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ECG - "Extracardiac Guidance"

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Abstract:

The electrocardiogram (ECG) continues to remain a crucial investigation in cardiology even after a century of its discovery despite so much sophisticated investigations today. It not only gives the diagnosis but also gives other crucial information such as prognosis, management plan and result of the treatment in many cardiac diseases. But it should be realised that the ECG also gives almost so much information in many non-cardiac situations. This article explores the role of ECG in so many non-cardiac conditions such as Lung diseases, Gastrointestinal diseases, Neurological illnesses, Electrolyte abnormalities and poisoning. Just a change in posture and skeletal abnormalities can also alter ECG to mimic a cardiac disease. In each of these conditions ECG changes are explained with representative ECGs and its importance in managing that particular non-cardiac condition is also discussed.

Introduction:

Since its discovery in 1902 by William Einthoven, the electrocardiogram (ECG) has served as the most cost-effective investigation. Its usefulness in cardiac conditions, both in coronary and non-coronary heart disease is well established. However, most often it is believed that the ECG is a cardiac investigation, utilised only for diagnosing cardiac conditions. The beauty of ECG is that it can provide valuable information in a variety of non-cardiac conditions. In this article, we explore the usefulness of ECG in many non-cardiac situations.

I. Lung Conditions:

A. Chronic Obstructive Pulmonary Disease (COPD):

The ECG is very useful in COPD to assess the prognosis. A peculiar ECG sign in COPD is the 'Lead I sign' or 'Schamroth sign' which is low voltage P, QRS, T in L I because of the vertical axis of all the vectors[1] (Fig.1). The ECG may also show Right Ventricular hypertrophy and Right Axis Deviation which are the signs of Cor Pulmonale where the prognosis is bad. In addition to this, the symmetrical T inversion in chest leads may be due to Right Ventricular Ischemia rather than Coronary Artery Disease which also indicates a bad Prognosis.

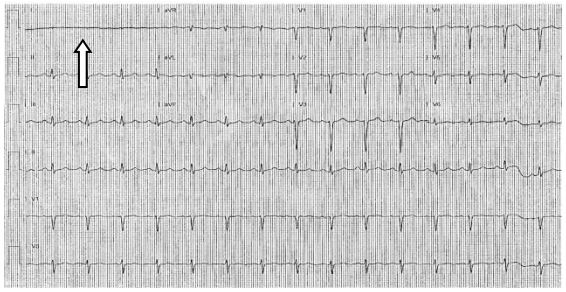


Fig 1. *ECG showing Schamroth sign. (arrow)*

B. Pulmonary Thromboembolism

There are many ECG signs described in pulmonary thromboembolism. The most important and common ECG sign is a symmetrical inversion of T wave in anterior chest leads[2]. (Fig.2) This is due to Right Ventricular Ischemia and dilatation where the Right ventricle occupies a region of V1-V3. This ECG sign in an appropriate clinical setting not only establishes the diagnosis but also indicates poor response to treatment as well as prognosis

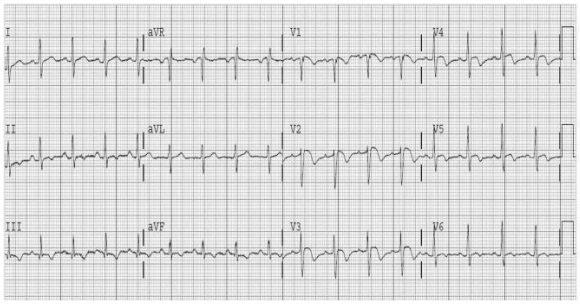


Fig.2 *ECG in acute pulmonary embolism showing symmetrical T inversion in V1 –V4*

C. Pneumothorax

Diagnosis of pneumothorax is purely clinical. ECG changes are due to shifting of the heart which gets normalised immediately after the relief of pneumothorax. (fig 3,4)

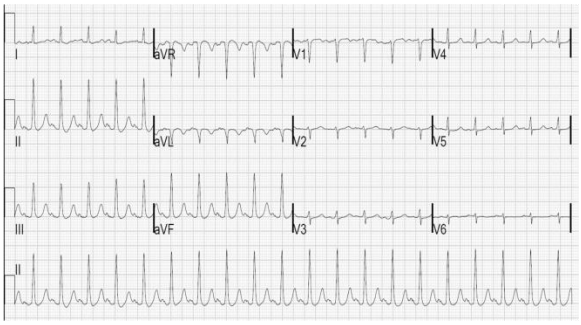


Fig.3*. ECG showing sinus tachycardia, low voltage and non-progression of R wave in the chest leads to Pneumothorax on the left side shifting the heart to the right side.*

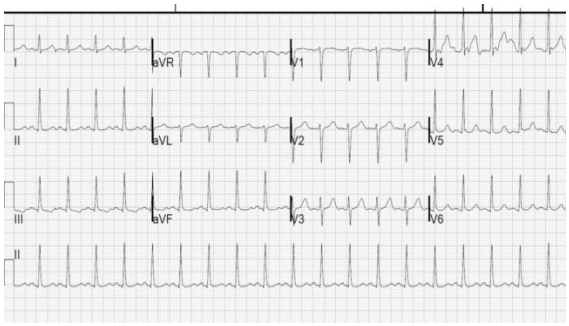


Fig.4*. ECG after the relief of Pneumothorax. Please note the progression and good voltage of the R wave in left-sided leads.*

II. Skeletal Abnormalities

ECG may be abnormal due to skeletal abnormalities such as kyphoscoliosis. The common ECG sign is non-progression of the R wave in the chest leads due to shifting of the heart (Fig.5a) Non-progression of the R wave is defined as R wave less than 3mm in V3 when chest electrodes are correctly placed. In this situation, taking ECG one space below or above may increase the R wave voltage in V3 in which case anterior MI as the cause of non-progression of R wave is unlikely. The skeletal abnormality of the pectus excavatum responsible for the non-progression of R waves is shown in Fig.5b.

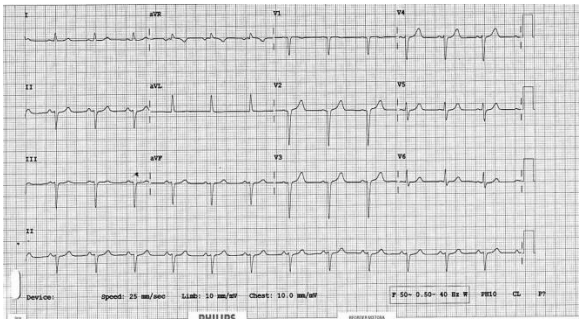


Fig 5a. *ECG mimicking anterior MI in a patient with skeletal abnormalities*

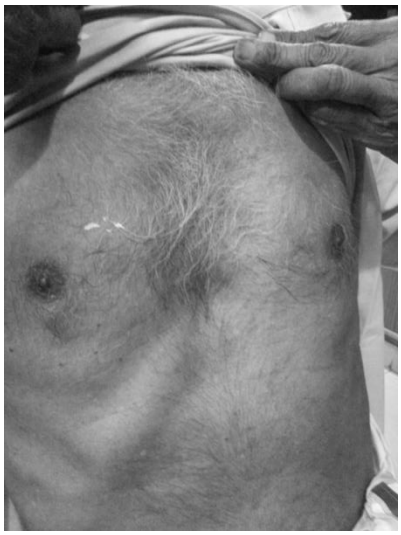


Fig 5b*. The patient showing skeletal abnormality whose ECG is shown in Fig 5a*

III. Central Nervous System Disorders:

ECG can be abnormal in certain CNS disorders. Subarachnoid haemorrhage (SAH) and some cases of stroke usually produce deep, broad T inversion (Fig.6). CAD also produces deep T inversion in chest leads. But in SAH, T inversion is deep, and broad with prolonged QT interval[3] (Fig.6a). Rarely in SAH, ECG may show ST elevation mimicking acute ST elevation MI (Fig.6b). This is due to excessive catecholamines released from the brain producing extensive myocardial injury. Thrombolysis here is disastrous. In some patients with vertebrobasilar insufficiency, atrial fibrillation can occur.

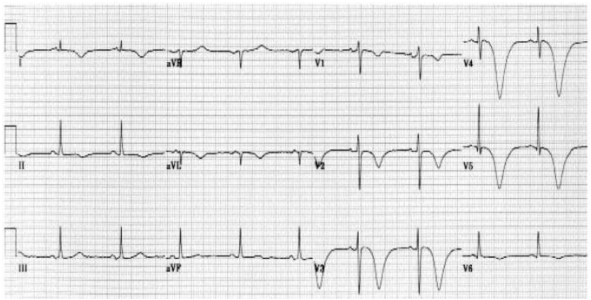


Fig 6a: *Deep broad T inversion with prolonged QT interval in SAH*

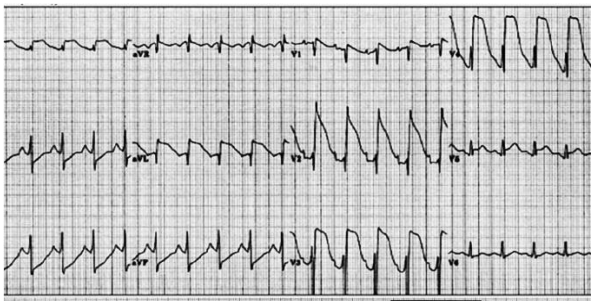


Fig 6b. *ST elevation in a patient with SAH*

III. Gastro Intestinal Disorders (GID):

Some GID may also produce ECG changes. Acute pancreatitis can sometimes produce ECG changes mimicking acute coronary syndrome (Fig.7) The ECG changes in pancreatitis are due to proteolytic enzymes released by the pancreas injuring the myocardium. The clinical correlation with ECG interpretation in this situation is crucial as the treatment given for Acute Coronary Syndrome will worsen pancreatitis. In some patients, oesophageal disorders not only mimic CAD but can also produce ECG changes[4] due to associated coronary spasm known as 'Linked Angina' (fig 8)

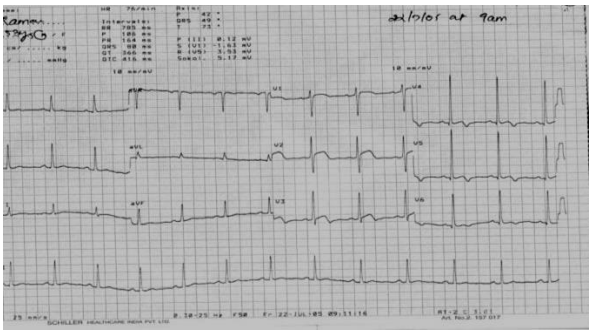


Fig 7. *ECG in a patient with acute pancreatitis showing ST coving mimicking acute coronary syndrome*

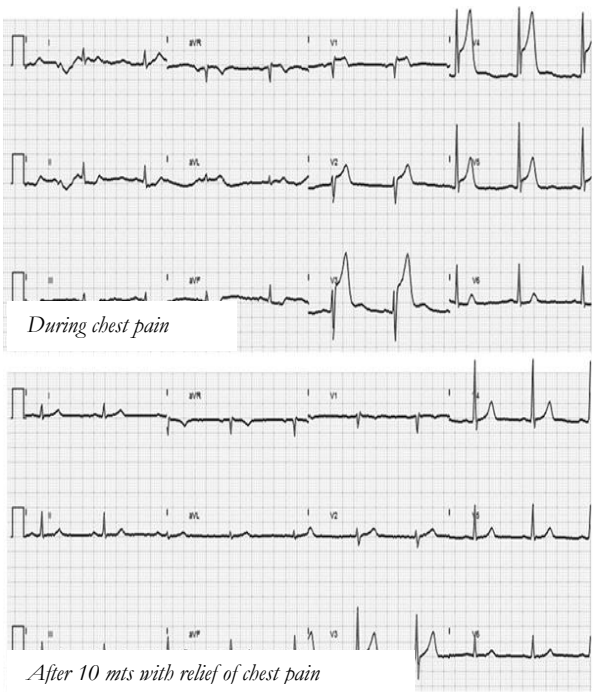


Fig.8. *A patient with oesophagal spasm showing transient ST elevation due to transient coronary spasm*

IV. Electrolyte Disturbances:

Electrolyte disturbance can cause significant ECG changes. The relationship between active potential and ECG is shown in Fig.9. The QRS corresponds to sodium entry, calcium to ST segment and potassium to T wave.

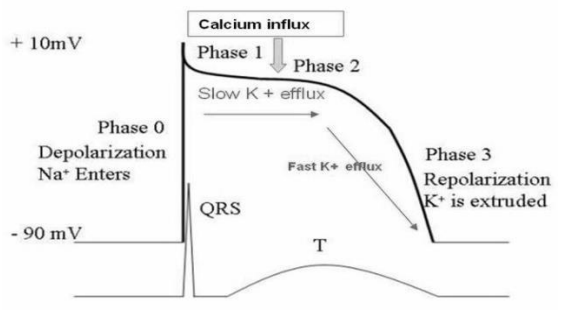


Fig.9. *Relationship between action potential, movement of ions and ECG.*

D. Potassium:

Hyperkalemia initially produces Tall T waves (fig 9), with increasing levels producing P and QRS changes[5]. The ECG changes appear beyond 6meq/L. When hyperkalemia produces tall T waves, it may be mistaken for acute subendocardial ischemia (Fig 10). Hyperkalemia produces Tall T with narrow base and sharp apex; acute ischemia produces Tall T waves with wide base and blunt apex. (Fig.11)

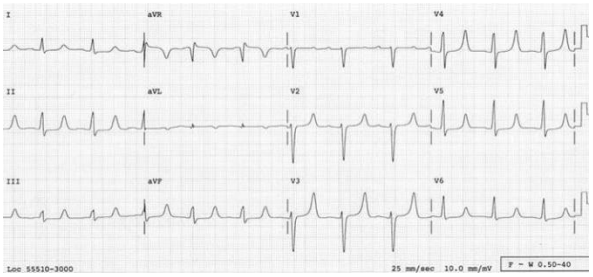


Fig 10. *ECG showing hyperkalemia. Tall T with a narrow base and sharp apex*

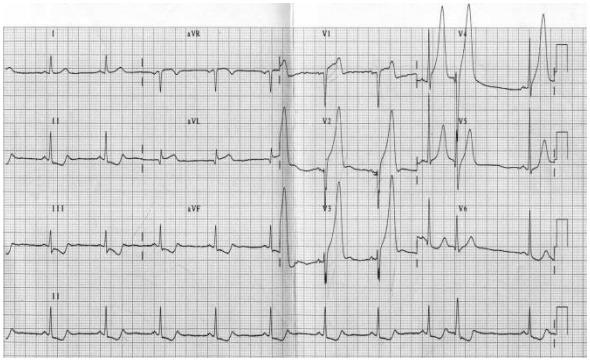


Fig.11. *ECG showing tall T waves due to sub endocardial ischemia. (broad base with blunt apex)*

Hypokalemia: Hypokalemia produces low voltage T waves with prominent U waves. Usually, the ECG changes occur when potassium is <2.7meg/l. Whenever there is a low voltage T wave, one should look for 'u' wave to rule out hypokalemia. When K is less than 1.7 meq./L, it produces significant ST depression, low voltage T and prominent U mimicking acute coronary syndrome[5](Fig.12).The apparent QT prolongation in hypokalemia differentiates it from acute myocardial injury.

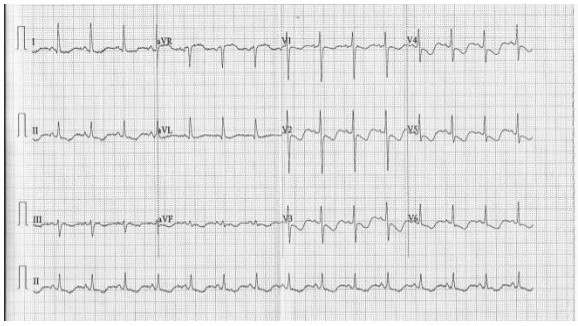


Fig 12. *ECG in severe hypokalemia showing down sloping ST depression, low voltage T wave and prominent U extending into next P wave.*

2. Calcium:

The abnormalities in calcium produce ST changes. Hypercalcemia produces short QT interval due to a short ST segment and hypocalcemia produces prolonged QT interval due to a prolonged ST segment[5] (Fig.13,14).

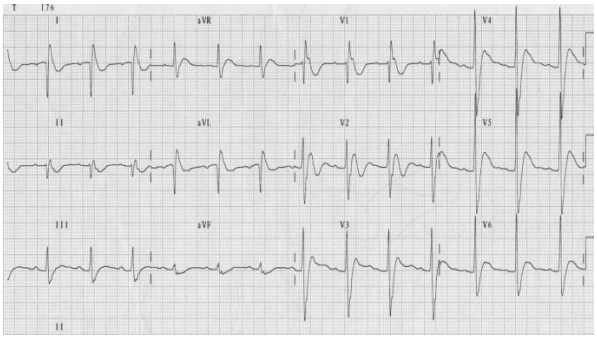


Fig.13. *ECG showing short QT due to shortened ST segment interval due to hypercalcemia.*

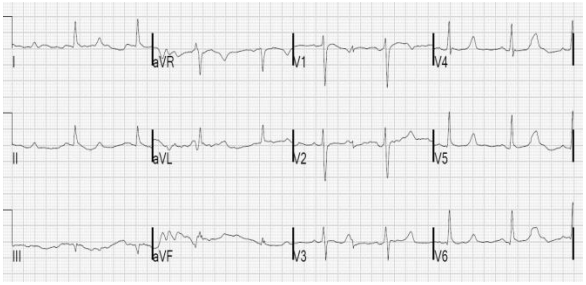


Fig.14. *ECG showing Prolonged ST segment due to hypocalcemia.*

Digoxin produces short QT interval due to shortening of ST segment because of intracellular hypercalcemia

V. Hypothermia

Hypothermia is defined as core body temperature below 95 degrees Fahrenheit. ECG changes appear below 90 deg. F and when the temperature approximates 86 deg. F, 80% of patients show an extra deflection at the end of QRS which is known as Osborn wave[6] (fig 15-arrow). This change which was described by Dr.John Osborn is due to the gradient of potassium current between epicardial and endocardial surfaces.

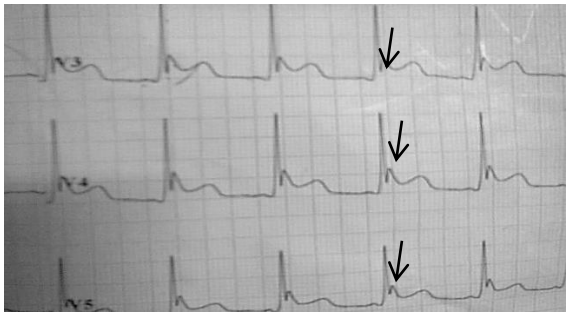


Fig.15. *Hypothermia showing Osborn Wave.(arrow).This hypothermia was due to paracetamol poisoning.*

VI. Drug Toxicity

Many non-cardiac drugs produce ECG changes at their toxic levels. Tricyclic antidepressant toxicity typically produces wide QRS, sinus tachycardia and terminal R in avR. Terminal R wave in avR more than 3mm, QRS duration more than 100m.sec and sinus tachycardia are bad prognostic signs[7] (Fig.16). Many chemotherapeutic drugs especially anthracyclines cause cardiac dysfunction and induce changes of myocardial ischemia

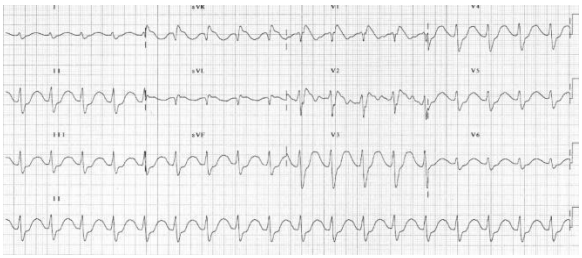


Fig.16. *ECG showing sinus tachycardia, wide QRS and tall* R *in av*R *due to tricyclic antidepressant toxicity.*

VII. Poisoning

Cardiac toxicity is a common finding in patients who have been poisoned with wide variety of chemical agents.

A. Carbon monoxide (CO) poisoning typically produces ischemic changes in ECG due to inhibition of cellular respiration[8]. (Fig.17)

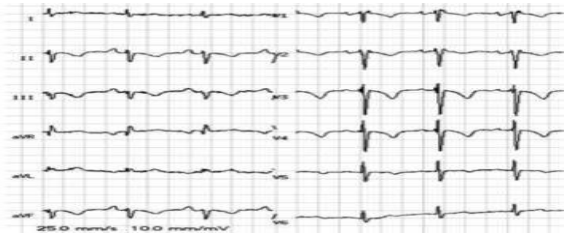


Fig.17. *ECG showing diffuse T inversion due to CO monoxide poisoning which is an indication for hyperbaric therapy.*

B. Organo phosphorous poisoning, cyanide poisoning and heavy metal poisoning produce arrhythmias and ECG changes of ST elevation mimicking STEMI.(Fig.18.)

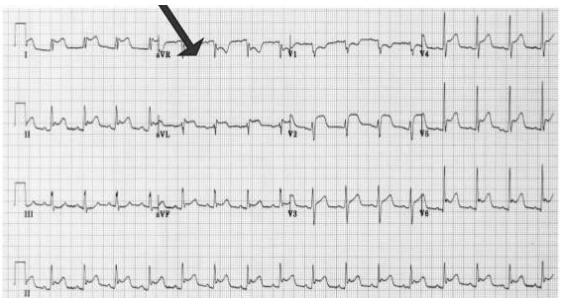
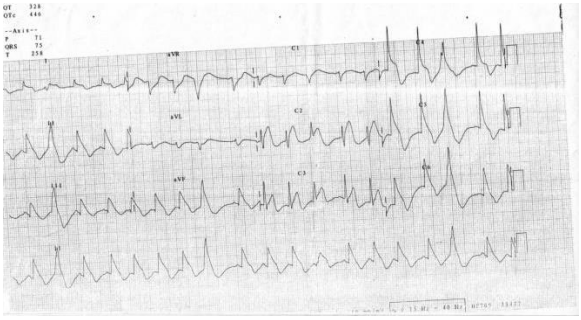


Fig.18. *ECG of a patient with OP poisoning*

C. Aluminium Phosphide Poisoning:

One of the common insecticides which is used in South India is Aluminium Phosphide (ALP). ALP poisoning produces cellular hypoxia due to inhibition of cytochrome oxidase in mitochondria. This may produce diffuse ST elevation mimicking Acute Myocardial Infarction (Fig.19).



Fig, 19*. ECG showing diffuse ST elevation due to ALP poisoning.(see text)*

VIII. Tremors

Tremors due to various reasons especially Parkinsonism produce somatic tremor artefacts (STA). This STA will mimic arrhythmias such as atrial flutter, Torsade de pointes and may be wrongly treated with powerful antiarrhythmic agents and DC shock[9]. The clinical examination during the arrhythmia will show disparity between pulse and ECG. The ECG in Parkinsonism is shown in Fig.20, which exactly looks like Torsade de pointes. Careful examination of L II which is simultaneously recorded with other leads did not show the arrhythmia, confirming the diagnosis of tremors. Further careful examination of limb leads confirm that the leads using left arm such as L1, L III, avL showed the ECG changes and not L II which is not using left upper limb indicating the tremor is maximum in left upper limb. So the ECG can be utilized not only to diagnose tremors but also the limb of tremors!

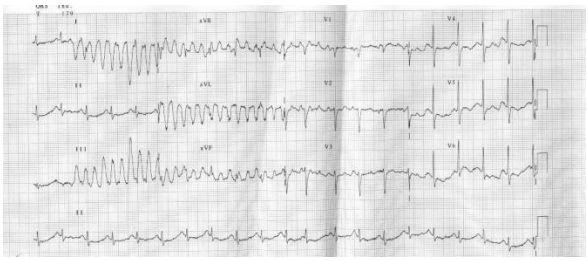


Fig.20*. ECG showing Parkinson tremor mimicking Torsade de Pointes. Note that LII which is simultaneously recorded with LI and LIII does not show same ecg changes confirming STA*

IX. Lead Misplacement:

A. Upper arm lead reversal is well known to cause technical dextrocardia where limb leads show the evidence of dextrocardia (P,QRS negative in L I and positive in avR) but chest leads show normal R wave progression (fig 21).

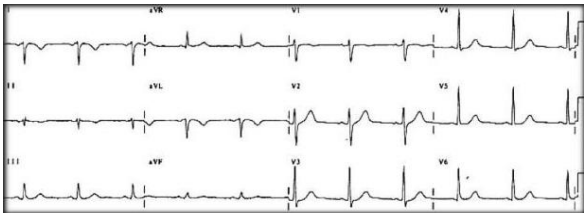


Fig 21: *Right arm and Left arm lead reversal leading to positive complexes in lead aVR and negative complexes in lead I (Technical or pseudo dextrocardia)*

B. Lower Limb Lead Reversal:

Less well known is the reversal of electrodes between upper and lower limbs[10]. In Fig.22a and Fig. 22b upper, lower limb lead reversal actually changes site of infarction. The actual inferior wall MI is shown as High lateral MI due to upper, lower limb lead reversal.The clue for lower limb lead reversal is the inveted P in LIII.

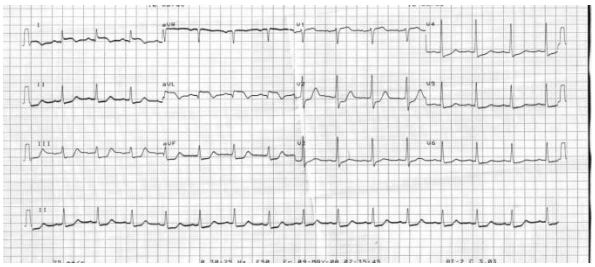


Fig 22a*. ECG showing High lateral MI like picture because of upper limb, lower limb lead reversal.*

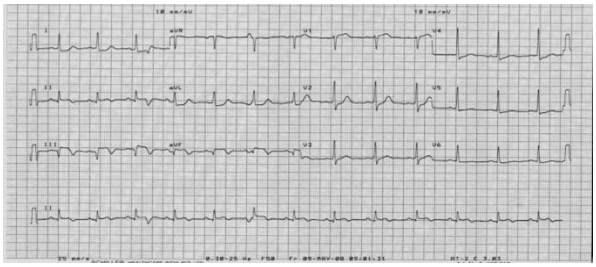


Fig.22b. *Correctly recorded ECG showing actual inferior MI*

X. Pregnancy

Pregnancy produces a lot of ECG changes such as Sinus tachycardia, non specific ST T changes, short PR, rare premature beats and minor axis deviation towards left due to elevation of diaphragm[11] (Fig.23).

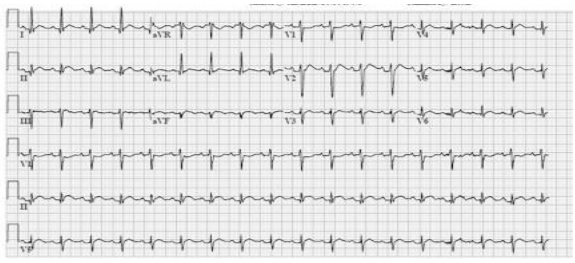


Fig 23: *ECG changes in pregnancy*

The pathological changes in ECG during pregnancy are listed in Table 1

Table 1: Pathologic changes in ECG in pregnancy

Sinus Bradycardia
A.V.Blocks (New onset)
Complex Premature beats
Atrial Fibrillation
Significant chamber enlargements (LA,LV,RV)
Ischemic changes(deep T inversion, ST elevation or depression)

XI. Posture:

Changes in posture itself can produce significant ECG changes. Standing may produce T wave changes and axis shift (Fig.24 a)when compared to ECG in lying (Fig.24b); so when interpretating ECG it is important to know in which position the ECG is taken

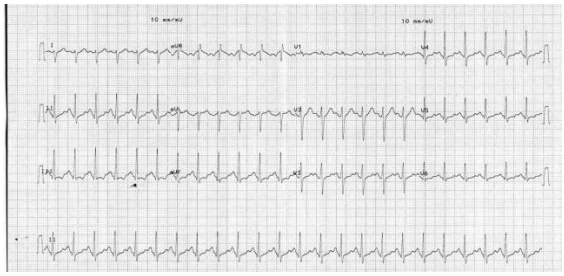


Fig 24a*: ECG in erect posture- Right axis*

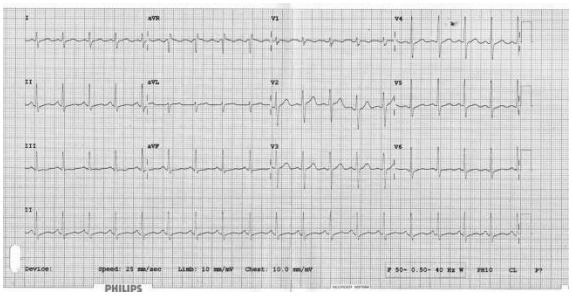


Fig.24b: *ECG of the same pt. in fig .24a in lying posture- axis normalized.*

XII. Renal Disease:

ECG in chronic kidney disease (CKD) usually shows LVH, Left Atrial Enlargement and most often hyperkalemia[12]. Sometimes combination of electrolyte abnormalities may produce some typical ECG changes which are diagnostic of chronic renal diseases. The combination of hypocalcemia and hyperkalemia show prolonged ST segment (Hypocalcemia) and peak T waves (hyperkalemia) (Fig.25). Although in this ECG, Twave is not typical of hyperkalemia because of decreased amplitude, one must suspect associated hyperkalemia because of T waves with a sharp apex.

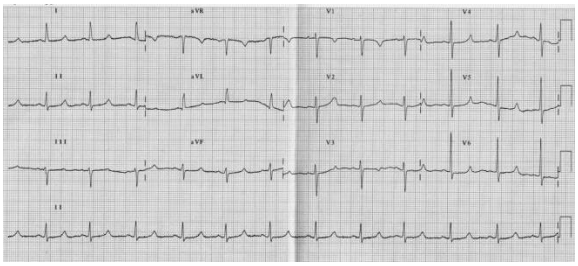


Fig 25: *ECG in a CKD patient with hypocalcemia and hyperkalemia*

Conclusion:

Most often, whenever there are ECG changes it is presumed, it is due to cardiac disease. It should be realised that many non-cardiac conditions can produce significant ECG changes which are mistaken for cardiac disease and wrongly treated especially in critical care settings. The clinical correlation, careful study of ECG and awareness of ECG changes in non-cardiac conditions will prevent many such therapeutic disorders. So, the ECG gives you a lot of "**E**xtra **C**ardiac **G**uidance".

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