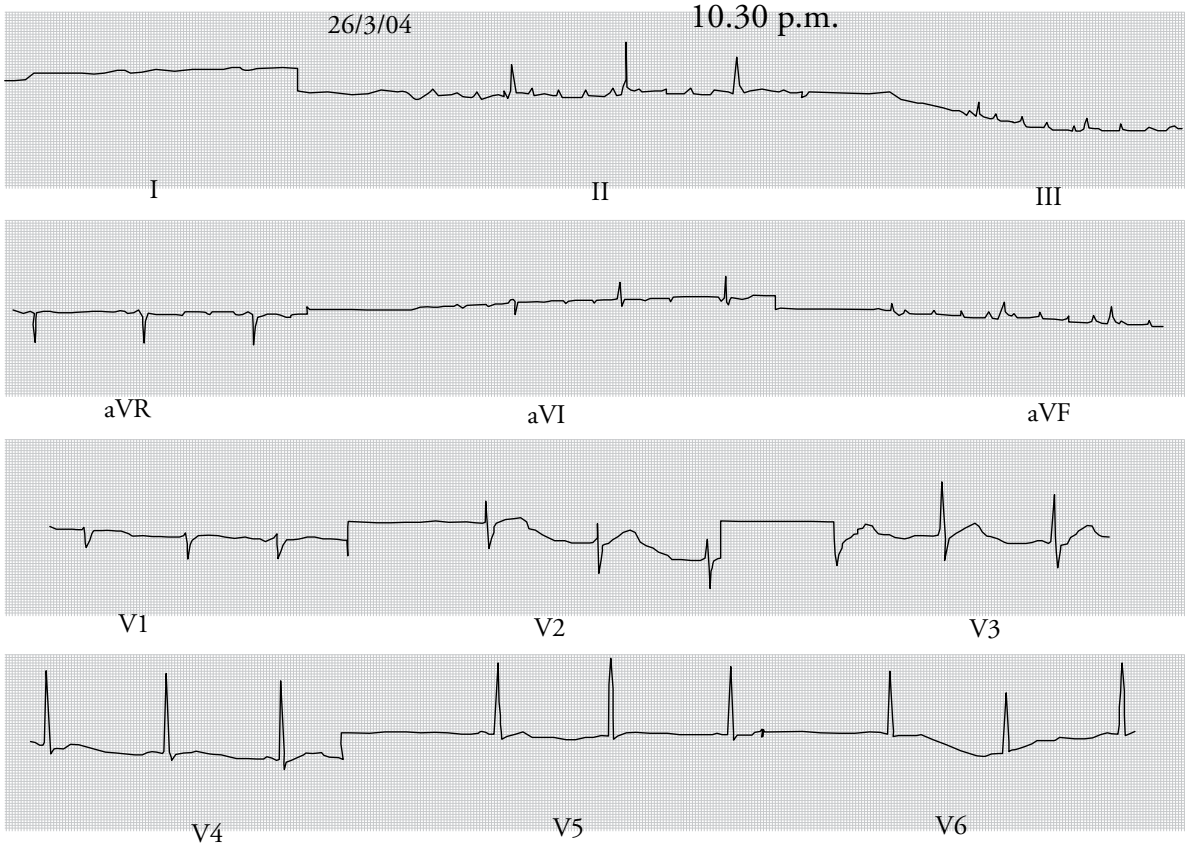


**Fig. 2a** : ECG showing pseudo atrial flutter because of tremors

The mechanism of the ECG changes in these conditions is not clear. The most accepted explanation is altered autonomic tone, as the autonomic nervous system has been shown to affect ventricular repolarisation and the morphology of ST-T complex. Catecholamines have been implicated as one of the mediators of ECG morphology and rhythm changes. Biochemical evidence of myocardial damage with elevated serum CPK levels has been reported in some SAH patients, which may due to catecholamine-induced vasoconstriction.[3]

Kiyoshi Yuki proposed that increased catecholamines cause coronary vasospasm, resulting in prolonged QT intervals, ST segment elevation and arrhythmias, including ventricular tachyarrhythmias.[4]

### Neuromuscular disorders

ECG changes are often seen in disorders of CNS like muscular dystrophies, myotonic dystropy, mitochondrial myopathies and myasthenia gravis.

### Cerebellar ataxia: Friederich's ataxia

Cardiac lesions are seen in 50% to 75% of cases. The cardiac conditions seen most often are hypertrophic cardiomyopathy, muscular subaortic stenosis and hypokinetic dilated left ventricle. Abnormal ECG is seen in 75 to 92 percent of cases.

The most frequent ECG abnormalities consist of T wave changes and right or left axis deviation in the frontal plane. There can be abnormal Q waves in the inferior and lateral leads, usually with tall R waves in the right precordial leads, simulating myocardial infarction (MI) of the inferior and poster. lateral wall.[5, 6, 7]

The Arrhythmias are less common. Atrial arrthymias including atrial flutter and atrial fibrillation are seen with hypertrophic cardiomyopathy.Ventricular arrthymias are seen with dilated cardiomyopathy.

### Myasthenia gravis

In patients of myasthenia gravis, the ECG may show nonspecific T wave changes, prolonged QT interval, and right bundle branch block (RBBB). Paraplegic and quadriplegic individuals have higher incidence of RAD due to decreased left ventricular mass.[8]

### Muscular Dystrophies

ECG changes are seen in 75 to 95% of cases.[9] The most frequent abnormalities are T wave changes, tall R waves in right precordial leads waves, deep Q waves in inferior and lateral leads, right or left axis deviation. These changes, probably due to myocardial fibrosis of the posterolateral wall of the left ventricle, resemble pseudo-infarction pattern of HCM.[10]

### Tremors

Muscle tremors and movement may impair the quality of recordings. Failure to minimize the artifact while recording, and failure to recognize artifacts during interpretation may lead to incorrect diagnosis of arrhythmias and unnecessary interventions (Fig. 2A and 2B)

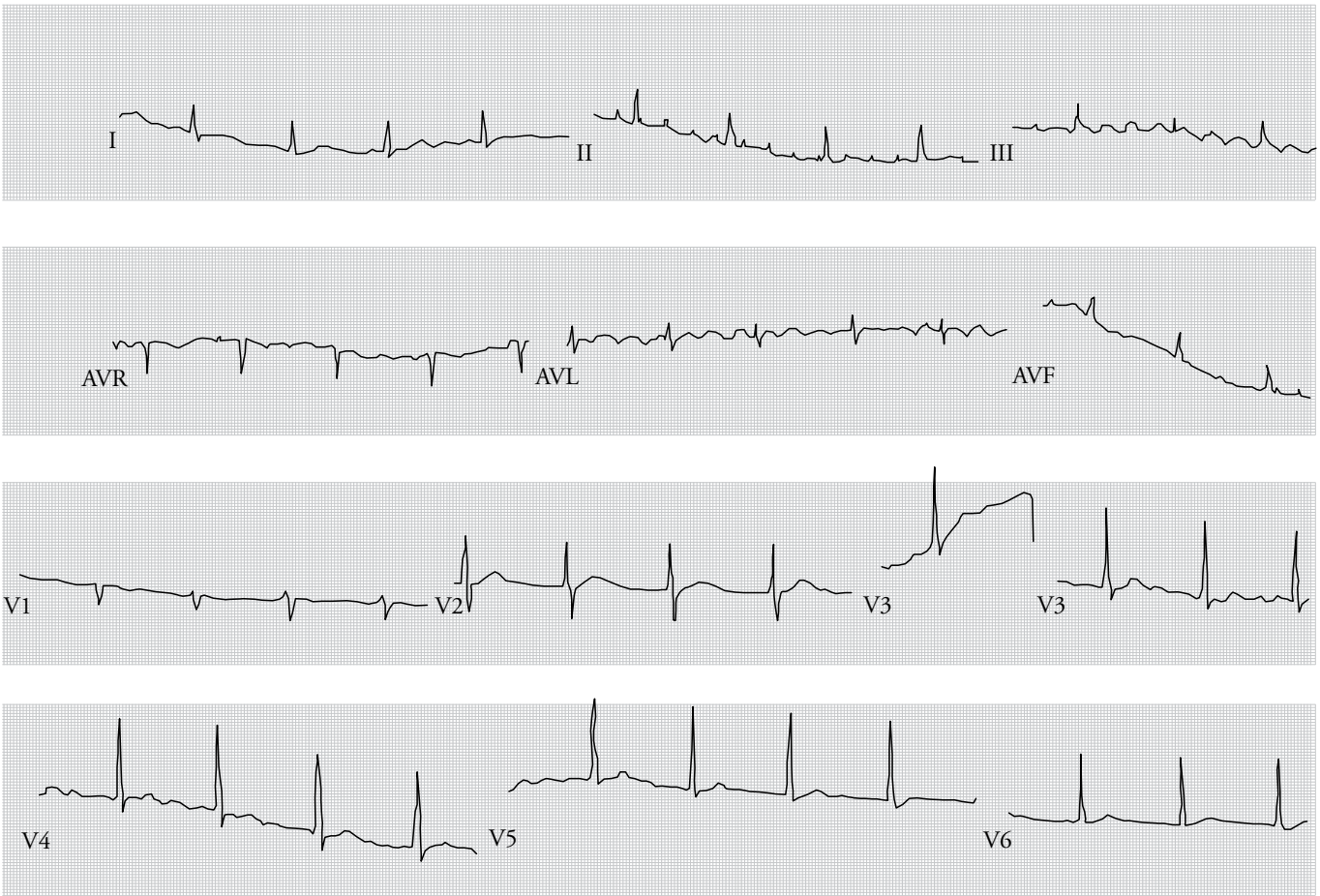


**Fig. 2b** : ECG showing artifacts due to benign tremors

## HYPOTHERMIA

Hypothermia is diagnosed when the core body (rectal) temperature is less than 35°C. The earliest change seen in the electrocardiogram is an artifact due to shivering, although some hypothermic patients have relatively normal ECG. As body temperature falls further, all metabolic and cardiovascular processes slow progressively. This results in bradycardia, heart block, and prolongation of the PR, QRS, and QT intervals. When body temperature falls below 32°C, regular and organized atrial activity is replaced by varying degrees of slow, irregular, and disorganized activity, resulting in atrial fibrillation with slow ventricular rate. If core temperature falls below 28°C, a junctional bradycardia may be seen.

The J wave (Osborn wave) is the most specific ECG finding in hypothermia. The J wave is most commonly characterized by a "dome" or "hump" elevation in the terminal portion of the QRS deflection and is best seen in the left chest leads. The size of the J wave often correlates with the severity of hypothermia (< 30°C) but the exact etiology is not known. The J wave may even occur because of a drug effect or rarely may it be a normal variant.

Ventricular arrhythmias are the most common mechanism of death in hypothermia. They seem to be more common during rewarming as the body temperature rises through the 28°32°C range.[11]

## HYPERKALEMIA

It is a potentially life-threatening illness that can be difficult to diagnose because of a paucity of distinctive signs and symptoms. It can lead to sudden death from cardiac arrhythmias. So any suspicion of hyperkalemia requires an immediate ECG along with the biochemical investigation to rule out the diagnosis.

Hyperkalemia is graded as follows

5.5 - 6.5 mEq/L - Mild Hyperkalemia

6.5. - 7.5 mEq/L - Moderate Hyperkalemia

>7.5 mEq/L and greater - Severe Hyperkalemia

Progression of ECG changes roughly correlates with the potassium level, but potentially life-threatening arrhythmias can occur without warning at almost any level of hyperkalemia.[11]

[K+] = 5.5 – 6.5 mEq/L

- Tall, peaked, narrow based T waves
- Shortened QT interval
- Reversible left anterior and posterior fascicular block

[K+] = 6.5 – 7.5 mEq/L

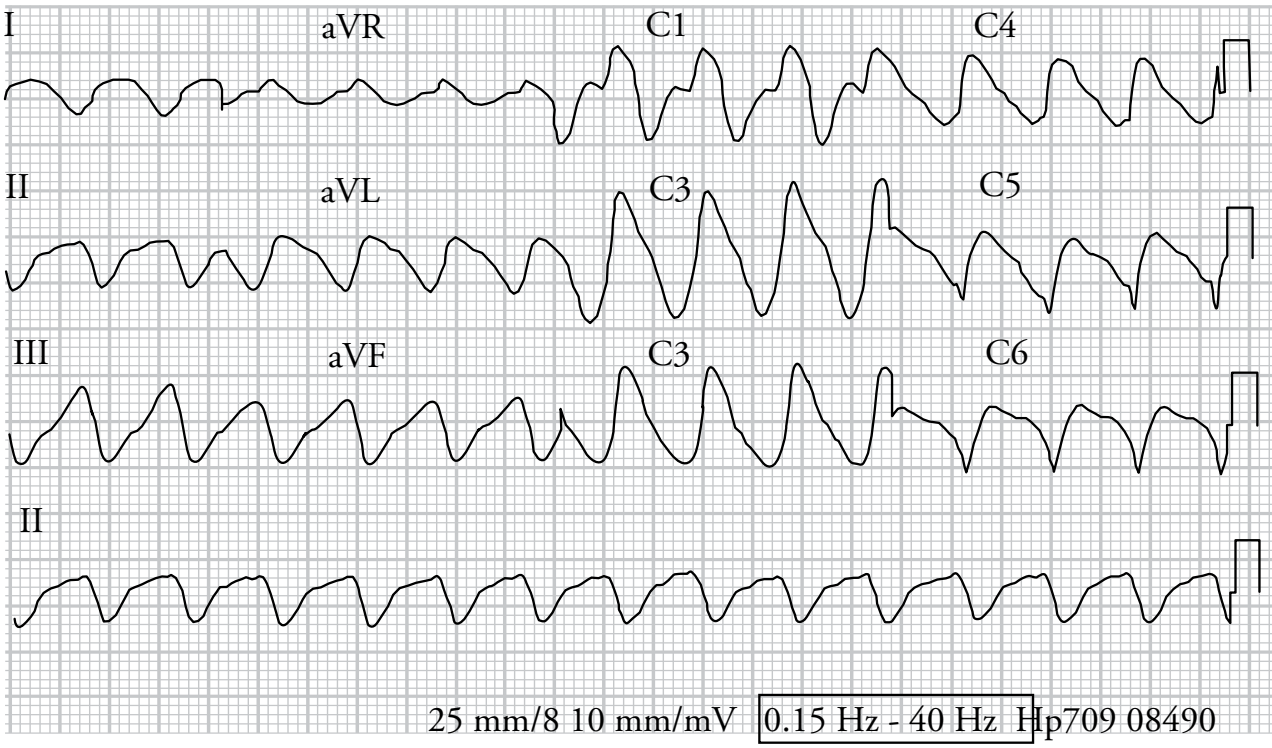


**Fig. 3** : ECG of severe hyperkalemia; P wave not seen, QRS morphology widens to resemble a sine wave

First degree AV block

- Flattening and widening of P wave
- ST segment depression

[K+] > 7.5 mEq/L (Fig. 3)

- Disappearance of P waves
- Bundle branch blocks causing a widening of the QRS complex
- Arrhythmia and conduction disturbances including ventricular tachycardia, ventricular fibrillation, idioventricular rhythm or asystole

## HYPOKALEMIA

Electrocardiographic changes are not common with mild and moderate hypokalemia and reliably seen when serum potassium is below 3 mEq/L. The ECG changes seen most often are prominent U waves, ST segment depression, flattened T waves, prolonged QT interval, and various rhythm changes. The important arrthymias are ventricular ectopy leading to ventricular tachycardia and ventricular fibrillation.[11]

Hypokalemia is an important cause of acquired long QT syndrome that predisposes to torsades de pointes. It can also predispose to tachyarrhythmias from digitalis.

## HYPERCALCEMIA

The duration of the phase 2 of the action potential, which determines the duration of the ST segment, increases at low extracellular calcium concentration and shortens at high calcium concentrations. So the changes in calcium concentrations predominately affect the duration of the ST segment and thereby the duration of the QT interval.

There is shortening of the QT and QTc interval, which may be because of shortening of the ST segment.[12] Cardiac arrhythmias are uncommon.

## HYPOCALCEMIA

Prolonged QTc is observed which may be because of extended ST segment. Occasionally flattening, peaking or inversion of T waves is seen.[11]

## HYPOGLYCEMIA

Hypoglycemia is a common medical emergency, although ECG changes are not very common. The electrocardiographic features include flattening of the T wave and QT prolongation.

## pH

Acidosis and alkalosis do not cause specific ECG changes

## HYPONATREMIA

The effect of plasma sodium level on the ECG is negligible, if present it is in the range compatible with life.

## PULMONARY EMBOLISM (FIG. 4)

Pulmonary embolism (PE) is a potentially lethal condition. Unfortunately the diagnosis is often missed as it produces vague symptoms and signs. The ECG is often abnormal in PE, but findings are neither specific nor sensitive.

The most frequent EGG manifestation of PE is sinus tachycardia. Other common rhythm disturbances are premature atrial,ventricular beats and atrial fibrillation.

The classic findings of right heart strain and acute cor pulmonale are seen in only 20% of patients with proven PE. These findings are tall, peaked P waves in lead II (P pulmonale), right axis deviation, RBBB, an S1-Q3-T3 pattern.

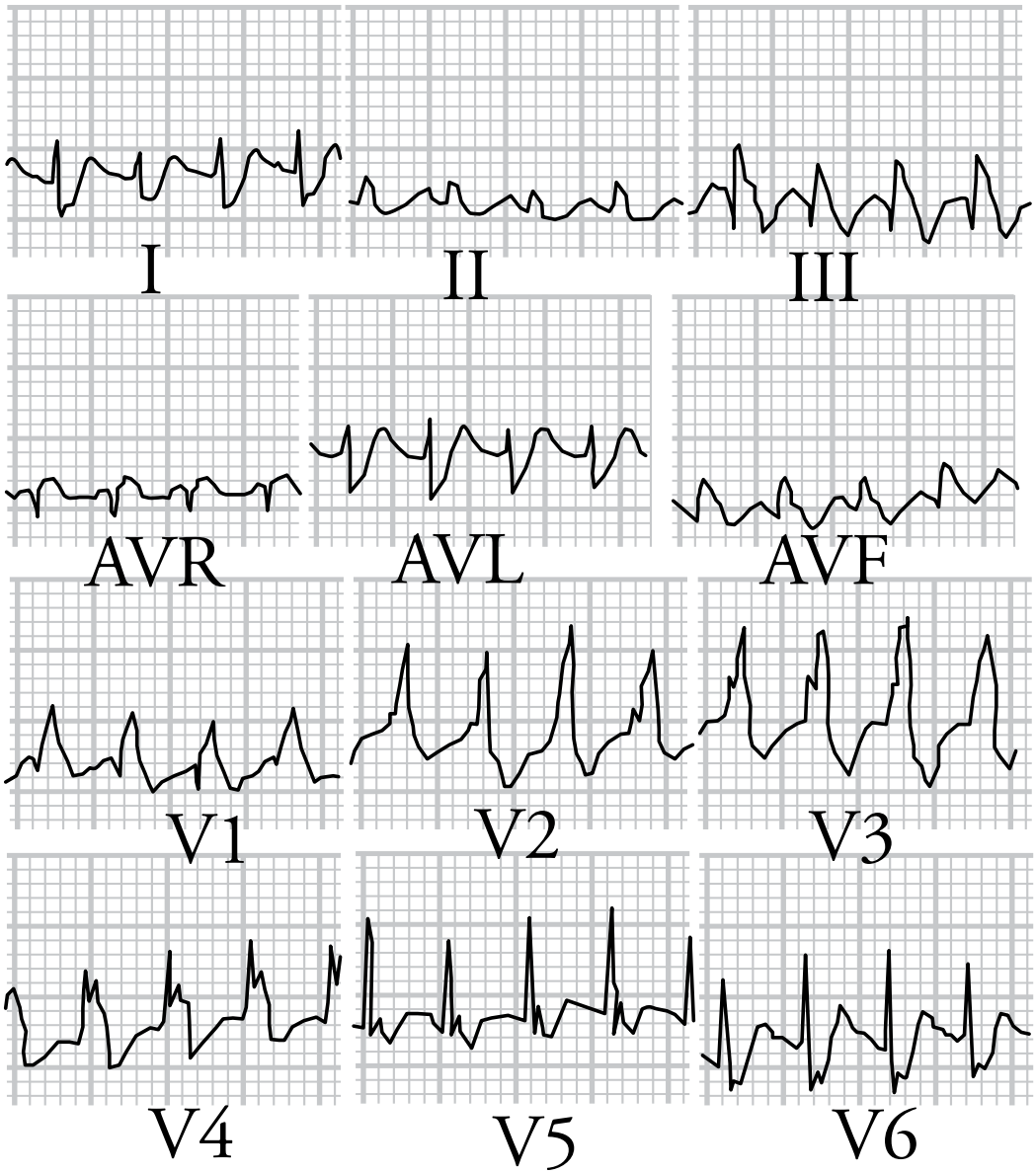


**Fig. 4** : ECG of pulmonary embolism; S1Q3T3 pattern, RBBB and T inversion in V1-V4

S1Q3T3 (S in lead I, and Q wave and T inversion in lead III) was first described by McGinn and White in 1935. S wave in lead I signify a complete or more often incomplete RBBB; Q wave, slight ST elevation and an inverted T wave in lead III are due to the pressure and volume overload over the right ventricle. Any etiology of acute cor pulmonale can cause the S1Q3T3 finding on the ECG.[13]

Ferrari E et al observed that anterior T wave inversions had a sensitivity of 85%, specificity of 81% for massive PE in 80 patients with suspected to have PE; this was the most common finding on ECG (68%), followed by S1Q3T3 (50%).[14]

Rodger M et al studied the diagnostic value of the electrocardiogram in suspected pulmonary embolism. The ECG findings were compared to the controls and only tachycardia and incomplete RBBB could differentiate PE from other diseases.[15]

Stein PD et al studied clinical characteristics in 117 patients with acute PE. Non-specific ST-T wave changes were the most common finding on the ECG (49%). [16]

To sum up, ECG is an adequate diagnostic test for PE. The greatest utility of the ECG in the patient with suspected PE is ruling out other potential life-threatening diagnoses such as myocardial infarction.

## HYPERTHYROIDISM

The cardiovascular system is very sensitive to thyroid hormone. The most common ECG changes seen are various supraventricular arrhythmias.[17] Elderly patients may develop ischemic ST and T wave changes because of their tachycardias. Atrial fibrillation is the most common sustained arrhythmia in thyrotoxicosis, occurring in about 20% of all cases. It is most common in elderly patients, and those with a particularly high concentration of thyroid hormone.[18] Ventricular arrhythmias may be seen, though much less frequently.

## HYPOTHYROIDISM

The most common electrocardiographic changes seen in hypothyroidism are sinus bradycardia, low QRS voltages, a prolonged QT interval and non-specific T waves. Prolonged QT interval is because of prolongation of the depolarization phase of the ventricles. Patients with pre-existing heart disease may also develop increasing degrees of heart block or bundle branch block, especially right bundle branch block. Torsades de pointes and ventricular tachycardia have also been reported. Uncommonly, patients may develop large pericardial effusions, which give rise to electrical alternans.[19]

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