# **EMC 451**

Advanced ECG Interpretation

Unit 14: Miscellaneous Patterns I

EMC 451: Miscellaneous Patterns

# Unit Objectives

- Upon completion of this unit, you should be able to:
  - List the causes, EKG changes, and treatment of hyperkalemia.
  - List the causes, EKG changes, and treatment of hypokalemia.
  - List the causes, EKG changes, and treatment of hypocalcemia.
  - List the causes, EKG changes, and treatment of hypercalcemia.
  - List the causes, EKG changes, and treatment of Torsade de Pointes.

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# Hyperkalemia

- Normal serum potassium is 3.5 5.0 mEq/L
- Principal intracellular cation. Over 98% of the body's potassium is contained within the cells.
- Serum potassium has major effect on cardiac and neuromuscular function.
- Serum potassium concentration reflects only the amount of ECF potassium.
- Acidosis shifts potassium out of cells into ECF
- Causes
  - Renal failure
  - Muscle injury
  - Acidosis (DKA or metabolic)
  - Insufficient corticosteriods (Addison's disease)
  - Dehydration
  - Potassium-sparing diuretics or ACE inhibitors
  - Potassium supplements

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# EKG Changes of Hyperkalemia all, peaked, T avaves (5.5 - 7.0 nEq/L) Twoses on till ord peaked the narrow Two avaves (7.0 - 8.0 nEq/L) Two avaves on till ord peaked the narrow Two avaves (9.0 - 0.0 0.00 mEq/L; reterminal stage) systole EKG Changes of Hyperkalemia Makka Andersita Makka Andersita Makka Andersita Two avaves on till ord peaked the narrow Two avaves on till ord peaked the narrow Two avaves on till ord Two avaves on

# Treatment of Hyperkalemia

- Be sure that T wave changes are not reciprocal changes of posterior wall ischemia (look for CP or T wave inversion and ST depression in inferior leads)
- Identify underlying cause
- · Correct acid-base disturbances
- Discontinue potassium-retaining diuretics, potassium supplements, or ACE inhibitors
- Administer calcium antagonizes effects of potassium but does not alter serum levels
- Administer glucose and insulin which drives potassium intracellularly
- Administer Kayexalate which removes potassium from the body

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# Hypokalemia

- Potassium level below 3.5 mEq/L
- Causes
  - Malnutrition
  - Excessive vomiting or diarrhea
  - Potassium-wasting diuretics (Lasix or thiazides such as HCTZ)
  - Cirrhosis of the liver
  - Diabetic coma after vigorous treatment
  - Hypochloremic alkalosis
  - Excessive secretion or administration of corticosteroids (Cushing's syndrome)
  - Excessive sodium bicarbonate administration

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# PENC SSI: Miscellancons Pagents 1 EKG Changes of Hypokalemia T waves flatten Nonspecific T wave changes U waves (may be result of quinidine, procainamide, phenothiazines, LVH) When present, U waves best seen in leads V<sub>2</sub> – V<sub>5</sub> EKG lacks sensitivity and specificity for hypokalemia

# EKG Changes of Hypokalemia

# Treatment of Hypokalemia

- Treat underlying cause
  - N&V
  - Change to potassium-sparing diuretic
- Correction of alkalosis
- Treat Cushing's syndrome
- If necessary, administer potassium supplements

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# Hypercalcemia and Hypocalcemia

- Calcium affects ventricular repolarization, which recognized by the QT interval.
- QT interval is measured from the beginning of the QRS complex to the end of the T wave.
- Normal QT intervals are dependent on the heart rate; as the HR increases, the QT normally shortens.
- You must use a table to determine normal QT interval, or use a rate-corrected QT (QTc).

 $QT_C = \frac{QT}{\sec RR}.$ • Normal QTc is  $\leq 0.44$  sec RR.

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# Hypercalcemia and Hypocalcemia

- Hypocalcemia prolongs the QT interval.
- Hypercalcemia shortens the QT interval.

Table 2.1 QT Interval: Upper Limits of Normal			Hypocalcemia	
Measured RR interval (sec)	Heart rate (per min)	QT interval upper normal limits (sec)		
1.50	40	0.50		
1.20	50	0.45		
1.00	60	0.42	- Am	
0.86	70	0.40		
0.80	75	0.38		
0.75	80	0.37		
0.67	90	0.35	1 1	
0.60	100	0.34		
0.50	120	0.31		
0.40	150	0.25	QT = 0.48 sec QT <sub>C</sub> = 0.52 sec	

Hypocalcemia	Normal	Hypercalcemia
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Mul	hh	~~~
		u III
-had	Jaha	TT
QT = 0.48 sec QT <sub>C</sub> = 0.52 sec	QT = 0.36 sec QT <sub>C</sub> = 0.41 sec	QT = 0.26 sec QT <sub>C</sub> = 0.36 sec

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- Most abundant mineral in the body.
- Regulated by the parathyroid gland.
- Required for clotting.
- Essential for the release of neurotransmitters in the central and peripheral nervous systems.
- Plays a critical role in muscle depolarization.
- Important for maintaining the strength of bone.

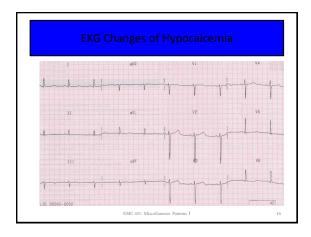
# **Hypocalcemia**

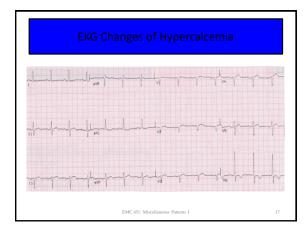
- Shock
- Sepsis
- Pancreatitis
- Hypomagnesemia
- Alkalosis
- Hypoparathyroidism
- Fat embolism syndrome
- Renal failure

# Signs and Symptoms

- Parasthesias around the mouth or in the fingertips
- Chvostek sign twitch at the mouth following tapping over the facial nerve
- Trousseau sign carpal spasm produced when BP cuff is inflated above systolic pressure for 3 minutes.

- Malignancies
- Hyperparathyroidism
- Hyperthryroidism
- Adrenal insufficiency
- Drugs (lithium, thiazides)
- Signs and Symptoms
  - Malaise
  - Dehydration, polydipsia
  - Confusion, hallucinations, ataxia, hyporeflexia
  - Fractures
  - Arrhythmias
  - Anorexia, weight loss, constipation, N&V, abdominal pain Renal insufficiency





# **Treatment of Calcium Derangements**

- Hypocalcemia
  - Treat underlying cause
  - Calcium chloride or calcium gluconate
  - Should be administered following transfusion of 6 units of blood
- Hypercalcemia
  - Correct dehydration
  - Promote excretion of calcium with diuretics
  - Calcitonin
  - Glucocorticoids

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# Torsade de Pointes

- A form of polymorphic VT
- Occurs in the setting of delayed ventricular repolarization, evidenced by prolonged QT intervals or prominent U waves
- Often initiated by a PVC that occurs on or near the T or U wave.
- Causes
  - Drugs (quinidine, disopyramide, procainamide, TCA, phenothiazines)
  - Electrolyte Disturbances (hypokalemia, hypomagnesemia, hypocalcemia) which prolong repolarization
  - Severe bradycardia
  - Hereditary long QT syndrome
  - Liquid protein diets

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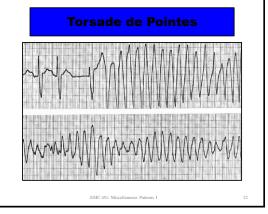
## Torsade de Pointes

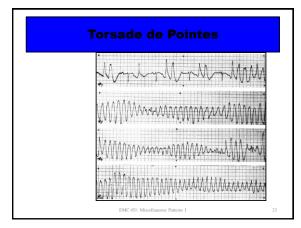
- · Warning Signs
  - Prolonged QT
  - Usually longer than 0.50 secs
  - Quinidine induced TdP has QT interval > 0.60 secs
  - Others have prolonged QT >0.44 secs
  - T waves
    - T-wave alternans or bizarre T-wave aberration following a postextrasystolic pause
    - T-wave distortions in congenital LQTS
  - Prominent U waves

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# Torsade de Pointes Grade II Grade II Grade II





# Treatment of Torsade de Pointes

- Correct underlying electrolyte imbalances
- Discontinue drugs that are potential causes (quinidine, procainamide, TCAs, amiodorone, phenothiazines, Seldane)
- · Magnesium Sulfate
  - 1-2 grams over 5 minutes
  - Followed by 1-2 gram infusion over 4-6 hours
- Overdrive pacing
- DC cardioversion is usually only transiently effective until underlying cause is corrected

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