



ECG & ELECTROLYTES IMBALANCE

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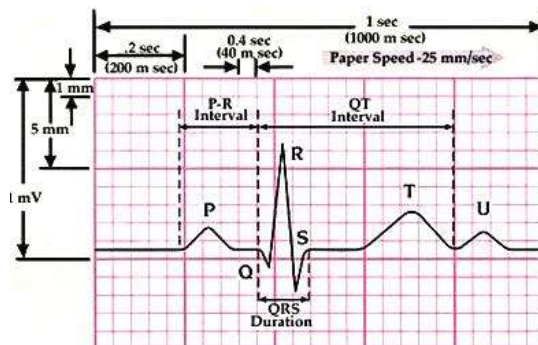
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Systematic ECG analysis and interpretation

- 1- Calibration / speed
- 2- Rate
- 3- Rhythm / Regularity
- 4- Cardiac Axis
- 5- P wave
- 6- QRS Complex
- 7- T wave
- 8- U wave
- 9- PR interval
- 10- QT interval
- 11- PR segment
- 12- ST segment
- 13- Other waves (delta, osborne, epsilon, pacing spikes, artifacts)



Case1

A 65-year-old man known hypertensive and CRF, presented with generalized weakness, muscle cramps and palpitation

O/E:

Pulse:100/m

B.P:180/90



The T waves are tall especially in lead V3; they are symmetric, narrow, pointed and tented, as if pinched from above. These findings are characteristic of **hyperkalemia**

CAUSES OF HYPERKALEMIA

- Renal failure
- Hypoaldosteronism (Addison's disease)
- Drugs
 - ACE- inhibitors
 - Potassium-sparing **diuretics**
- Obstructive uropathy
- Redistribution of potassium
 - acidosis
 - hyperkalemic periodic paralysis (familial)
 - hypertonicity (mannitol, hyperglycemia)
 - cellular damage - burns, crush injury
 - severe physical exertion

Pseudohyperkalemia

- *hemolysis during blood sampling or centrifuging

ECG CHANGES

ECG signs may be absent if the onset of hyperkalemia is slow - as seen in chronic renal failure - even though the serum potassium is in the range of 7 - 7.5meq/L;

Acute hyperkalemia can produce ECG signs at much lower serum potassium levels.

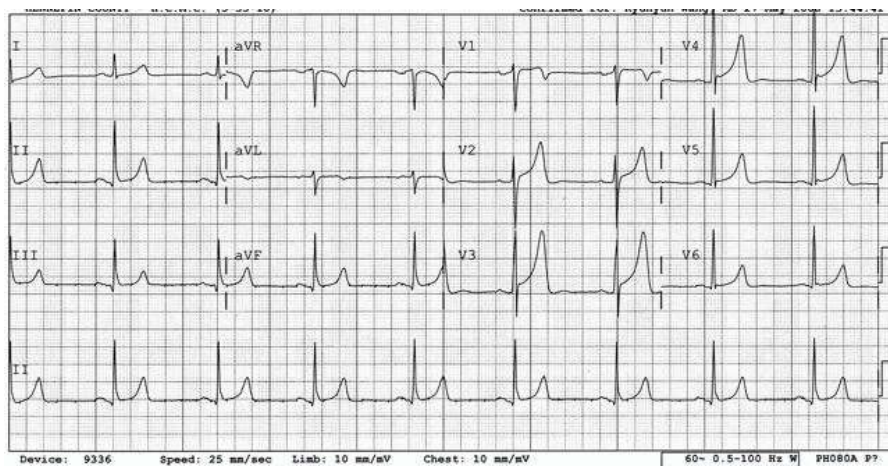


- **Serum K⁺ > 5.5 mEq/L** is associated with **repolarization abnormalities**:
Peaked T waves (usually the earliest sign of hyperkalaemia)
- **Serum K⁺ > 6.5 mEq/L** is associated with **progressive paralysis of the atria**:
P wave widens and flattens
PR segment lengthens
P waves eventually disappear
- **Serum K⁺ > 7.0 mEq/L** is associated with **conduction abnormalities & bradycardia**:
Prolonged QRS interval with bizarre QRS morphology
High-grade AV block with slow junctional and ventricular escape rhythms
Any kind of conduction block (bundle branch blocks, fascicular blocks)
Sinus bradycardia or slow AF
Development of a **sine wave** appearance (a pre-terminal rhythm)
- **Serum K⁺ > 9.0 mEq/L** causes **cardiac arrest** due to:
Asystole
Ventricular fibrillation
PEA with bizarre, wide complex rhythm

acute STEMI, the T wave is initially tall and is called hyperacute T wave changes -- "more acute than acute." In such a case, the T wave is symmetric but not narrow, not pointed, and not tented



T waves are tall in leads V3 and V4; they are not symmetric (the upstroke takes more time than the downstroke) and they are not narrow. In some patients, **tall T waves like those shown are normal.**



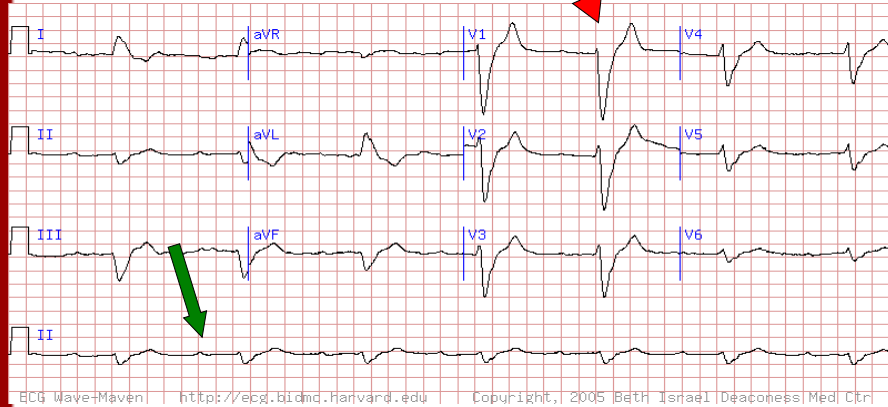
<p>Hyperkalemia</p> <p>Symmetric, narrow-based, pointed, tenting</p>	<p>Hyperacute Ischemia</p> <p>Symmetric, broad-based, not tented, not pointed QT interval tends to be long (not in this example)</p>	<p>Normal Variant</p> <p>Asymmetric and not narrow</p>
<p>Classic Definition of Early Repolarization: ST Elevation</p> <p>Classic Early Repolarization Without a J-wave Classic Early Repolarization With a J-wave</p>		<p>New Definitions of Early Repolarization</p> <p>Normal QRS Downstroke without STE J-point of the new 'J-point Elevation' without STE</p>

Hyperkalemia
 Sinus rhythm
 The QRS complexes are wide even for the RBBB.
 The PR interval is long.
 T waves are tall, tented, and pointed, especially in V4

COMMENTS:

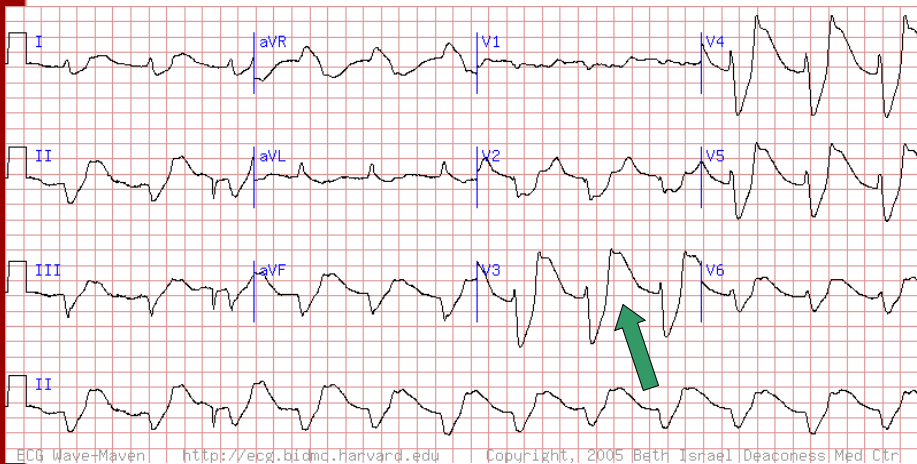
25mm/s 10mm/mV 150Hz 8.0.1 12SL V 241 CID. 0 EID. EDIY ORDER: 156743931 ACCOUNT: EB47 Page

case. The patient is an elderly man who presented to the emergency ward with dizziness and new renal failure.



- **Hyperkalemia (7.6 mEq/L) secondary to renal failure. widening of the QRS complex. There is also peaking of the T waves with prolongation of the PR interval and flattening of the P waves.**

case. The K⁺ was 10.5 mEq/L in a patient with renal failure.



- **Note the wide QRS complexes with no evident P waves.**
- **CK was normal and ST elevations were likely due to hyperkalemia.**

TABLE 9-10 Differential Diagnosis of ST Segment Elevation

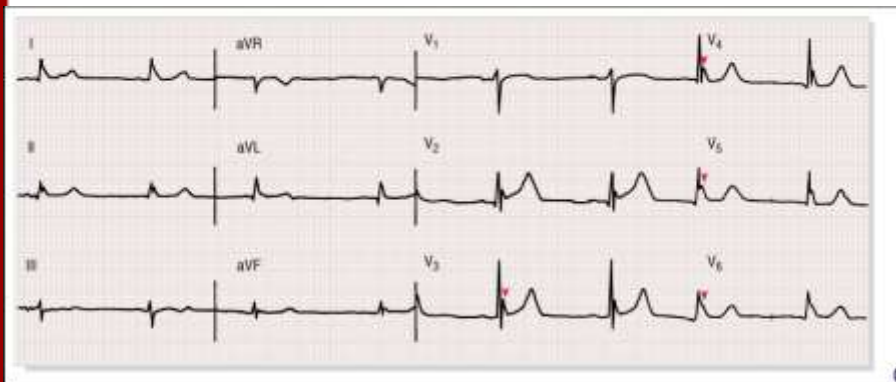
Myocardial ischemia/infarction
Noninfarction, transmural ischemia (Prinzmetal angina pattern) (see Fig. 9-40)
Acute myocardial infarction (see Fig. 9-37)
Post myocardial infarction (ventricular aneurysm pattern)
Acute pericarditis (see Fig. 9-48)
Normal variant ("early repolarization" pattern) (see Fig. 9-16)
LVH/LBBB (V ₁ -V ₂ or V ₃ only)
Other (rarer)
Myocardial injury
Myocarditis (may look like myocardial infarction or pericarditis)
Tumor invading the left ventricle
Trauma to the ventricles
Hypothermia (J wave/Osborn wave) (see Fig. 9-53)
After DC-cardioversion
Intracranial hemorrhage
Hyperkalemia*
Brugada pattern (RBBB-like pattern and ST elevations in right precordial leads)*
Type 1C antiarrhythmic drugs*
Hypercalcemia*

LBBB = left bundle branch block; LVH = left ventricular hypertrophy; RBBB = right bundle branch block.

Modified from Goldberger AL: Clinical Electrocardiography: A Simplified Approach, 6th ed. St Louis, CV Mosby, 1999.

*Usually localized to V₁ to V₂.

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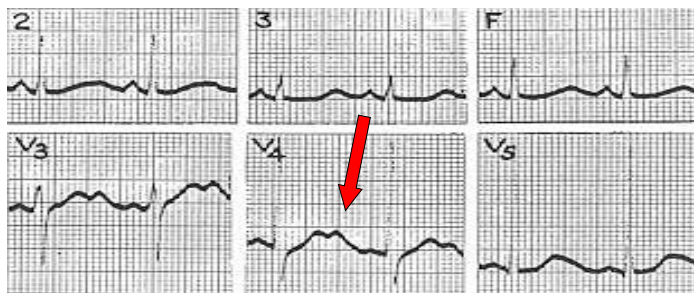
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Systemic hypothermia. The arrows (V3 through V6) point to the characteristic convex J waves, termed *Osborn waves*. Prominent sinus bradycardia is also present.

HYPOKALEMIA

ECG changes in hypokalemia

- 1. Flattened T waves and prominent U waves (apparent QT interval prolongation)



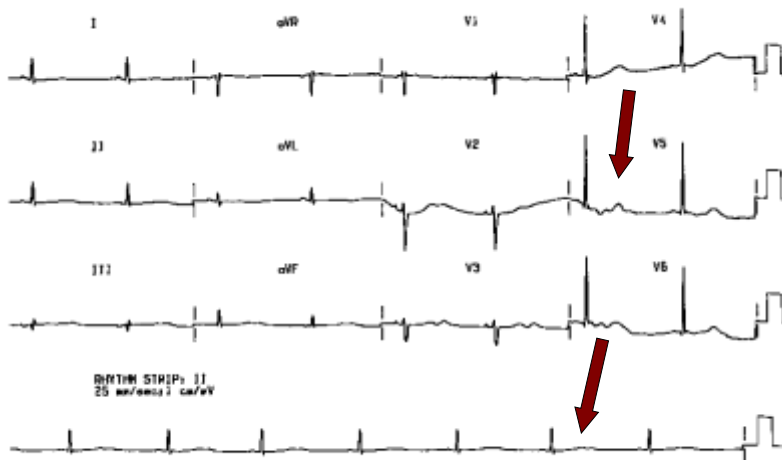
Note the prominent U wave in leads V3 and V4 giving the conjoined T-U wave the appearance of a "camel's hump"

2. Prominent U waves combined with depressed ST segments and flattened T waves ("roller-coaster effect")



- note the depressed ST segments with flattened/equiphasic T waves in leads V4 and V5 and V6
- note the small flattened T wave in leads V2 and V3 and how the ST segment dips down slightly (just after the flattened T wave) before rising to the prominent U wave (that has merged with the following P wave) creating a "roller-coaster" effect

case. A 22 year old lady with prolonged vomiting.



This lady's serum potassium was 1.8 mmol/L.

Causes of Hypokalemia

Inadequate intake

- diet very low in potassium

Excessive gastro-intestinal loss

- secretory diarrhea
- GIT fistula or small bowel enterostomy
- malabsorption syndrome
- excessive, voluminous vomiting
- laxative abuse

CAUSES OF HYPOKALEMIA

Excessive urinary loss

- hyperaldosteronism - primary or secondary
- osmotic diuresis
- diuretic drugs
- renal tubular diseases

CAUSES OF HYPOKALEMIA

Redistributional

- acute insulin administration during DKA => rapid potassium uptake by the cells
- beta-sympathomimetic therapy
- hypokalemic periodic paralysis - familial or associated with thyrotoxicosis
- excessive sweating or skin loss secondary to extensive burns

Determining the cause of the hypokalemia

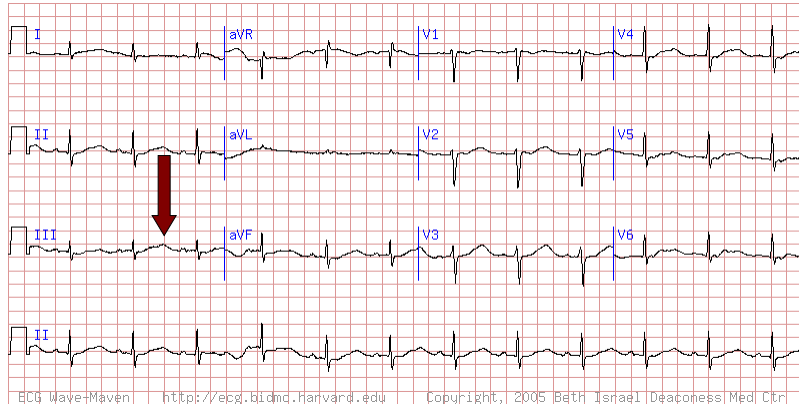
Diuretic therapy is the most common cause of hypokalemia in ED patients

Alcoholism is also a common cause of hypokalemia in ED patients

Drug-induced hypokalemia

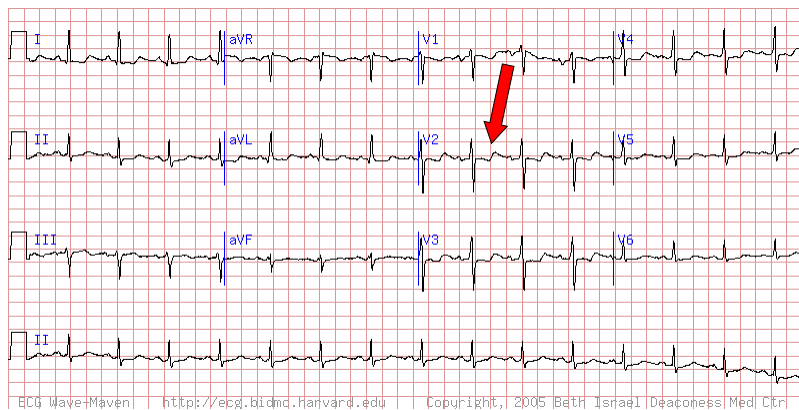
- | | |
|--------------------------|--------------------------------|
| • beta agonists | • Mineralocorticoids |
| • theophylline | • aminoglycosides (Gentamycin) |
| • caffeine | • chewing tobaccos |
| • verapamil intoxication | • glucocorticoids |
| • insulin overdose | • <u>High dose antibiotics</u> |
| • thiazides | • penicillin |
| • furosemide | • ampicillin |
| | • carbenicillin |

case. 30-yr-old man, not on medication, he had severe hypokalemia (1.5 mEq/L) due to diarrhea. Calcium and magnesium were normal.



- There are prominent U waves with Q-T(U) prolongation.
- this acquired-type long-QT(U) syndrome puts subject at risk for torsade

case. 31-year-old woman with ECG simulating ischemia. What metabolic abnormality should you suspect?



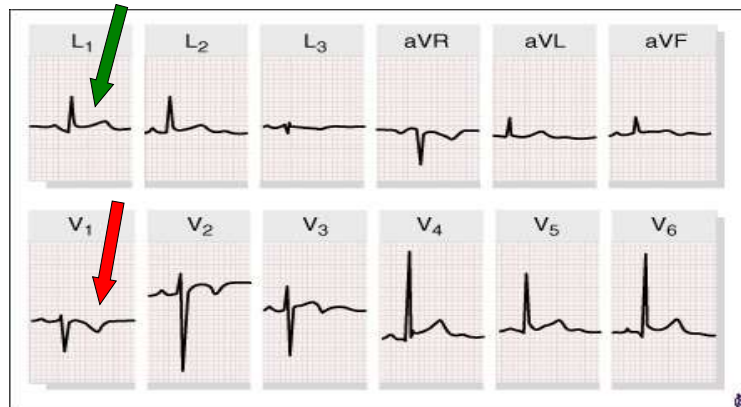
- K⁺ here was 2.2mEq/L.
- Very severe **hypokalemia** can cause ST depressions and T-wave inversions as seen here, mimicking ischemia. But also note large U wave (positive deflection after ST-T and before P).

TABLE 9-11 Differential Diagnosis of Prominent T Wave Inversion

Normal variants Juvenile T wave pattern (see Fig. 9-15) Early repolarization
Myocardial ischemia/infarction (see Fig. 9-49)
Cerebrovascular accident (especially intracranial bleeding) and related neurogenic patterns (e.g., radical neck dissection, Stokes-Adams syndrome) (see Fig. 9-49)
Left or right ventricular overload Classic "strain" patterns Apical hypertrophic cardiomyopathy (Yamaguchi syndrome) (see Fig. 9-49)
Post-tachycardia T wave pattern
Idiopathic global T wave inversion syndrome
Secondary T wave alterations: bundle branch blocks, Wolff-Parkinson-White patterns
Intermittent left bundle branch block, preexcitation, or ventricular pacing ("memory T waves")

Modified from Goldberger AL: Clinical Electrocardiography: A Simplified Approach. 6th ed. St Louis, CV Mosby, 1999.

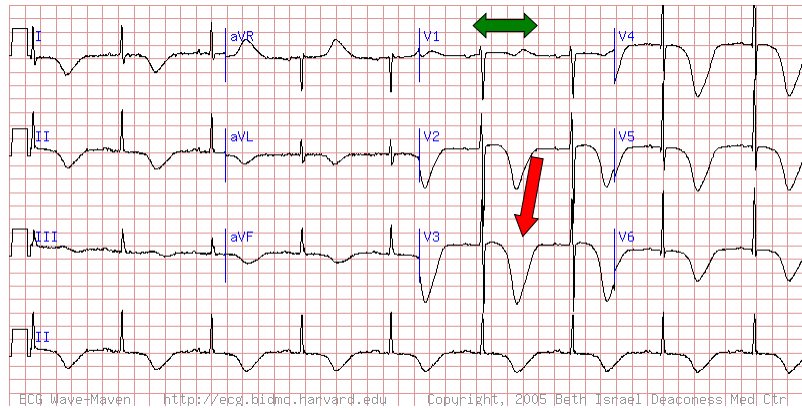
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Normal tracing with a juvenile T wave inversion pattern in leads V1, V2, and V3, as well as early repolarization pattern manifested by ST segment elevation in leads I, II, aVf, V4, V5, and V6.

A 71-year-old woman in the ER with end stage renal disease developed mental status changes and had this ECG.



- "CVA-T wave pattern," especially with intracranial bleeds
- This ECG shows massive diffuse, very broad T wave inversions with marked QT or QT-U prolongation (see lead V3).

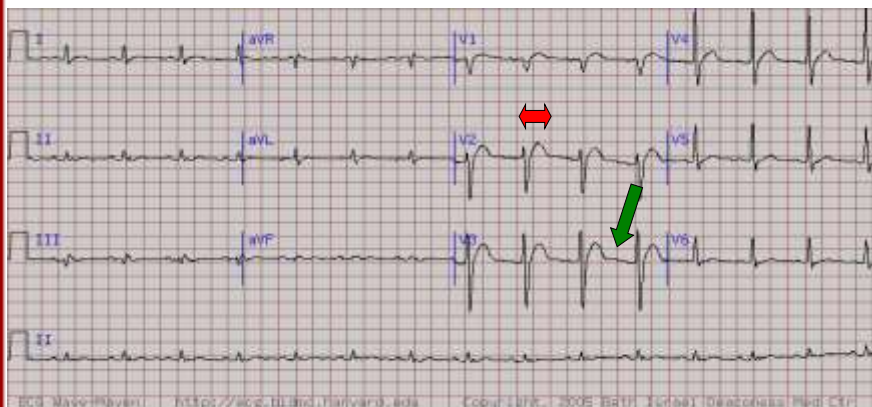
HYPERCALCEMIA

- **Hpercalcemia** is defined as a **serum calcium > 10.5 mg/dl** and severe hypercalemia is defined as a **serum calcium > 14 mg/dl (> 3.5 mmol/L)**

ECG CHANGES IN HYPERCALCEMIA

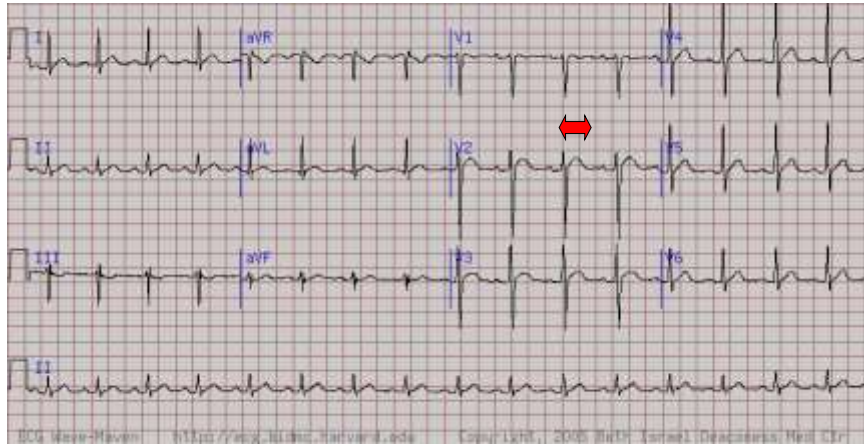
- **ECG** may demonstrate a **shortened QT interval** and **bradyarrhythmias**

case. 49-year-old man who presented with progressive muscle weakness, paresthesias, and constipation.



- Very short ST segment with a consequently short QT interval; and prominent U waves (esp. in V3). These changes are very suggestive of hypercalcemia.
- This patient's serum calcium was 16 mg/dl. He was found to have hyperparathyroidism.

case. 35-yr-old female in the ICU just admitted with suspected intestinal obstruction



Classic ECG for **hypercalcemia** (13.9 mg/dl here). Note the very abbreviated ST segment such that the T wave looks like it takes off directly from QRS in some leads.

Risk factors for hypercalcemia

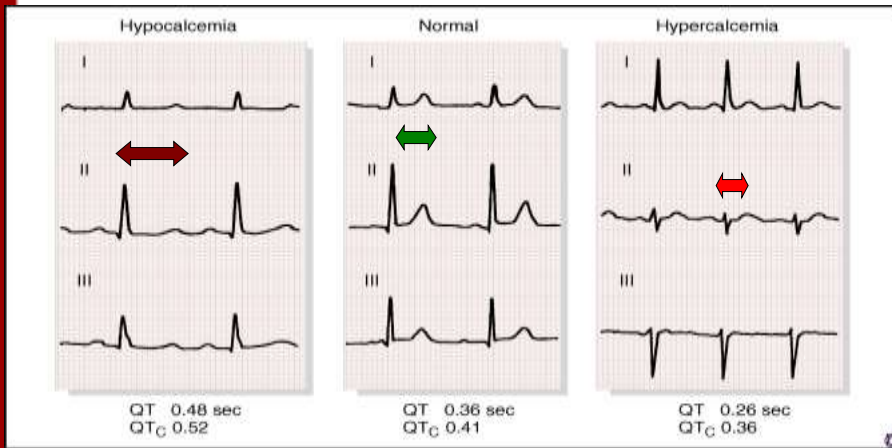
- **Hyperparathyroidism**
- **Malignancy**
- **Granulomatous disease**
- **Non-parathyroid endocrine disorders**
 - hyperthyroidism
 - adrenal insufficiency
 - pheochromocytoma

Risk Factors for Hypercalcemia

- **Vitamin D intoxication**
- **Vitamin A intoxication**
- **Mild alkali syndrome**
- **Chronic renal insufficiency, or dialysis patients taking vitamin D replacement therapy**
- **AIDS**
- **Drugs**
 - lithium
 - aminophylline
 - thiazide diuretics

Hypocalcemia

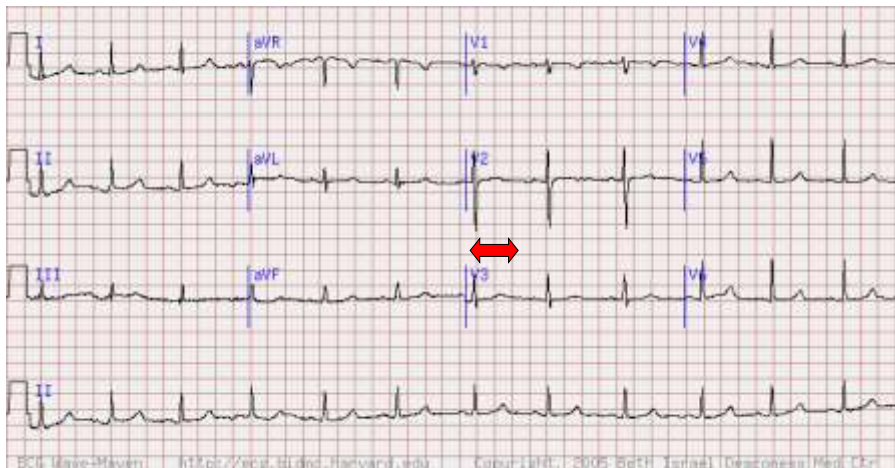
ECG CHANGES



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- Prolongation of the QT interval (ST segment portion) is typical of hypocalcemia.

Case. 34 yr Male, with muscle cramping & carpopedal spasms post thyroidectomy



Check the calcium level = was low 8.2 mg/dl in patient post thyroidectomy.
ECG shows QT prolongation. Most important is that **QT is long in this case because the ST segment is stretched out.**

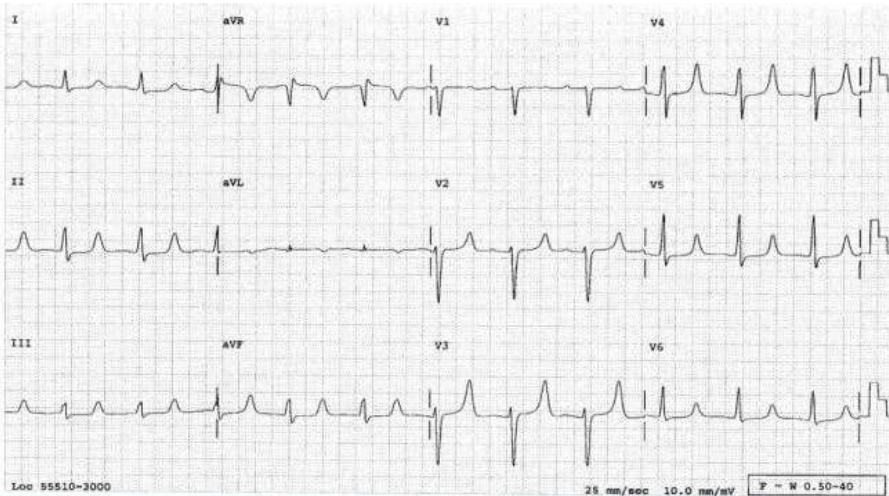
CAUSES OF HYPOCALCEMIA

1. It may be consequence of thyroid or parathyroid surgery.
2. If phosphate raised then either:
 - Chronic renal failure
 - Hypoparathyroidism or pseudoparathyroidism.
3. If phosphate is normal or low then either:
 - Osteomalacia or pancreatitis

Hyperkalemia and Hypocalcemia (Ch. Renal Failure)

The QT interval is prolonged, especially in leads II, III, aVF, and V4-6, typical features of **hypocalcemia**. The T wave is typically left unchanged. T waves are tall, symmetric, narrow, and tented and are typical of **hyperkalemia**.

This combination of electrolyte problems is common in patients with renal failure.



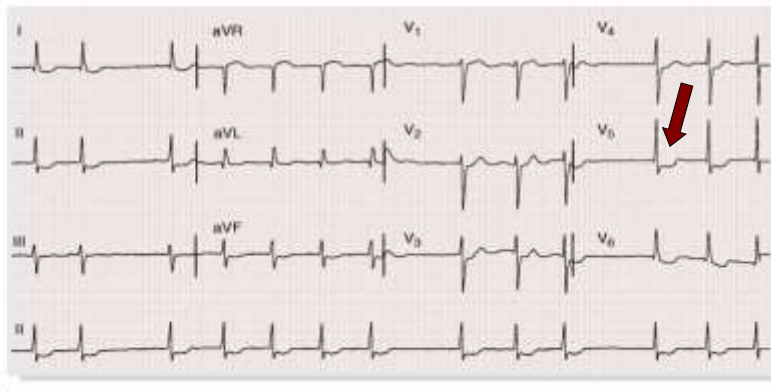
SYMPTOMS OF HYPOCALCEMIA

- Symptoms include **muscle cramping or carpopedal spasms**, distal extremity paresthesia, positive Chvostek's and Trousseau's sign
- Smooth muscle contraction can cause laryngeal stridor, dysphagia and bronchospasm
- Altered mental status or seizures may occur



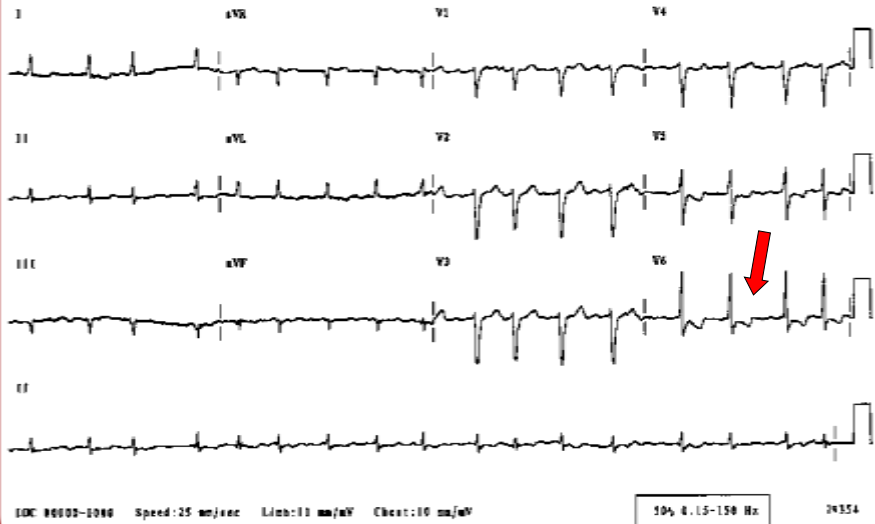
Digitalis effect

Shortened QT interval
 Characteristic down-sloping ST depression,
 reverse tick appearance.
 Dysrhythmias



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A 64 year old lady on digoxin



THANK YOU