AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
SCIENTIFIC ASSEMBLY
1999

Electrolyte Emergencies

Although it is almost routine in the busy emergency department to order a set of "lytes," there really are clear-cut situations in which electrolyte abnormalities may be the primary problem or a major part of a bigger problem. This lesson will cover most of the common and not-so-common electrolyte problems you will eventually encounter. The speaker will discuss potassium, calcium magnesium, and phosphorous.

- List the signs and symptoms suggestive of electrolyte abnormalities.
- List the most useful old treatments for common electrolyte abnormalities.
- Discuss new approaches to old problems.

MO-22
Monday, October 11, 1999
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Room # N223
Las Vegas Convention Center

FACULTY

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Electrolyte Emergencies

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HYPERKALEMIC/HYPOKALEMIC EMERGENCIES

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HYPERKALEMIA

Hyperkalemia is the most dangerous acute electrolyte abnormality.

Most K lives inside the cell (ICF = 140-155 meq/L), while a small amount, about 2% total body potassium, is in the extracellular compartment (ECF = 3.5 5.0 meq/L).

The potassium difference between the ICF and ECF is the key determinant of the resting membrane potential. As the serum potassium rises, PVCs, VT, a wide complex sine wave, or VF may occur.

There are many causes of hyperkalemia, but two rules must always be followed prior to treating patients:

1) **The number one cause of hyperkalemia is hemolysis after (or as) the patient's blood is drawn.**
2) **Treat the patient based on laboratory values and ECG changes, and not just lab values.**

There are 10 "common" causes of hyperkalemia:

- **Spurious** due to hemolysis during or after phlebotomy
  thrombocytosis
  leukocytosis
  - abnormal erythrocytes
- **Acidosis**
- **Renal Failure**
- **Iatrogenic** (usually associated with renal failure)
  - intravenous potassium containing medications
  amino acid infusions
- **Cell death**
  rhabdomyolysis
  - crush injuries
  burns
  tumor lysis syndrome
- **Addison's Disease** (or any low aldosterone state)
- **In vivo hemolysis**

- **Hematologic**
  WBC > 100,000
  HCT > 55-65
  PLTs > 1,000,000
  Tumor lysis syndrome

- **Hyperkalemic Periodic Paralysis**

- **Drugs**
  Aldactone and K sparing diuretics
  Captopril and other ACE inhibitors
  - NSAIDS
  Heparin
  Succinylcholine
  - Glucagon
  Beta Blockers
  - Calcium Blockers
  Digitalis (acute OD)

TREATMENT OF HYPERKALEMIA

There are 3 ECG changes due to hyperkalemia

1. **Tall Peaked T waves** (Seen as K rises above 5.5-6.0)
2. **P - R prolongation** followed by **loss of the P wave** (begins as K rises above 6.0-6.5)
3. **Widening of the QRS** (K usually above 7)

There are three steps to treating hyperkalemia.

Step 1: Reverse the deleterious electrical effects of potassium

a) 5 - 10 cc of 10% CaCl

Step 2: Drive potassium into the cell

a) 2 amps D_{50} over 5 - 10 minutes with 10 units of regular insulin IV push.

b) 2 amps (1 meq/kg) $NaHCO_3$ over 5 - 10 minutes - only if acidotic!

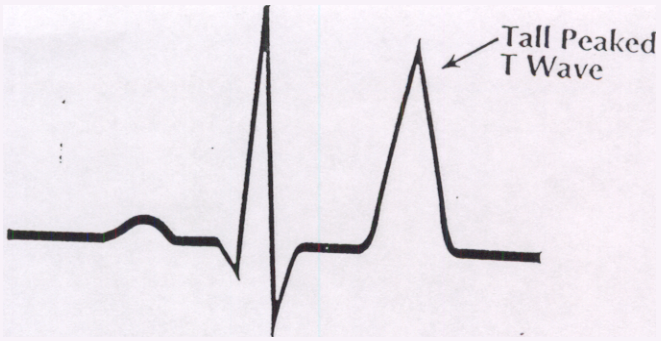
See pages 4-6 for additional methods for moving K into cell
(steps a and b are "standard')

Step 3: Remove potassium from the body

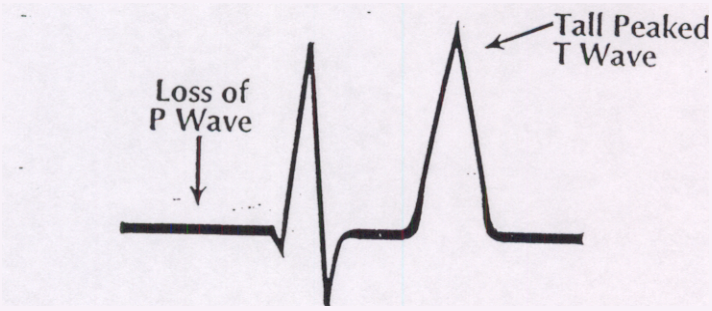
a) NSS at 200 cc/hr and lasix (40 - ? mg) to achieve urine output approaching 150 cc/hr.

b) Kayexalate 50 G in sorbitol PO or by enema

c) Hemodialysis

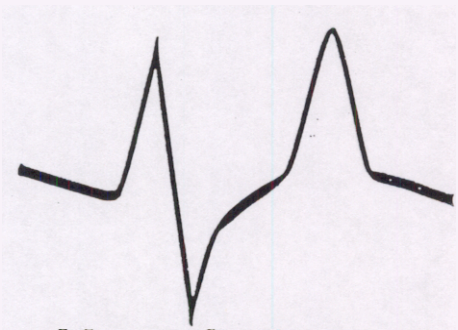


Hyperkalemia 1



Hyperkalemia 2

Widened QRS Merging With Tall T Wave



Hyperkalemia 3

Comments:

Step :

Calcium is **used only if the QRS is widened.** If unsure, use 5 cc to start. Use 10 cc in arrest.

- Calcium works by "tricking" the cell into thinking there's more of an electrical difference between intracellular and extracellular compartments.
- Ca does not move K intracellularly
- Ca does work for l-30 minutes to restore a more normalized electrical gradient and will temporarily narrow the QRS.

CaCl = 3x Ca gluconate

Approximately 13.6 meq of Ca in 10 cc of CaCl

vs

Approximately 4.6 meq of Ca in 10 cc of Ca gluconate

Calcium is highly sclerosing - try to give it via a large peripheral vein

Dose: In general 10 cc is an appropriate dose. You may want to start with 5 cc over 10 - 20 seconds if unsure; do not give more than 20 cc of CaCl in the first 30 minutes.

Step 2: Moving K Intracellularly

Glucose and Insulin

- Moves K intracellularly by stimulating glucose pump
- Drops K by about 1 .O meq over 20-60 minutes
- Steepest drop is in first 20 minutes
- Use 50 grams of glucose and 10 units insulin
- May give both IV push or over 5-15 minutes

BEWARE HYPOGLYCEMIA 1 HOUR LATER

Bicarbonate

- **Has no effect in non-acidotic patients**
- Bicarbonate is most useful in patients with serum HCO_3^- levels below 5-10
- Bicarbonate moves K intracellularly in acidotic patients
- Bicarbonate is an adjunct to CaCl, glucose and insulin
- USE bicarbonate in acidotic hyperkalemia patients
- USE 1 meq/kg over 10-20 minutes for most patients
- USE 1 meq/kg IV push in patients with sine wave QRS patterns
- USE IV push for hyperkalemic EMD/PEA

When things were simple, using bicarbonate for hyperkalemia was easy to explain:

For every 0.1 increase in pH, K falls by 0.6 meq

Unfortunately things are never as simple as in the old days.

Changes in plasma potassium concentration during acute acid-base disturbances.
Androgue HG, Madias NE. Am J Med 1981; 71:456-467.

There are varying effects on serum K when the pH changes and there are different changes with respiratory vs metabolic and in alkalosis and acidosis. Many factors other than just the bicarbonate may be at work. Reviews approximately 30 studies. "Acute acidemia usually results in hyperkalemia and acute alkalemia usually reduces plasma potassium. "

Effect of various therapeutic approaches on plasma potassium with major regulating factors in terminal renal failure.
Blumberg A., et al. Am J **Med 1988;** 85:507-512.

Bicarbonate (2-4 meq/min; total dose 120-240 meq) was ineffective in lowering K values in CRF dialysis patients. Glucose and insulin worked (K from 5.62 to 4.70). Note: pH of patients was 7.37 and went up to 7.51 with bicarbonate.

Bicarbonate in the treatment of severe hyperkalemia (letter)
Spittal. Am J Med 1989; 86:5 11.

Points oat patients of above study had normal pHs and urged bicarbonate use continue in acidotic patients.

Alkalinization Is Ineffective for Severe Hyperkalemia in Nonnephrectomized Dogs
Acad Emerg Med 1997;4:93-99.

A repeat variation of prior studies. This was a controlled canine study. Hypertonic Saline just as effective as HCO_3 in lowering K.

Beta Agonists

- cAMP mediated K pump stimulation
- Like epinephrine, stimulates K migration into cell
- Works additively with glucose and insulin
- Usually blocks the hypoglycemia seen with glucose and insulin
- Most studies have used IV albuterol NOT inhaled.
- Dose is usually 0.5 mg diluted in 100 cc given IV over 10-15 min. Lowers K by 0.5 meq 1 .0 meq over 15-30 min.
- May nebulize in 10-20 mg in 4 ml NS over 10 min

STOP BETA AGONISTS IF PVC's DEVELOP

Magnesium

- Stimulates Na-K ATPase pump
- Moves K into cell rapidly
- Works within 5 minutes
- Lowers K by about 0.5 meq
- Dose is 1-2 grams over 5-20 minutes
- May cause hypotension in dehydrated patients
- Be careful in patients with CRF
- An excellent antiarrhythmic for K induced ectopy

Volume

- Helps restore cellular Na-K gradient
- Especially good in dehydrated patients
- Especially bad in CHF/CRF
- Beware pulmonary edema

Step 3: Moving K Out of the Body

- Saline should be infused more rapidly in hypovolemic patients and slower in patients with CHF.

- Don't use saline and lasix if the patient cannot make urine.

- Each gram of resin binds 0.5 - 1.0 meq of potassium.

- Dialysis is excellent, but usually not readily available in the ED.
  Both HD and PD may be required for massive crush injuries.
  Continuous A-V hemofiltration may also be used.
  HD removes up to 50 meq of potassium/hour.
  PD removes only about 1/5 of that.

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Halperin ML, Kamel KS. Potassium. Lancet 1998;352:135-40.

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HYPOKALEMIA

Hypokalemia is a very common electrolyte abnormality; it is usually benign. Its two worst complications are:

1) **Cardiac Arrhythmias**
2) **Rhabdomyolysis**

Hypokalemia has many causes, the two most common causes for patients presenting to the ED are:

1) **Diuretic use**
2) **Malnutrition,** especially in association with **alcohol abuse.**

The most common causes of hypokalemia are:

1. **Renal losses**
   - Diuretic use (blocked reabsorption)
   - Steroid excesses (aldosterone excess)
   - Metabolic alkalosis (K lost in attempt to retain H^+)
   - Drugs (Amphotericin, Gentamycin, Carbenicillin, Ticarcillin)
   - DKA (Patients may present with elevated K)
   - RTA (May have huge renal losses)
   - ETOH (a diuretic)

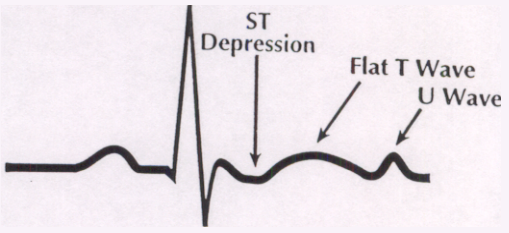
2. **Increased non-renal losses**
   - Sweating (especially in "untrained" athletes)
   - Diarrhea (also with villous adenoma)
   - Vomiting (Losses are via kidney!)

3. **Decreased intake**
   - ETOH (a diuretic and potassium deficient food all in one)
   - Malnutrition (CA, AIDS, poverty)

4. **Intracellular shift**
   - Hyperventilation
   - Drugs (epi, insulin, inhaled beta agonist, theophylline OD)

5. **Endocrine**
   - Cushing's Metabolic Alkalosis Syndrome (or steroid ingestion)
   - DKA
   - Barter's Syndrome

SIGNS AND SYMPTOMS OF HYPOKALEMIA



Hypokalemia

- Usually nonspecific
- Weakness, easy fatiguability
- Muscle pain
- Rhabdomyolysis

ECG in Hypokalemia:

Unreliable
Loss of T waves
Atrial Arrhythmias (PSVT, AF, etc.)
U waves
PVCs
NSSTW
VT, VF
Prolonged Q-T

TREATMENT OF HYPOKALEMIA

The treatment of hypokalemia is easy; just give potassium

General requirements:

Serum K	Total Body Deficit
Below 3.5	100
Below 3.0	200
Below 2.5	300 - 400
Below 2.0	400 600
Below 1.5	600 - 1000

Note: Alkalosis will drive potassium into cell and give a falsely low K value. The exact correction factor is not known, but is less than a .5 .6 falling K/each .1 pH rise.

Severe hypokalemia or chronic hypokalemia = hypomagnesemia until proven otherwise.

K burns, is sclerosing, and causes platelet aggregation due to microvascular irritation. One trick is to add lidocaine heparin and hydrocortisone. Only use when a small vein is to be used for lots of KCl:

Three ways to keep veins open and painless:

1) Lidocaine 100 mg.
2) Heparin 1000 units
3) Hydrocortisone 5 mg

Use in 1000 cc of fluid when large amounts of KCl is to be administered.

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Hyponatremia and Hypernatremia
Treating Two Similar Opposites

Rules

1. **Secure the ABC's.** Airway, Breathing, and Cardiovascular Stability override any and all sodium calculations.

2. **Stabilize BP and perfusion with Normal Saline** (0.9NSS) before beginning "tailored" therapy.

3. **NGT after ABC.** Consider the use of naloxone, glucose, and thiamine -- these patients may also use narcotics, be hypo or hyperglycemic and some are chronic alcoholics.

4. **Classify** each patient into Acute or Chronic and Symptomatic or Asymptomatic.

5. **No Therapy** is the best therapy for chronic and asymptomatic patients.

6. Only use **Hypertonic Saline (3%** Saline) for **2-3** house **at a maximum rate of 100cc/hr.**

7. **Hypertonic Saline** should only be used in **symptomatic** hyponatremia if patient is (1) acutely comatose, (2) has new AMS and/or (3) is seizing.

8. **Correct <u>All</u> patients at 0.5meq/hr**. And 12meq/day maximum (except if rule 7 is invoked for 2-3 hrs).

9. **Hyponatremia** at levels between 120-140meq/dl **doesn't hurt** anyone if it's corrected slowly.

10. **Always replace K aggressively** when treating hypokalemic patients with sodium disorders.

HYPONATREMIA

Overview

- **Most patients** presenting to the ED with hyponatremia are **stable** and **require no emergency therapy.**

- The most common cause of hyponatremia in the ED is **diuretic** use in conjunction with a **low salt diet** in a patient with **CHF.**

- **Severe hyponatremia** (Na below 1 10) **or symptomatic hyponatremia** (with Na below **120)** requires **immediate** therapy **either** with normal or hypertonic saline.

Brain Damage Due to Hyponatremia can be due to one or two different mechanisms:

(1) **Edema Induced:**
↑ brain swelling + ↑ ICP = I cerebral blood flow (in possible association with:)
a) increased pressure of brain against calvarium
b) cerebral hypoxia, and
c) herniation.

(2) **Therapeutically Induced:**
Hyponatremic brain cells become depleted of Na, K, and some proteins. This is an attempt by the brain to limit its osmolarity and thus limit the extent of cerebral edema.
If fluid therapy raises Na levels too quickly, cerebral edema, and demyelinization (via an unknown mechanism) may occur,

Although hyponatremia has many causes, they fall into 4 general categories.

THE FOUR CAUSES OF HYPONATREMIA

Pseudo Hyponatremia
Hyponatremia with dehydration
Hyponatremia with increased total body water
Hyponatremia with excesses of both total body sodium and total body water

Pseudo hyponatremia:

hyperglycemia (water pulled out of cells)
Na falls by 1.5-1.6 meq/L for every 100 mg% rise in glucose
hyperlipidemia (10 - 20% of measured volume is lipid and not fluid)
lab error or blood draw error (blood drawn near IV of D_5W or D_5 ½NS)

Hyponatremia with dehydration:

Also called "hypovolemic hyponatremia"
Patients are hyponatremic and are. dehydrated

causes:

1) **Body fluid losses**
   - Vomiting
   - Diarrhea
   - Third Spacing

2) **Renal losses**
   - Diuretics
   - Adrenal insufficiency
   - Renal disease (RTA)

To decide whether the kidney is causing the hyponatremia, **get a urinary sodium and/or urinary chloride.**

Urinary chloride is more reliable but fewer people are comfortable with ordering it.

Very low urine sodium or chloride (less than 10-20 meq/L) **= dehydration**
The kidney is holding on to any and all of the body's depleted sodium or chloride

High urine sodium or chloride (above 20 meq/L, may go as high as 70), **= kidney wasting Na and Cl and thus the kidney (or adrenal), and NOT dehydration, is the cause of the hyponatremia.**

Until proven otherwise:

Hyponatremia plus **Hyperkalemia** and **Dehydration = Adrenal Insufficiency.**

Treatment of hyponatremia in association with hypovolemia is easy:

I) Rehydrate the patient with NSS
2) Use NSS at 500-1000cc/hr until BP stable
3) **Go to NSS at 200 cc/hr and recheck serum Na at 2, 4, 6 hrs.**
4) Treat underlying cause of hypovolemia
5) Reassess serum sodium

Hyponatremia due to increased total body water
Also called "euvolemic hyponatremia" to confuse you; it is in fact, hyponatremia due to hypervolemia

Causes of Increased TBW Hyponatremia without edema

- **SIADH**
- **Psychogenic Polydipsia**
- **Hypothyroidism**
- **Adrenal Insufficiency**
- **Diuretic USE with mild CHF**

This hyponatremia is usually due to:
(1) the syndrome of inappropriate antidiuretic hormone [SIADH], or
(2) elderly patients using thiazide diuretics.

Note: Patients with SIADH have an excess volume,
but
no signs of edema, ascites or CHF.

Because ADH is an Antidiuresis hormone
Excess ADH causes water ↑ ∴ Na ↓

There are no signs of edema because most of the increased body water is intracellular and not intravascular.

CAUSES OF SIADH

1) Lung masses

Cancer, especially oat cell
Pneumonia
TB
Abscess

2) CNS disorders

Infection (meningitis, brain abscess)
Mass (Subdural, tumor, post-op, CVA)
Psychosis (may also have psychogenic polydipsia)

3) Drugs'

Thiazide diuretics
Narcotics
Oral hypoglycemics
Barbiturates
Antineoplastics

* many others reported, these are the most common

The key to treatment is based on:

(1) Presence or lack of symptoms
(2) How long it took for sodium to fall
(3) Absolute serum sodium value.

There has been much controversy over "slow" **vs "fast"** correction,

For relatively asymptomatic patients with Na values above 115 - 120, free water restriction is usually all that is required. In severe cases when AMS, seizures or coma is present, hypertonic saline is indicated. Normal or hypertonic saline may be used in conjunction with lasix. Lasix is used to block secretion of ADH which occurs when salt is infused. I think of this like an "exchange transfusion"--saline in, more dilute urine out.

Central Pontine Myelinolysis (CPM) also now called Osmotic Demyelinating Syndrome (ODS)

Demyelinating disease of pons and CNS
Flaccid paralysis, dysarthria, dysphagia, hypotension
Alcoholics, malnourished, severely ill
Rapid correction of hyponatremia is causative
May be seen 1 to 2 days after correction and after clinical improvement has occurred
Women and children may be at highest risk
Beware hypokalemia; replace K aggressively!

RATE OF CORRECTION:

There is some controversy in correction rates. The following is the best advice for severe hyponatremia (under 120 meq/L):

Correct hypotension as rapidly as possible with NSS.
Use hypertonic saline at 100cc/hr for AMS, seizure, coma
Supplement with lasix, ensure a negative fluid balance.
Correct Na level by maximum of 12 meq/day
Never aim for a near normal sodium of 130 on day 1

Acute symptomatic cases: Hypertonic saline and lasix (See below)
Raise Na by 2 meq/L/hr.

Chronic cases: If asymptomatic--water restriction, supplement with lasix.
If symptomatic--hypertonic saline and lasix.
Raise Na by 0.5 meq/L/hr once stable.

Lasix appears protective.
Maintain a negative water balance.

Protocol for Correcting Symptomatic Hyponatremia:

1) Start 3% saline at 100 cc/hour
2) Repeat SMA-7 in 2 hours
3) Correct to serum sodium by a maximum ↑ of 12 meq during each 24/hr period (try not to correct by more than 10 - 15 meq if possible)
4) Supplement with Iasix--start at 20 mg IV and follow urine output; increase lasix as needed to maintain a negative fluid balance. (more out than in)
5) Water restrict and take 48 - 72 hours to fully correct patient.

Rules for Using Hypertonic Saline (HSS)

- **HSS should be 3% Saline Solution**
- **Maximum infusion rate is 100cc/hr or 1/cc/kg/hr**
- **Maximum infusion duration is 2-3 hours**
- **Recheck Na levels at hours 2, 3, and 6**
- **Serum Na level must be below 120 for HSS *to* be *considered***
- **Serum Na level should usually be in the 100-110 meq range for HSS to be used**
- **Hyponatremia must be symptomatic in order to use HSS**
- **Symptomatic means Seizures, Coma, or Acute AMS**
- **1.2 cc/kg HSS will raise Serum Na by lmeq**
- **USE low dose lasix to block ADH release induced by HSS (ADH will block diuresis!)**

Important notes:

- Hypertonic saline at high flow rates may cause:
  - Seizures
  - Brain damage
  - Pulmonary edema
  - Hemolysis
- Lasix is a great drug in water excess.
- Correction to normal is malpractice.

Calculations and Formulas

Because (1) patients are usually only corrected to ⅓-½ of their estimated deficit in the first 24 hours; (2) are only first brought to a sodium level of 120 and (3) the SMA-7 is easy to get, I think these calculations are somewhat of an academic pursuit, and not usually clinically helpful.

Two formulas may be helpful in appreciating either the amount of Na in meq needed or the amount of excess total body water (TBW). As an example, assume a 70 kg patient has a serum Na of 120. TBW is approximately 60% of patient's body weight (42L for 70 kg patient).

meq of Na needed:

# of meq low x # of L in TBW
20 x 42 = 840

# of L of excess TBW:

Excess = Predicted - Desired

$$\frac{\text{pts Na}}{\text{normal Na}} \times \text{TB W} = \text{desired body water}$$

120/140 x 42 = 36L
therefore excess = TBW predicted - desired
= 42 - 36 = 6L

Hyponatremia due to excess Total Body Sodium and Excess Total Body Water (Also called Hypotonic Hyponatremia with Excess Sodium and TBW; or Hypervolemia with Edema)

Hyponatremia with increased total body sodium due to hypoperfusion of' kidneys causing high aldosterone secretion occurs in patients with heart (CHF), Kidney (CRF), or hepatic failure (LF).

Patients are usually asymptomatic or minimally symptomatic.

Saline and/or hypertonic saline causes pulmonary edema in these patients!!!

Treat with: (1) lasix, (2) water restriction, and (3) improve underlying pathology.

CHF: Digoxin, nitrates, ACE inhibitors, diuretics
CRF: Lasix, dialysis
LF: Albumin, lasix, mannitol, paracentesis

THE FIVE MOST COMMON ERRORS TO AVOID IN TREATING HYPONATREMIA

(1) DO NOT START NSS at 150cc/hr and Forget to Repeat SMA-7 in 2 hours

(2) DO NOT USE Hypertonic Saline unless Absolutely Indicated

(3) DO NOT Think of 130-140 as Normal in the Acute Correction of Severe Hyponatremia -- Reset Your Osmostat to 120-125!!!

(4) DO NOT Correct Faster Than 0.5 meq/hour

(5) DO NOT Treat the Value -- Treat the Patient

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HYPERNATREMIA

Overview

- Hypernatremia has the worst prognosis of any electrolyte abnormality
  (HyperK is acutely more deadly. but there is a lot more mild HyperK with a better prognosis)
- Hypernatremia always equals total body water deficit in the adult
- It is rarely seen in conscious awake patients
- Cellular dehydration usually causes AMS or deep coma
- Dehydration with hypotension supersedes serum sodium values when making therapeutic decisions.

NOTE:

Usually a disease of the elderly
Often due to AMS
Dramatically increases mortality for any coexisting disease
Rapid correction increases acute mortality

Correct Patients at a Rate of 0.5 meq/hr.

Never Change Serum Na Level by More Than 12 meq/day

There **are Three Types of Hypernatremia:**

(1) Hypernatremia due to **Dehydration** and **Low Total Body Sodium**
(2) Hypernatremia with **Low Total Body Water** and **Normal Total Body Sodium**
(3) Hypernatremia with **Normal Total Body Water** and **Increased Total Body Sodium**

HYPERNATREMIA WITH DEHYDRATION AND LOW TOTAL BODY SODIUM

Causes:

Heatstroke
Diarrhea
Osmotic Diuresis:
Glucose
Mannitol
Enteral feedings

Treatment:

- NSS until hemodynamically stable
- ½ NS at 100 cc/hr once patient is stable
- Lower Na at 0.5 meq/hr. once total body water normalized

HYPERNATREMIA WITH LOW TOTAL BODY WATER AND NORMAL TOTAL BODY SODIUM

Diabetes Insipidus
Elderly with "reset" osmostat
Hypothalamic dysfunction

Treatment:

- ½ NS or encourage fluid intake
- Corrects underlying etiolery if possible

HYPERNATREMIA WITH INCREASED TOTAL BODY SODIUM

Usually iatrogenic:

Salt tablets
NSS infusion
IV Sodium bicarbonate
Feeding Formula Error

Occasionally endocrine:

Cushing's syndrome
Conn's syndrome

Treatment:

- Water PO at high rate
- D_5W or ½ NS and PO fluids
- Dialysis if CRF

FORMULA FOR CALCULATING TBW DEFICIT

For calculating total body water deficit the following formula can be used:
NOTE: *Formula's are nice but only repeat serum sodium's are going to define therapy!!*

Step 1: How many meq elevation is there?
Step 2: What % elevation above 140 is this?
Step 3 : Multiply % elevation times total body water (TBW)

(TBW is 50% of ideal body weight in elderly men and women)

i.e., 70 kg patient; Na of 160: excess Na = 20, TBW = 42:

$$\frac{20}{140} \times 42 = 6L \text{ of body water needed}$$

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HYPERCALCEMIA

Calcium is measured as either (1) *serum calcium* or (2) *ionized calcium.* (Ionized is the active form)

Serum calcium: (three forms):

I. Ionized (50% of total)
2. Protein bound (40%)
3. Chelated (I 0%)

. Only ionized calcium is active form
. for *hypercalcemia* total is all that is required
. for diagnosis for *hypocalcemia*, ionized is much more accurate.

Hypercalcemia may be due to a large number of causes: The most common causes of hypercalcemia seen in the ED are:

MOST COMMON ED CAUSES OF HYPERCALCEMIA

1. **Malignancy** (up to 1/5 CA patients; breast is #1)
2. **Drugs** (Thiazides, calcium, estrogens, lithium, Vit. A & D)
3. **Hyperparathyroidism** (Simple adenoma in 3/4 of patients)
4. **Iatrogenic** (Overly aggressive replacement therapy in rhabdo, ETOH etc.)
5. **Infections** (AIDS, sarcoid, TB, granulomatous diseases)

MOST COMMON CAUSES OF HYPERCALCEMIA

MALIGNANCY
- Direct bony destruction
- Ectopic PTH
- Ectopic PTH-like substances
- Osteoclast activating factor
- Prostaglandins
- Cytokines

Endocrine
- Hyperparathyroidism (1º and 2")
- Multiple endocrine neoplasias
- Hyperthyroidism
- Pheochromocytoma
- Myxedema, acromegaly, adrenal insufficiency

Granulomatous Diseases
- Sarcoid
- Tuberculosis
- Histoplasmosis, Coccidioidomycosis, Berylliosis

Pharmacologic Agents
- Calcium supplements
- Vitamin D intoxication
- Vitamin A intoxication
- Thiazide diurectics
- Estrogens
- Milk-alkali syndrome
- Lithium

Renal Diseases
- Pseudohyperparathyroidism
- Post renal transplant

Miscellaneous
- Dehydration
- Prolonged immobilization
- Iatrogenic
- Hyperproteinemia
- Rhabdomyolysis
- Idiopathic infantile hypercal
- Familial
- Laboratory error

MOST COMMON TUMORS CAUSING HYPERCALCEMIA

- Breast
- Lung
- Hematologic (Lymphoma, Myeloma, Leukemia)
- Kidney
- Prostate

Malignancy may cause hypercalcemia by a number of mechanisms:

PTH-like hormonal substances
Bone destruction from metastasis
Osteoclast activating factor
CA mediated increases in 1,25 dihydroxy vitamin D
1° hyperparathyroidism

Asymptomatic Patient:

DX:

In patients who are asymptomatic and have an isolated elevated calcium of: 10.5 - 12.5 mgidl: think of the following:

- Patients on thiazides or other calcium-elevating medication
- Sample drawn post-meal may raise Ca by 1 - 2 mg/dl
- Cancer or Pulmonary lesion
- Hyperparathyroidism

PLAN:

Ifpatient looks great you may:

1) get repeat fasting Ca in 24 - 48 hours and;
2) refer to private M.D.

If patient on *meds causing hypercalcemia:* refer to private M.D after discontinuing medication.

If patient looks *at all ill:* or patient and/or private M.D. wants work-up, the keys are:

1. Do breast exam
2. Do CXR (R/O CA, sarcoid, mass, fungal diseases)
3. Check routine lab: PO_4: Often low in hyperparathyroidism
   HCO_3^-: Often low in hyperparathyroidism
   Cl^-: Often elevated in hyperparathyroidism
4. Order PTH level if private M.D. requests (Expensive; usually not an ED test for a single elevated Ca).
5. Document follow-up plan and copy private MD.

Most common ED presenting signs and symptoms of Hypercalcemia

- **Lethargy, AMS, Coma, Seizure**
- **Nausea, Vomiting, Anorexia**
- **Fecal Impaction**
- **Abdominal pain due to**
  - **a) PUD**
  - **b) Pancreatitis**
  - **c) Stone**
- **Digitalis Toxic (PAT with block, junctional tach etc.)**

The classic ECG finding of a short Q - T interval is uncommon and only seen in 20-50% of patients.

The most common causes of death in hypercalccmia are due to complications caused by:

- Coma
- Dehydration
- Electrolyte disturbances
- Cardiac abnormalities

Electrolyte Disturbances Associated with Hypercalcemia

Hypokalemia: Common in both hyperparathyroidism and cancer
Hyperkalemia: Due to CRF
Hypematremia: Due to dehydration
Hypomagnesemia: Common whenever there is hypokalemia

Initial Acute Therapy of Hypercalcemia:

1. Secure ABC's (and consider NGT)
2. Saline
3. Lasix
4. Follow K and Mg
5. Call internist/private MD for his/her use of biphosphonates vs steroids vs. mithramycin vs indocin vs calcitonin.

The key to early therapy is restoring volume and increasing Ca excretion. Patients are often hypokalemic and hypomagnesemic (although if CRF is chronic and severe, they may be hyperkalemic and hypermagnesemic). As patient begins to improve, they may develop ventricular arrhythmias.

STEPWISE THERAPY OF HYPERCALCEMIA

SECURE ABCS: *(Consider NGT)*

Patients usually dehydrated, obtunded and predisposed to arrhythmias due to other electrolyte problems besides hypercalcemia. Some may be dehydrated& have narcotics on board.

SALINE: *Saline inhibits proximal reabsorption of Ca*

Start NSS wide open until BP &/or perfusion is normal;
continue NSS at 150 250 cc/hr depending on age and renal function
Follow cardio-pulmonary status.

LASIX: ***Lasix*** *inhibits* ***distal reabsorption*** *of* ***Ca***

Once volume status secure only!
40 mg I.V.
Repeat 20 40 mg q 2H to maintain I/O balance

FOLLOW K and Mg:

Get stat level q 2 hrs
- Give 20-40 meq/L of KCl once urine output is up
- Get Mg level daily
- Add 0.5 gm $MgSO_4$ (4 meq or 1 cc of 50% to each bag of saline)

CALL Internist/Oncologist

OTHER IN-HOSPITAL OR SPECIALIST CONSIDERATIONS

Biphosphonates:	Favored agents in malignancy
Steroids:	Excellent in sarcoid, myeloma, lymphoma
Calcitonin:	Usually excellent in CRF, CHF; takes 8 hours; fails 25% of time
Mithramycin:	For myeloma osteoclasts; takes 1 - 2 days
NSAIDs:	Prostaglandin mediated hypercalcemia

Biphosphonates

Chemical analogues ofpyrophosphate
Blocks bone absorption by adhering to surface of hydroxyapatite
Inhibits metabolic activity of osteoclasts
Two most common: Etidronate (Didrones) and Pamidronate (Aredia)
Dosing is over hrs to days
Effects in 24-48 hrs
Not an ED decision or administration

Life-Threatening Hypercalcemic Arrhythmias and Heart Block

(1) **Antiarrhythmics:**

2 - 5 mg of Verapamil over 1 2 minutes

(2) **Phosphates:** For life-threatening emergencies ONCE BP is normal:

Acute binding of Calcium
Also Blocks Vitamin D activation
Do not use routinely; may precipitate ARF;
Do not give if Ca x PO, is above 70.
50 mM (1.5 grams) over 6 - 8 hours
Take 6 - 8 hours to work
May lower calcium values from 1 - 5 mg/dl
EDTA works similarly without Ca/PO_4 precipitation

(3) **Dialysis:**

Life-saving in patients with CRF, Pulmonary Edema

Hypercalcemia References:

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HYPOPHOSPHATEMIA

Hypophosphatemia, like hypomagnesemia, often goes unrecognized. Although most patients remain asymptomatic. severe hypophosphatemia may result in potentially life-threatening complications.

Phosphate Formulas

Serum levels: 3. l mg/dl = l mmol/L = 3 l mg/L = l .8 meq/L
Solutions: I gram PO, = 30 mmol

1 cc K_2PO_4 = 4.4 meq K + 3 mmol of PO, = 93 mg PO,

ED Patients Most Likely To Have Hypophosphatemia

Alcohol withdrawal
Alcoholic ketoacidosis
Diabetic ketoacidosis
Malnourished COPD
Any chronic malnourished patient with acute hyperventilation

Most Common Causes Of Acute Hypophosphatcmia Due To Rapid Intracellular Shift

Hyperventilation
Glucose
Insulin
Volume
Resolving Acidosis

Table 1 - The Most Common Causes of Hypophosphatemia in the Emergency Department

1. Decreased Intake or Decreased Absorptive States:	Chronic alcoholism Home parenteral nutrition AIDS Chemotherapy Vomiting Malabsorption syndromes Secretory diarrhea Vitamin D deficiency
2. Hyperventilatory states:	Sepsis Alcohol withdrawal Salicylate poisoning Neuroleptic malignant syndrome Panic attacks Diabetic ketoacidosis Gout Hepatic coma
3. Hormonal and Endocrine effects:	Insulin loading Glucose loading Exogenous epinephrine Hyperparathyroidism
4. Medications:	Diuretics Chronic antacid ingestion Steroids Phosphate binders Xanthine derivatives Beta-2 agonists
5. Disease states:	Trauma Severe thermal burns Acute renal failure

Table 2 - Manifestations of Severe Hypophosphatemia

Central nervous system:	Irritability Confusion Parasthesias Depression Dysarthia Seizure Coma
Cardiac:	Cardiomyopathy Depressed myocardial contractility Arrhythmias
Respiratory:	Acute respiratory failure Depressed diaphragmatic contractility
Musculoskeletal:	Joint pain Myalgia Rhabdomyolysis Osteomalacia Generalized weakness Muscle edema
Endocrine:	Insulin resistance Hyperparathyroidism
Hematologic:	Decreased levels of 2,3-DPG and ATP Leukocyte dysfunction Hemolysis Platelet dysfunction
Renal:	Acute tubular necrosis Metabolic Acidosis Hypercalcemia

Rules and Guidelines
Therapy of Hypophosphatemia

- Treatment recommended as levels fall below I .5-2.0 mg/dl
- Treatment mandatory as levels fall below I .0 mg/dl
- Use only one type of IV phosphate solution and learn its contents
- Learn it in meq of K/ml and mgs/mmol of PO_4/ml
- I cc K_2 PO, = 4.4 meq of K

. 1 cc K_2PO_4/ml = 3 mmol PO, = 93 mg PO_4

usual replacement therapy = 1 gram/day - 30 mmol/day = 10 cc/day

usual maximal therapy = 3 grams/day - 90 mmol/day = 30 cc/day

Usual Dosing

In general, some helpful guidelines for routine replacement are:

give approximately 1/2 cc/hr of K_2 PO,, this equates to:

2 **cc's K_2 PO, in 1000 cc over 5 hrs at 200/hr**

or

4 **CC'S K_2 PO, in 1000 cc for Q8H IV fluids**

(1 cc = 4.4 meq K = 93 mg PO, = 3 mmol)

Table 3 - Recommendations for Intravenous Phosphate Repletion (based upon body weight of 70 Kg).

Author	Author's Specific Recommendation	Maximum Extrapolated 24 Hr Dose	Maximum Extrapolated Hourly Infusion of K_2PO_4
Clark (Crit Care Med 1995)	Up to **0.65** mmol/Kg/d	44.8 mmol	*
Hodgson (Endo Met Clin N Amer 1993)	24.4 mmol over 12 hours	69 mmol	0.90 cc/hr
Kingston (Crit Care Med 1985)	Up to 0.5 mmol/Kg over 4 hrs	*	2.73 cc/hr
Lentz (Ann Intern Med 1978)	Up to 0.24 mmol/Kg over 6 hrs	67.2 mmol	0.88 cc/hr
Loven (Acta Chir Scan 1983)	0.4 mmol/Kg/d	38 mmol	0.36 cc/hr
Peppers (Crit Care Clin 1991)	Up to 0.5 mmol/Kg	35 mmol	1.56 cc/hr
Rosen (Crit Care Med 1995)	15 mmol over 2 hrs	45 mmol	2.34 cc/hr
Rubin (Semin Nephrol 1990)	Up to 90 mmol/d	90 mmol	*
Wilson (Arch Intern Med 1982)	Up to 45 mmol over 10 hrs	*	1.41 cc/hr

*Unable to determine extrapolated dose based upon author's recommendation

ALCOHOLIC KETOACIDOSIS

. Not uncommon
- Develops in chronic ETOH abusers. especially hinge drinkers
- Five requirements:

Underlying state of malnutrition
Dehydration
Low glycogen stores
No available carbohydrate
A precipitating event

- Results in:

Protein and fat break down
Rising lactate and beta-hydroxy-butyrate levels
A low bicarbonate (5-15)
A low serum pH (7.10 7.35)

Treatment of Alcoholic Ketoacidosis

1. Hydrate and give Glucose
2. Treat withdrawal
3. Replete minerals and vitamins
4. Replete carbohydrates stores (Feed!)
5. Treat underlying cause: Pancreatitis, CNS events, sepsis, pneumonia, GI bleed, etc.)

AKA Cocktail

D_5NSS* at 200 cc/hr
1 amp MVI
l-5 mg Palate
40 meq/KCL
5 grams magnesium

*all alcoholics receiving glucose should receive 100 mg IV thiamine once IV has been started

Phosphorus in AKA

N o proven role however
Always give PO, if levels below 1.5-2.0
Use K_2PO_4

Phosphorus in AKA Cocktail

Give 1/4 I /2 of K as K_2PO_4
K_2PO_4 = 4.4 meq WCC = 93 mg PO, = 3 mmol/PO_4

For Mild Hypophosphatemia (below 2.0 mg/dl):
add 2 cc of K_2PO_4
(and decrease KCL to 30 meq)

For Severe Hypophosphatemia (below 1.0 mg/dl):
add 4cc of K_2PO_4
(and decrease KCL to 20 meq)

KEYS TO GOOD CARE

- There is no role for bicarbonate in AKA (unless the pH is below 7.0; R/O other causes of acidosis!).
- There is no role for insulin in AKA (even with hyperglycemia unless glucose above 500).
- Follow K, PO, and venous pH values.
- **If pH falls once treatment has begun, this is NOT just AKA!**

DIABETIC KETOACIDOSIS

There are five (5) key elements to the treatment of patients with DKA.

1. ***Volume***
2. ***Insulin***
3. ***Potassium***
4. ***Bicarbonate***
5. ***Phosphate***

PHOSPHATE THERAPY IN DKA

- DKA patients are initially hyperphosphoremic
- Hypophosphatemia often occurs as insulin and fluids begun
- As PO, goes intracellularly, serum PO, levels drop to 2 - 3 mg/dl
- Levels 1.5 - 1.O are not uncommon in malnourished patients

At PO, levels of 1.5 mg/dl and below, potential complications to a patient in DKA are:

1. Hypophosphatemic muscle weakness
2. Respiratory failure or relative lack of adequate hyperventilation
3. Decreased 2,3 DPG with poor 0_2 transport in poorly perfused patients
4. CHF
5. AMS

No study has shown significant benefit to routine PO, therapy in DKA. Risks of routine in treating patients in DKA include:

- Hyperphosphatemia
- Renal failure
- Hypocalcemia
- Hypomagnesemia

PO, Recommendations in DKA:

At present, routine phosphate is not indicated. In patients with malnutrition or significant hypophosphatemia, K_2PO_4 may be used at a dose of 1/4 of the K requirement given as K_2PO_4.

Each cc of K_2PO_4 = 4.4 meq KCL and 3 mmol of PO,

Put 2 cc's in each bag = 3 mmol/hr = 93 mg/hr at 500 cc/hr

Note: Do not use more than 60 mmol/day without reason!

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MAGNESIUM

One of the biggest problems in discussing magnesium is that Mg can be talked about in mg, meq, mmol, and gram quantities. I have listed the most important conversions below:

Key Magnesium Conversions:

1 gram $MgSO_4$ = 8.12 meq Mg

1 meq Mg = 0.5 mmol Mg = 12.3 mg Mg

1 meq/dl Mg = 1.2 mg/dl

1 gram $MgSO_4$ = 98 mg of elemental Mg

1 Mg oxide table = 111 mg Mg = 9 meq Mg

1 10cc Amp of 50% $MgSO_4$ = 5 grams of Mg or 40.6 meq Mg

DON'T DEPEND ONLY ON A TOTAL SERUM MAGNESIUM LEVEL --YOU WILL MISS CLINICALLY SIGNIFICANT HYPOMAGNESEMIA

Although there are many causes of hypomagnesemia, they can be subdivided into 5 causes:

5 Causes of Hypomagnesemia

Dietary
Gastrointestinal
Renal
Drug
Endocrine-Metabolic

*See table, page 41

Although there are a large number of patients who are at risk for this deficiency, only a few of these patients are likely to present to the ED. The 5 most common patient types likely to be clinically hypomagnesemic:

High Risk for Clinically Significant Hypomagnesemia

Patients on Diuretics
Alcoholics or Malnourished Patients
Patients with Hypokalemia
Patients with AMI
Patients with Ventricular Arrhythmias

There are many signs, symptoms and ECG changes reported to correlate with hypomagnesemia; however, it is unclear if any are reliable when the patient does not also have hypocalcemia, hypokalemia and/or hyponatremia.

Reported Signs and Symptoms Associated With Hypomagnesemia

Neuromuscular

Tremor, spasm, tetany
Confusion, psychosis, coma
Ataxia, nystagmus, seizures
Paraesthesias, weakness

Cardiovascular

ECG changes
CHF
Arrhythmias
Vasospasm
Hypertension
Digitalis and Quinidine sensitivity

Gastrointestinal

Nausea
Anorexia
Abdominal pain
Electrolyte disturbance
Hypokalemia
Hypocalcemia
Hypophosphatemia
Hyponatremia

Note: Hypomagnesemia may make hypokalemic and hypocalcemic ECG changes more pronounced. It probably causes no ECG changes itself.

THE MOST COMMON CAUSES OF HYPOMAGNESEMIA

Dietary

Alcoholism
Chronic Diseases
Hyperalimentation
Long term
  IV hydration

Gastro-Intestinal

Malabsorption
N/G Suction
Fistulas

Renal

Glomerulonephritis
Tubular Dysfunction
Interstitial Disease
ATN-Diuretic Phase
Secondary to
  Endocrine disease

Drug

Common:
  Diuretics
  Digitalis
  Aminoglycosides

Less Common:
  Amphotericin B
  Cis-Platinum
  Carbenicillin
  Thyroxin

Acute Change:
  Insulin
  Catecholamines
  Citrate
  Glucose
  Amino Acids

Endocrine-Metabolic

Acute Diseases:
  DKA
  AKA
  Burns
  Sepsis
  Bypass
  Hypothermia

Chronic:
  Pregnancy
  Lactation
  Hungary Bone
  Sweating
  Mg free dialysis
  Re-feeding s/p
    starvation

Endocrine:
  Hyperaldosteronism
  Hypercalcemia
  Hyperthyroidism
  SIADH
  Hypophosphatemia

DOSE OF MAGNESIUM SULFATE

The dose of $MgSO_4$ is determined by:

(1) Clinical setting
(2) Patient's serum level

for repletion: A constant infusion without a loading dose is essentially without complications--as long as the patient:
(1) does not have CRF
(2) is not already hypermagnesemic

for therapy: Patients are loaded over 0 - 60 minutes and then usually placed on a drip.

Standard loading dose of $MgSO_4$:
1 - 2 grams over 0 60 minutes

Standard Maintenance dose of $MgSO_4$:
1/2 - 1 gram/hour (4 - 8 meq/hr)

Serum levels and $MgSO_4$ Dosing:

Loading doses:
1 - 2 grams over 1 hour: serum levels usually normal
2 - 3 grams over 1 - 5 minutes: serum levels usually double

Maintenance doses:
4 - 8 meq/hr maintains serum level
16 meq/hour (2 grams) elevates and maintains serum level by about 1 mg/dl

LOAD WITH 1-2 GRAMS OVER 0-60 MINUTES

MAINTENANCE INFUSION IS 0.5 - 1 GRAM PER HOUR

MAGNESIUM AS AN ANTIARRHYTHMIC

Magnesium appears to be an effective antiarrhythmic for treating ventricular ectopy due to a number of causes:

Magnesium for Ventricular Arrhythmias:

AMI
Refractory VT
Refractory VF
Refractory Ventricular Arrhythmias
Torsades de Pointes
TCA OD
Digitalis OD
Quinidine Toxicity
Hypokalemic Arrhythmias
Prolonged Q-T Syndromes

Any ventricular arrhythmia not due to hypermagnesemia

Magnesium has multiple beneficial electrophysiologic effects. These include:

(1) Increasing resting membrane negativity (phase 4)

(2) Calcium channel blockade (phase 2)

(3) Direct myocardial stabilization (not yet elucidated)

Thus:

- Magnesium stimulates the Na-K ATPase pump to make the cell more negative
- Decreases the relative refractory period by speeding repolarization and by decreasing the duration of phase 2.
- Decreases ischemic and free radical induced myocardial injury

During AMI magnesium levels fall due to a number of factors including an intracellular shift of Mg. Hypomagnesemia with or without hypokalemia (potassium levels also may fall or may be low in AMI); predisposes to arrhythmias.

MAGNESIUM FOR SPECIFIC VENTRICULAR ARRHYTHMIAS

Magnesium for Torsades

Torsades de Pointes or "twisting of Points" Ventricular Tachycardia is being seen and recognized on an increasing basis. It has numerous etiologies but generally can be grouped into the causes of the prolonged Q-T syndromes listed below:

COMMON ETIOLOGIES OF PROLONGED Q-T INTERVAL

HEREDITARY

Romano- Ward
Jervell-Lange-Nielson

ELECTROLYTE DEFICIENCY

Hypokalemia
Hypomagnesemia
Hypocalcemia

DRUGS

Centrally Active	Cardiac
Cyclic Antidepressants	*Quinine, Quinidine, Disopyramide*
Phenothiazines	*Procainamide*
Lithium	*Encainide, Flecainide*
Terfenadine (Seldane)	*Amiodarone, Ibutalide*

STRUCTURAL

Cardiac (Ischemia, Contusion, Myocarditis)
Neurologic (Stroke, Bleed, Tumor)

MISCELLANEOUS ("The Hypos")

Hypothermia
Hypothyroidism
Panhypopituary
Bradycardia

A number of therapies have been recommended for Torsades:

Potential Treatment of Torsades:

Lidocaine
Isoproterenol
Beta Blockade
Overdrive pacing
Cardioversion
Defibrillation

Magnesium has a number of theoretical benefits in treating Torsades;

Magnesium for Torsades:

Reverses underlying etiology
Decreases Q-T interval
Non Arrhythmogenic
Rapidly effective
Successful in multiple series

Dose of Magnesium for Torsades:

Unstable Patient:
2 grams IV push
l-2 grams Q 1 minute up to a total of at least 5 grams

Stable Patient:
2 grams over l-5 minutes
2 grams per hour

REMEMBER:

Dose of Magnesium Sulfate:

A bolus dose of 0.3 meq/kg (about 2.5 grams) will double a serum Mg level.
A constant infusion of magnesium (4 - 8 meq/hr) will usually only maintain, but not raise a level

Treat the underlying etiology of Torsades!

The loading dose of magnesium is 1-2 grams over O-60 minutes **The constant infusion dose of magnesium is 1/2-1 gram per hour**

Magnesium for Torsades

Remember:

- Correct underlying etiology, i.e.. correct hypoMg, hypoK
- Use adjunctive care
  i.e. Bicarbonate for TCA OD
  Fab fragments for Digoxin OD
- Beware Hypokalemia magnesium drives K into cells
- Beware recurrence
- Overdrive pacing for refractory pattern
- Use pacing or isoproteronal for bradycardic patients

THE CURRENT ACLS TEXT RECOMMENDS MAGNESIUM AS THE DRUG OF CHOICE FOR TORSADES

CLINICAL EFFECTS OF HYPERMAGNESEMIA

Effect	***Mg** Level **(mg/dl)***
Decreased DTR's	*4-5*
Hypotension	*5-7*
Respiratory Insufficiency	*10*
Heart Block	*IO-15*
Respiratory Paralysis	*15+*
Cardiac Arrest	*15 - 24*

Magnesium References

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Electrolyte Emergencies
Questions and Cases

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Hyperkalemia

1) Name 5 most common causes of hyperkalemia:

_____
_____
_____
_____
_____

2) What are the ECG changes of hyperkalemia? Please list them in order and estimate the K level associated with them.

	ECG Change	Approximate K Level
A)	_____	_____
B)	_____	_____
C)	_____	_____

3) What is calcium's effect on serum potassium?

4) How many drugs can be used to drive K into the cell?

- _____
- _____
- _____
- _____
- _____

5) A patient presents with life-threatening hyperkalemia. Describe a three part organized approach:

1)

2)

3)

Hypokalemia

6) What are the two most common causes of hypokalemia?

_____

_____

7) Name 5 potential ECG changes or rhythm disturbances that are attributed to hypokalemia.

-
-
-
-
-

8) Severe or refractory hypokalemia equals _____

9) What is the fastest way you can give IV KCL safely?

10) A malnourished child with diarrhea has a K of 2.1. How can you 1) keep her pain free and 2) keep her small IV line patent while you give high quantities of KCL?

Hyponatremia

11) A patient has a glucose of 1000mg and a sodium of 120. Is she Hyponatremic?

12) What are the 3 main causes of SIADH?

13) How quickly can you safely raise someone's serum sodium?

14) What electrolyte (other than sodium) predicts a risk for CPM?

15) Hypertonic Saline

a. Indications _____
_____
_____

b. In order to use, Serum Sodium is usually in what range - _____
c. What concentration?
d. What rate?
e. For how long?
f. With Lasix?

Hypercalcemia

16) What are the most common symptoms associated with hypercalcemia.

-
-
-
-
-

17) What are the most common causes of:

1) asymptomatic hypercalcemia

(2) symptomatic hypercalcemia in the ED

(3) significant hypercalcemia - asymptomatic

18) What is the simple best ED therapy for symptomatic hypercalcemia.

19) What is a 5 step approach to symptomatic hypercalcemia in the ED?

1.
2.
3.
4.
5.

20) What is the biggest ED error on volume therapy in hypercalcemia?

21) When should you start a biphosphonate?

Hypophosphatemia

22) What one rate of administration of K_2PO_4 makes you an expert?

23) When is PO, indicated - what value?

Hypomagnesemia

24) Name the 5 causes of a prolonged Q-T interval

-
-
-
-
-

25) What dose of $MgSO_4$ makes you an expert?