Certified Emergency Training

ECG Recognition of Electrolyte Abnormalities

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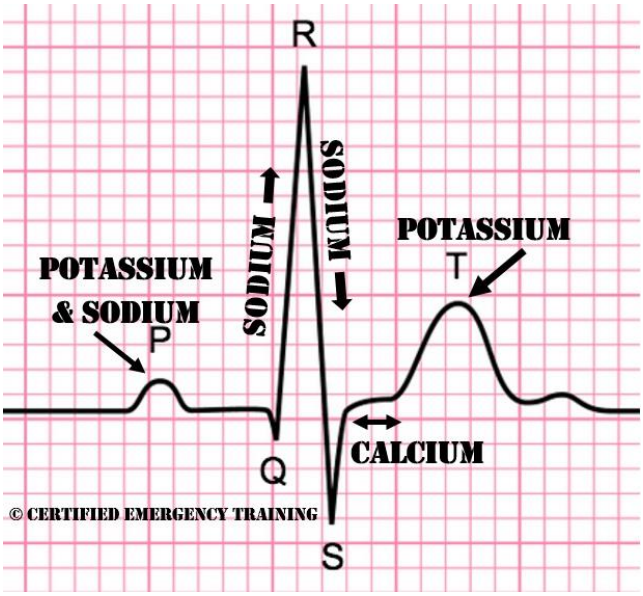
How do Electrolyte Abnormalities Affect the Heart?

Electrolytes & Cardiac Function



1.1. Charge of a cellular membrane depends on 4 ions:
   A. Sodium (Na^+)
   B. Calcium (Ca^{++})
   C. Potassium (K^+)
   D. Chloride (Cl^-)

1.2. Chloride doesn't play much of a role in the cardiac potential

1.3. Remember that Sodium and Calcium predominantly exist in the extracellular space, so when the membrane of a cell becomes permeable to their passage there is a massive in-rush into the cell and give it a large (+) electrical charge. Potassium has the opposite effect.

1.4. Electrolytes sodium, potassium, magnesium and calcium are involved in heart contraction and relaxation. Cardiac nerve impulse conduction begins when the calcium ions trigger calcium channels to open. At the same time the Calcium channel opens, Potassium rushes out of the cell and Sodium rushes into the cell. The heart muscle contracts. Almost immediately after the contraction, magnesium ions trigger potassium to rush back into the cell. Sodium is pushed back out of the cell. The heart muscle will relax.

2. Electrolyte Imbalances

2.1. According to the American Heart Association, ***potassium imbalances are the most common electrolyte-associated cardiac arrhythmias***.

   A. Potassium plays a role in both nerve conduction and the heart's ability to send an electrical impulse.
      i. Low potassium levels often result in relatively stable arrhythmias
      ii. High potassium levels often very quickly lead to lethal arrhythmias.

   B. Sodium, magnesium and calcium imbalances also place the heart at risk for arrhythmias. However, per the American Heart Association, arrhythmias caused by these electrolytes will only occur when the electrolyte levels are extremely low or high. Specifically, levels that are typically incompatible with human functioning, and are generally **FATAL.**

3. Specific Electrolyte Disorders

3.1. Sodium

A. Increased/Hypernatraemia or Decreased/Hyponatraemia Sodium levels do not have any effect on the ECG/Cardiac Rhythm/Impulse Conduction

3.2. Calcium

A. Hypercalcemia

i. Primary hyperparathyroidism and malignancies cause 90% of all Hypercalcemia.

ii. Can also be caused by: D Vitamin and Calcium Supplement Overdose, Immobilization, Thiazide Diuretics, Lithium, Addisons Disease, Renal Failure, Thyrotoxicosis, Sarcoidosis and Familial Hypocalciciuric Hypercalcemia.

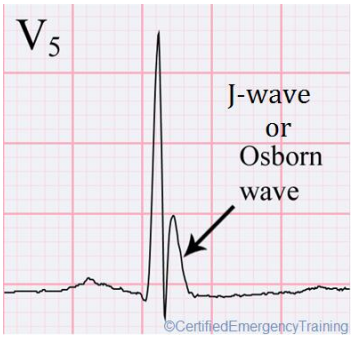
iii. ECG Changes Due to Hypercalcemia:

a. **Common ECG Changes**:

1. Shortened QT Interval (<0.33s)
2. Lengthened QRS (>.12s)
3. Possible Occurrence of Bradycardia (HR <60bpm)

b. **Rare ECG Changes**:

1. Increased QRS amplitude
2. Diminished T-Wave Amplitude
3. J-Waves (Osborn waves) consist of an extra deflection at the end of the QRS complex.
   - ⇒ J-Waves or Osborn waves are also called camel-hump waves, and hypothermic waves.
   - ⇒ Also seen in patients who are hypothermic.
   - ⇒ Best seen the inferior and lateral precordial leads.



4. AV Blocks (Can be of any degree)
5. ST Segment Elevations
6. Sinus Node Dysfunction and Tach-Brady Syndrome
7. Ventricular Tachycardia or Monomorphic V-Tach
8. Torsades De Pointes or Polymorphic V-Tach
9. Ventricular Fibrillation

B. Hypocalcaemia

i. Can be caused by:

a. Parathyroid Gland Surgery

b. Excess Calcitonin

c. Acute Pancreatitis, Chronic Pancreatitis, or Pancreatic Surgery

1. Ex. Total Pancrectomy, Whipple procedure (pancreaticoduodenectomy) or Distal Pancrectomy, Stent Placement, Etc.

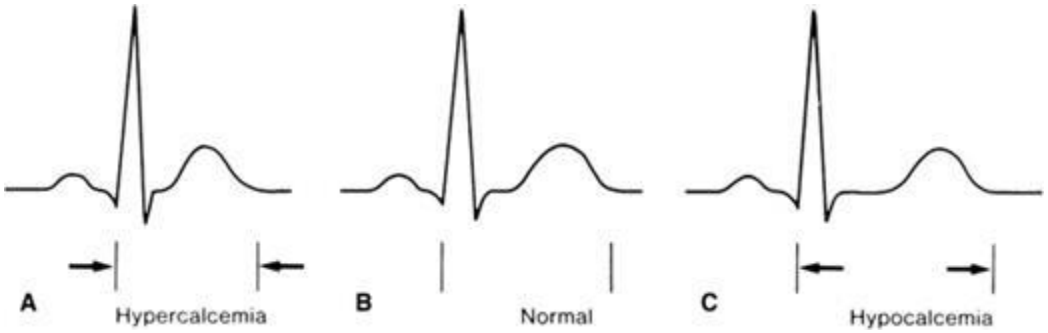
d. Alkalosis: Respiratory (Hyperventilation) or Metabolic

e. Abnormal Calcium Absorption (Gastrointestinal) and Abnormal Calcium Reabsorption (Kidney - From Primary Urine)
f. Osteolytic Cancer Metastases
g. Septicemia (Sepsis)
h. Rhabdomyolysis
i. Small Bowel Surgery
j. Use of Phenytoin, Biphosphonates, Phosphate Subsitution, or Forscarnet

ii. Common ECG Changes:
a. Shortened QRS (<.12s - No Clinical Significance)
b. Lengthened QT Interval or QT Prolongation (QT >.42S)

iii. Uncommon ECG Changes
a. Torsades De Pointes

iv. Rare ECG Changes
a. Sinus Bradycardia (HR ≤60 Bpm)
b. Sinoatrial Block (Block originating in the SA Node)
c. AV Block (Can be of any Degree)
d. Ventricular Fibrillation

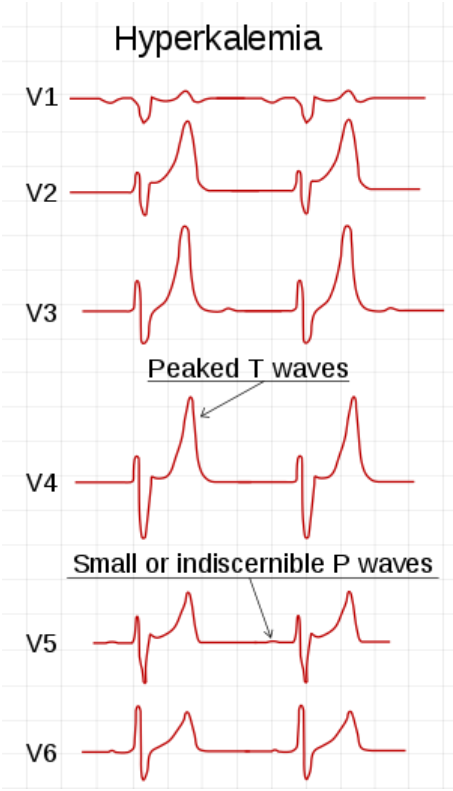


3.3. Potassium

A. Hyperkalaemia

i. Decreases impulse transmission in the entire heart.
ii. Symptoms occuer at 7mmol/L or Higher
iii. Can be caused by:
a. Usually the result of several interacting factors! Such as insufficient corticosteroid Substitution, Acidois, Hemolysis and massive muscle damages (Crush Syndrome), and renal failure
b. May be Caused by Potassium Substitution!
1. Potassium-sparing diuretics, ACE Inhibitors and Angiotensin Receptor Blockers (ARBs), may cause Hyperkalemia
c. Addison's Disease
d. Digoxin Toxicity (or Intoxication)
iv. ECG in **MILD** Hyperkalaemia

a. Earliest Sign: Pointed T-Waves



1. Most pronounced in the precordial or Chest Leads
2. Pointed T-Waves are tall and Narrow at top

b. In Patients with Left Ventricular Hypertrophy:

1. May display normalization of secondary T-Wave Inversions (I.e. Leads V5, V6, aVL, I)

v. ECG in **MODERATE** Hyperkalaemia

a. Pointed T-Waves become more pronounced

b. P-Waves:

1. Will become wider.
2. Amplitude will decrease.
3. May become difficult to discern.

c. P-R Interval

1. Becomes prolonged

d. Sinoatrial (SA) Block occasionally a Second- or third-degree AV Block can develop

e. Patients with WPW Syndrome

1. May lose their delta waves (Decreased Transmission through Accessory Pathway)

f. Possible S-T Segment Elevation in V1-V3

vi. ECG in **SEVERE** Hyperkalemia (Potassium >7.5mmol/L)

a. *All hyperkalemia changes become more pronounced*

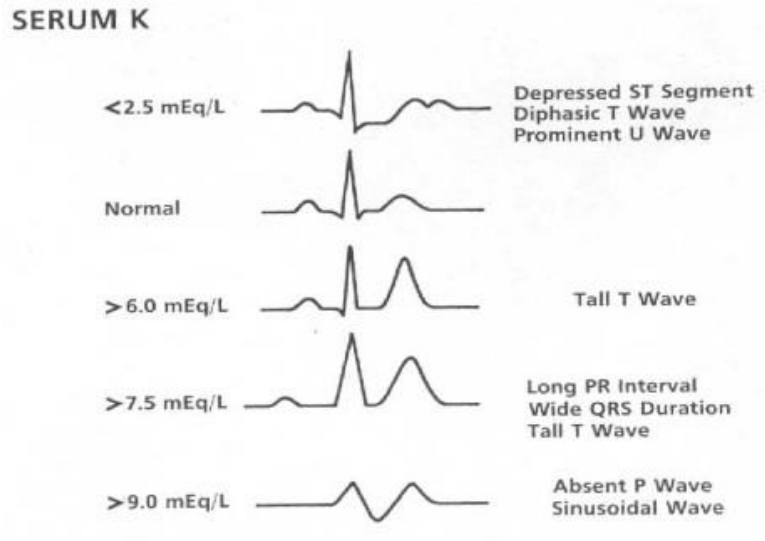
b. QRS Complex continues to widen

B. Hypokalemia

i. Serious complications may occur at 3 mmol/L and below!

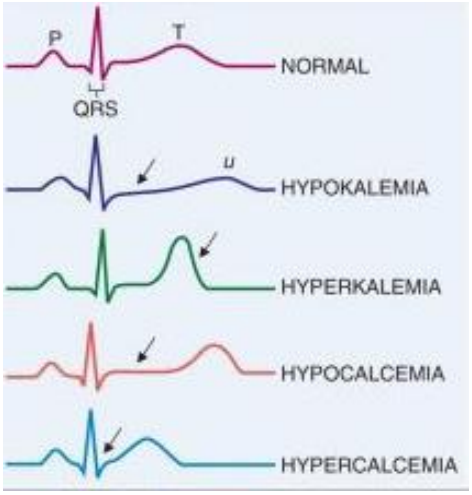
ii. Can be caused by:

a. Gastrointestinal Distress
   1. Diarrhea or Excess Vomiting
b. Acute or Chronic Pancreatitis
c. Malnutrition
d. Alcoholism
e. Acute Medical Illness
f. Excess intake of licorice
g. Aldosteronism
h. Glucose infusion
i. Insulin
j. Adrenergic Agonists
k. Diuretics



l. Corticosteroids
m. Theophyllamine

iii. ECG Changes in Hypokalemia
a. Occurs in Chronological Order as Potassium Level Decreases
1. T-Waves:
⇒ widen
⇒ decrease in amplitude
⇒ May invert in severe hypokalemia
2. S-T Segment:
⇒ Depression develops along with T-Wave Inversion this can simulate Ischemia!
3. P-Wave:
⇒ Amplitude Decreases
⇒ Duration and P-R interval Increases
4. U-Wave
⇒ Appear in Leads V2-V3
⇒ If Hypokalemia is severe the U-Wave may be larger than the T-Wave



b. May cause acquired Q-T Prolongation Syndrome or Long Q-T Syndrome (LQTS)
1. Patients predisposed to Tosades De Pointes (Polymorphic V-Tach)
c. May also cause Monomorphic Ventricular Tachycardia
d. Hypokalemia potentiates the pro-arrhythmic effects of Digoxin.

3.4. Magnesium

A. **Hypermagnesaemia** is rare but can be severe
i. May cause Atrioventricular and Intraventricular Conduction Disturbances
a. May result in Complete AV-Block (3rd Degree) or Asystole

B. Hypomagnesia
i. Can potentiate the pro-arrhythmic effects of Digoxin
ii. Patient predisposed to:
a. Atrial Tachyarrhythmia's
1. Supraventricular Tachycardia (SVT)
b. Ventricular Tachyarrhythmia's