EMERGENCY MEDICINE

BOOT CAMP

ENDOCRINE AND ELECTROLYTE DISORDERS

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Professor

FORMULAS YOU GOTTA KNOW

- **Anion Gap = | Na+ - (Cl- + HCO3-) |**
- **Osmol Gap = (2 x Na+) + Glucose/18 + BUN/2.8**
- Winter's Formula = 1.5 x HCO3 + 8 = expected CO2
- Corrected calcium
  - [Ca] + (4 - [albumin])(0.8)
- Corrected sodium (if Glc > 200 mg/dL)
  - for every 100 mg/dL of Glc, add 1.6 to Na
- Free water
  - (0.6)(body weight)[([Na]/145)-145]

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WHAT IS THE ANION GAP FOR THE FOLLOWING PATIENT? NA = 130; K = 3; CL = 80; HCO3 = 10; BUN = 35; CREAT = 2; GLC = 240

- a. 40
- b. 60
- c. 15
- d. 35

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WHAT IS THE SERUM OSMOLALITY FOR THE FOLLOWING PATIENT? NA = 130; K = 3; CL = 80; HCO3 = 10; BUN = 28; CREAT = 2; GLC = 360.

- a. 230
- b. 290
- c. 310
- d. 320

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Table 2. The Differential Diagnosis Of A Wide-Gap Metabolic Acidosis: The "MUDPILES" Mnemonic.

M	Methanol
U	Uremia
D	Diabetic ketoacidosis
P	Phenformin (metformin)
I	Iron, isoniazid
L	Lactic acidosis (shock, cellular toxins such as cyanide or carbon monoxide)
E	Ethylene glycol, ethanol (alcoholic ketoacidosis)
S	Salicylates

WHICH OF THE FOLLOWING IS NOT A CASE OF HIGH ANION GAP ACIDOSIS?

- a. DKA
- b. Uremia
- c. Iron
- d. Acetazolamide
- e. Salycylates

CASE

- 13yo male presents with nausea, vomiting, and diffuse abdominal pain. Has been having cold for the past week and started having symptoms this morning. Hx/o noncompliance with his medications.

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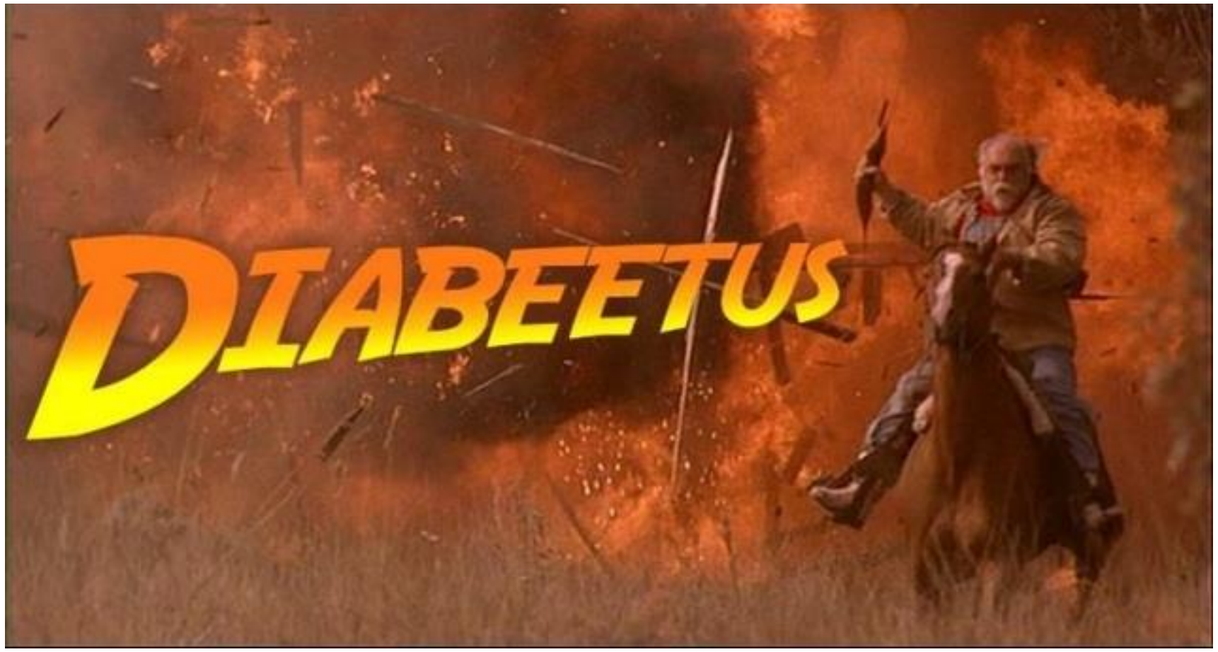
UA Ketones +++

Sodium 130

POCT 460



ABG pH 7.05 HCO2 5 CO2 18

K+ 3.8

Anion Gap 18

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DIABETES TYPE I

- Accounts 5-10% of all DM
- 50% presents in childhood
- Pancreatic ß- cell destruction results in insulin deficiency
- 5% mortality
- Often associated with other autoimmune disorders: thyroid's, Addison's, celiac's, vetiligio

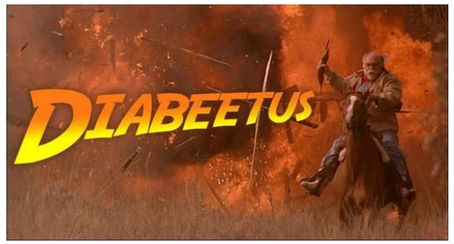
DIABETES MELLITUS

- Type I Diabetes Mellitus
  - Also called juvenile or insulin-dependent diabetes mellitus (IDDM).
  - Characterized by low production of insulin.
    - Closely related to heredity.
  - Results in pronounced hyperglycemia.
    - Symptoms of untreated Type I DM include polydipsia, polyuria, polyphagia, weight loss, and weakness.
    - Untreated or noncompliant patients may progress to ketosis and diabetic ketoacidosis.

DIABETES MELLITUS

- Type II Diabetes Mellitus
  - Also called adult-onset or non-insulin-dependent diabetes mellitus (NIDDM).
  - Results from decreased binding of insulin to cells.
    - Related to heredity and obesity.
    - Accounts for 90% of all diagnosed diabetes patients.
    - Less risk of fat-based metabolism.
  - Results in less-pronounced hyperglycemia.
    - Hyperglycemic hyperosmolar nonketotic acidosis.
    - Managed with dietary changes and oral drugs to stimulate insulin production and increase receptor effectiveness.

DKA



- Insulin deficiency = Hyperglycemia = Glucose urine dump = Osmotic diuresis = DEHYDRATION
- Ketones from lipolysis
- Causes: noncompliance, infx, MI, surgery, stress
- Polydipsia, Polyuria, Nausea/Vomiting, diffuse abd pain
- Anion-gap metabolic acidosis - respiratory compensation (**KUSSMAUL BREATHING**)
- Glucose elevated, but not crazy (<600), ketones

The most common cause of diabetic ketoacidosis in a known diabetic (other than noncompliance) is:

a. Renal disease

b. Infection

c. Alcohol ingestion

d. Increased glucose intake

e. Dehydration

DIABETIC KETOACIDOSIS

- Pathophysiology
  - Causes:
    - Not taking insulin (or new diagnosis)
    - Infection or illness (50%)
    - AMI
    - Pump not working
    - CVA
    - Trauma
    - Pregnancy
    - Hyperthyroidism
    - Pancreatitis
    - Emotional
    - Alcohol

High mortality

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DIABETIC KETOACIDOSIS

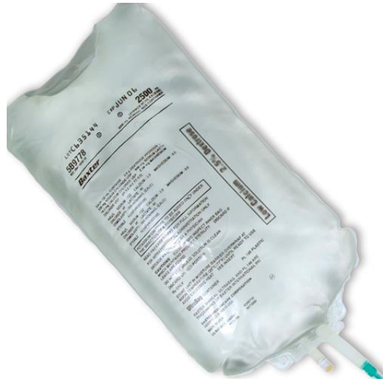
- Findings
  - Potassium-related cardiac dysrhythmias.
  - Kussmaul's respiration 2ry to metabolic acidosis.
  - Decline in mental status and coma.
  - Metabolic acidosis with serum ketosis and dehydration
  - Correct sodium

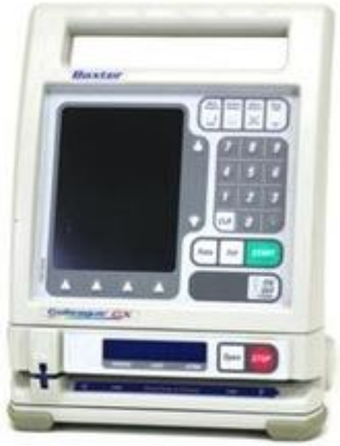
DKA

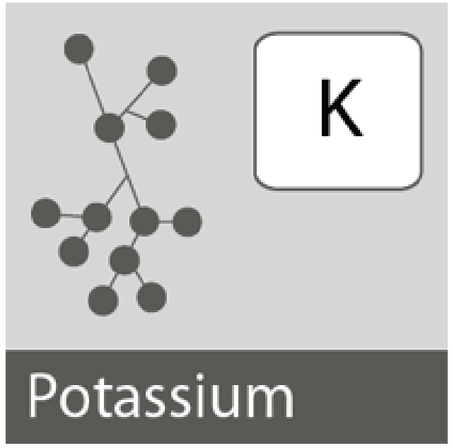
- Electrolyte changes
  - Glucose
    - Higher concentrations if dehydrated
  - Metabolic acidosis
    - Increased anion gap
    - Ketone production
      - Acetone, acetoacetate and BHBT (ß-hydroxybutyrate)
  - Serum sodium
    - Decreased due to fluid movement into intravascular space
    - Formula?
      - if Glc > 200 mg/dL, for every 100 mg/dL of Glc, add 1.6 to Na

DKA

- Electrolyte changes
  - Serum potassium
    - It moves from intracellular to extracellular space
    - It drops rapidly with insulin treatment
  - Serum phosphate
    - Concentration is normal but decreased with insulin treatment







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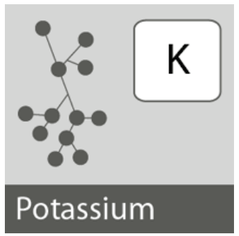
DKA TREATMENT



- FLUIDS! May require up to 6-8 L NS
- INSULIN: **BOLUS or not** = 0.1units/kg; **DRIP** = 0.1units/kg/hr. Add glucose to NS when glucose < 250
- POTASSIUM: Replace early, replace even if lab value is **NORMAL**
- No bicarb unless pH <7
- Phosphate/Magnesium not needed acutely

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A 30-YEAR-OLD MAN WITH TYPE 1 DIABETES PRESENTS TO THE EMERGENCY DEPARTMENT (ED). HIS BLOOD PRESSURE (BP) IS 100/70 MM HG AND HEART RATE (HR) IS 140 BEATS PER MINUTE. HIS BLOOD GLUCOSE IS 750 MG/DL, POTASSIUM LEVEL IS 5.9 MEQ/L, BICARBONATE IS 5 MEQ/L, AND ARTERIAL PH 7.1. HIS URINE IS POSITIVE FOR KETONES. WHICH OF THE FOLLOWING IS THE BEST INITIAL THERAPY FOR THIS PATIENT? (100 KG)

- a. Give normal saline as a 2-L bolus then administer 20 U of regular insulin subcutaneously.
- b. Bolus 2 ampules of bicarbonate and administer 10 U of insulin intravenously.
- c. Give him 5 mg of metoprolol to slow down his heart, start intravenous (IV) hydration, and then give 10 U of regular insulin intravenously.
- d. Give normal saline in 2-L bolus and then administer 10 U of insulin intravenously followed by an insulin drip and continued hydration.
- e. Give normal saline in 2-L bolus with 20 mEq/L KCl in each bag.

COMPLICATIONS

- Lethargy / Headache / Worsening nausea/vomiting = **CEREBRAL EDEMA**
- Common cause of DKA death. Can predict with elevated BUN.
- Tx with mannitol if concerned
- DKA w/ **cardiac arrest? HYPOKALEMIA!**

DKA TREATMENT IN CHILDREN

- 2 large bore IV's
  - One with NS
- Initial bolus of **10**-20 mL/kg if severe dehydrated
  - If hypotensive
  - If need to repeat, use 10 mL/kg
- If BP is normal, 7 mL/kg in one hour followed with 3.5-5 mL/kg/hr
- Glucose may falls with fluid hydration alone

DKA TREATMENT IN CHILDREN

- Insulin
  - DO NOT GIVE BOLUS
  - Start infusion at 0.1 U/kg/hr
  - Add D5 to IV fluids when glucose is <200 as well as decreased to half the drip
- Electrolytes
  - Add 40 mEq/L of KCl to IVF's when there is urine output and K < 5.5
- Bicarbonate
  - Routine not recommended
    - May increased hepatic ketone production
    - May increased risk of hypokalemia
    - May lead to paradoxical acidosis of CSF due to decreased respiratory drive and rise of partial pressure of CO2
    - May increase risk of cerebral edema
  - If pH<7, may need to intubate; give bicarbonate 1-2 mEq/kg over one hour
- Monitoring

CASE

- 80yo male presents with altered mental status. Lives at home by himself, has never seen a physician. Found by family members to be lethargic. No focal deficits. Dehydrated on exam. POCT 1100 by EMS.

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Serum osmolality HIGH

pH normal

Glucose 1123

TSH normal

Urine NO ketones, 3+ glucose

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HYPEROSMOLAR NON-KETOTIC COMA

- Hyperglycemia/hyperosmolality **WITHOUT acidosis** (**glucose > 1000**)
- Higher levels of insulin compared to DKA
- Usually **don't know they have type II DM**
- Can lead to DKA though
- Causes: INFX, MI, stroke, head injury
- Tx: SAME AS DKA (lots of fluid, drip, K+)

DKA VS HNKC

Elderly	HNKC
Glucose > 1000	HNKC
Acidosis	DKA
Potassium treatment	BOTH
Kussmaul breathing	DKA

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A 74-YEAR-OLD WOMAN WHO IS A KNOWN DIABETIC IS BROUGHT TO THE ED BY EMERGENCY MEDICAL SERVICE (EMS) WITH ALTERED MENTAL STATUS. THE HOME HEALTH AIDE STATES THAT THE PATIENT RAN OUT OF HER MEDICATIONS 4 DAYS AGO. HER BP IS 130/85 MM HG, HR IS 110 BEATS PER MINUTE, TEMPERATURE IS 99.8°F, AND HER RR IS 18 BREATHS PER MINUTE. ON EXAMINATION, SHE CANNOT FOLLOW COMMANDS BUT RESPONDS TO STIMULI. LABORATORY RESULTS REVEAL WHITE BLOOD CELL (WBC) COUNT OF 14,000/L, HEMATOCRIT 49%, PLATELETS 325/L, SODIUM 128 MEQ/L, POTASSIUM 3.0 MEQ/L, CHLORIDE 95 MEQ/L, BICARBONATE 22 MEQ/L, BLOOD UREA NITROGEN (BUN) 40 MG/DL, CREATININE 1.8 MG/DL, AND GLUCOSE 850 MG/DL. URINALYSIS SHOWS 3+ GLUCOSE, 1+ PROTEIN, NO BLOOD OR KETONES. AFTER ADDRESSING THE ABCS, WHICH OF THE FOLLOWING IS THE MOST APPROPRIATE NEXT STEP IN MANAGEMENT?

- a. Begin fluid resuscitation with a 2- to 3-L bolus of normal saline then administer 10 U of regular insulin intravenously.
- b. Begin fluid resuscitation with a 2- to 3-L bolus of normal saline then administer 10 U of regular insulin intravenously, begin phenytoin for seizure prophylaxis.
- c. Administer 10 U of regular insulin intravenously then begin fluid resuscitation with a 2- to 3-L bolus of normal saline.
- d. Order a computed tomographic (CT) scan of the brain, if negative for acute stroke, begin fluid resuscitation with a 2- to 3-L bolus of normal saline.
- e. Arrange for urgent hemodialysis.

Glucose LOW

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HYPOGLYCEMIA

- **MCC insulin**. Others oral hypoglycemic agents (SULFONYLUREAS, not metformin)
- Post-prandial hypoglycemia = DM
- Other: liver dx, starvation, renal failure
- Somogyi effect
  - It happens when the body's defenses respond to long periods of low blood sugar. This can occur when a person exercises a lot, goes a long time without a snack, or takes more insulin before bed than they need.
- Dawn effect
  - The dawn effect involves a rise in early morning blood sugar levels. This results from declining levels of insulin and an increase in growth hormones
- Tx: D50, glucagon, complex carbs
- Octreotide for oral agents OD
- Admission for oral hypoglycemics

BAL 0

POCT Glucose 160

ABG pH 7.05 HCO2 5 CO2 18

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ALCOHOLIC KETOACIDOSIS

- Abrupt alcohol cessation and decreased food intake
- Abdominal pain, nausea/vomiting
- Anion-gap metabolic acidosis
- BAL negative, glucose unremarkable
- Tx: **IV fluids + glucose** = D5NS
- Give **thiamine** 100mg to prevent Wernicke's encephalopathy (glucose metabolism uses thiamine)

- All of the following are true in alcoholic ketoacidosis except:

a. Large doses of insulin are usually required to correct hyperglycemia

b. The serum glucose is usually less than 200 mg/dL

c. The patient may have a normal blood pH

d. Bicarbonate is usually not needed

e. Serum ketones are elevated

If
Anion Gap Metabolic Acidosis
due to
Lactic Acidosis

=

Treat Underlying Disease

LACTIC ACIDOSIS

- Type A = Decreased tissue perfusion = **SHOCK**
- Type B1 = Medical disorders (liver, renal, lymphoma, etc)
- Type B2 = Drugs/Toxins (**metformin**, ethanol (MCC), etc)
- Type B3 = inborn errors

pH 7.53

HCO3 1000

CO2 8

Anion Gap 6


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RESPIRATORY OR METABOLIC?

 CO2 **Respiratory**

 CO2 **Metabolic**

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JUST A REVIEW!

- Metabolic acidosis
  - PaCO2 = last two numbers in pH (if pH > 7)
  - PaCO2 = 1.5 [HCO3] + 8 (± 2)
  - normal anion gap/hyperchloremic
    - occurs from a loss of HCO3
    - hypokalemic (renal losses, GI losses, RTA 1)
    - normokalemic/hyperkalemic (decreased aldosterone, RTA 2, RTA 4)
  - high anion gap
    - CAT MUDPILES

HIGH ANION GAP METABOLIC ACIDOSIS

CAT MUDPILES

- CN, CO
- AKA
- Toluene, theophylline,
- Methanol, metformin, MetHgb
- Uremia
- DKA, starvation ketoacidosis
- Paraldehyde, phenformin, propylene glycol
- INH, ibuprofen (high doses), Iron
- Lactic acidosis D (blind GI loops) and L (consider metformin)
- Ethylene glycol
- Salicylates, strychnine

HIGH ANION GAP METABOLIC ACIDOSIS
CAT MUDPILES

- CN/CO – causes lactic acidosis
- Alcohol (AKA) – causes high osmolar gap
- Toluene/theophylline
- Methanol – causes high osmolar gap; wood alcohol; becomes formaldehyde and formic acid (blindeness); metformin – causes severe lactic acidosis → formate
- Uremia – causes by H+ retention and from other organic acids
- DKA/starvation KA – causes ketoacidosis and lactic acidosis
  - How to manage ventilator?

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HIGH ANION GAP METABOLIC ACIDOSIS

CAT MUDPILES

- Paraldehydes – old medication
- Iron/INH - – cause lactic acidosis
- Lactic acid – type A (anaerobic) caused by tissue hypoxia and type B (aerobic) with no evidence of hypoxia
- Ethylene glycol – cause high osmolar gap; antifreeze; urine will fluoresce; kidney failure

glycolate → oxylate

- Salicylates - – causes lactic acidosis; first cause respiratory alkalosis and then, metabolic acidosis

NON ANION GAP METABOLIC ACIDOSIS

- Hyperchloremic metabolic acidosis
- Caused by
  - Loss of HCO3
    - GI: diarrhea, ureterosigmoidostomy
    - Renal: proximal RTA (type 2), ARF
    - Meds: acetazolamide, cholestyramine
  - Failure to excrete H+
    - Renal: obstructive uropathy, pyelonephritis, distal RTA (type 1 and 4)
    - GI: hyperalimentation
    - Meds: NH4Cl
    - Hypoaldosteronism
- The loss of HCO3 is compensated with the gain of a Cl-
  - Causing a normal gap

HIGH OSMOLAR GAP (IF >50, MOST LIKELY TOXIC ALCOHOL INDUCED)

- Hyponatremia
- HHNK coma
- Isopropanolol
- N-propanolol
- Propylene glycol
- Ethylene glycol*
- Methanol*
- Formaldehyde*
- Paraldehyde*
- Mannitol
- Diethyl ether ingestion
- Lithium overdose
  - *Associated with high anion gap metabolic acidosis

METABOLIC ALKALOSIS

- If plasma HCO3 >45, seek to lower it to <40
- Causes:
  - Chloride losing conditions result in hypovolemia and renal bicarbonate generation
    - GI: NGT suction/vomiting, diarrhea, cystic fibrosis, enteropathy
    - Medications: diuretics
  - Increased mineralocorticoid activity enhance renal bicarbonate generation
    - Normovolemic or hypervolemic
    - Endo: Conns, Cushings, Bartter
    - Renal artery stenosis, renin-secreting tumors, adrenal hyperplasia, hyperaldosteronism, Cushing syndrome, Liddle syndrome
    - Exogenous mineralcorticoids: licorice, fludrocortisone
  - Compensatory reduction in alveolar ventilation
    - Posthypercabnia – if chronic

RESPIRATORY ACIDOSIS

- Acute
  - HCO3 increases 1 mq/L for every 10 mmHg increase in pCO2
  - pH drops 0.08 for every 10 mmHg increase of pCO2
- Chronic
  - HCO3 increases 4 mEq/L for every 10 mmHg in pCO2

RESPIRATORY ALKALOSIS

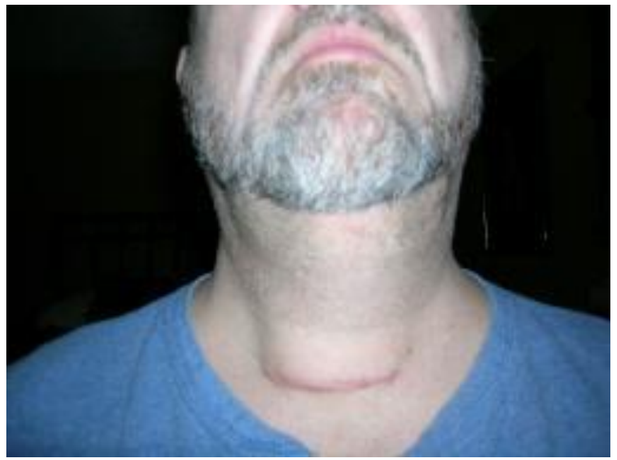
- Increased minute ventilation
- Acute
  - HCO3 drops 1 to 3.5 meq/L for every 10 mmHg drop in pCO2
  - Limit of compensation : bicarbonate is rarely below 18 meq/L
- Chronic (renal compensation starts within 6 hours and is usually at a steady state by 1.5 to 2 days)
  - HCO3 drops 2 to 5 meq/L for every 10 mmHg drop in pCO2
  - Limit of compensation : bicarbonate is rarely below 12 to 14 meq/L

OTHER CAUSES OF ACID/BASE DISORDERS

- Non-anion gap metabolic acidosis = **GI or renal losses** (diarrhea, renal tubular acidosis)
- Metabolic alkalosis = **vomiting**, NG, diuretics - low chloride = Tx with NS. Cushings - low potassium (urine chloride >10-20) = Tx potassium, not NS
- Respiratory acidosis = inadequate ventilation
- Respiratory alkalosis = hyperventilation/anxiety, PE, hypoxic

Pulse Ox 90% on RA

HR 45 BP 85/60



Hypothermic

Peripheral edema

Puffy eyes

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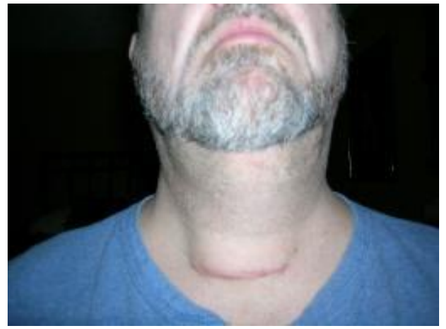
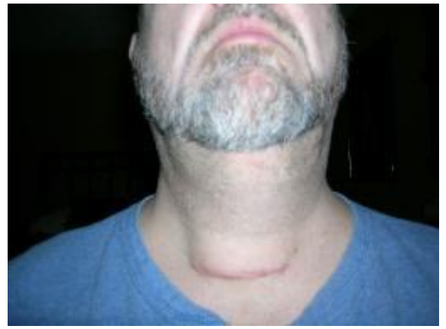
MYXEDEMA COMA



- Worsening severe hypothyroidism
- Hypothyroidism causes: **Hashimoto's thyroiditis** - autoimmune, prior tx of hyperthyroidism, iodine deficiency
- Drugs: lithium, amiodarone, sulfonamides
- Stress worsens hypothyroidism: **INFX** (pneumonia MCC)
- Hypoxemia / hypercapnia / **hypothermia** / bradycardia / puffy eyes / non-pitting edema / QT prolongation

MYXEDEMA COMA



- **Hyponatremia** / elevated CPK / cholesterol
- Tx: Supportive; +/- steroids
- Tx: thyroid replacement - **IV thyroxine**
- Hydrocortisone 100mg q8h for adrenal failure
- Active rewarming can cause hypotension

A 65-YEAR-OLD WOMAN IS BROUGHT INTO THE ED BY HER FAMILY WHO STATES THAT SHE HAS BEEN WEAK, LETHARGIC, AND SAYING "CRAZY THINGS" OVER THE LAST 2 DAYS. HER FAMILY ALSO STATES THAT HER MEDICAL HISTORY IS SIGNIFICANT ONLY FOR A DISEASE OF HER THYROID. HER BP IS 120/90 MM HG, HR IS 51 BEATS PER MINUTE, TEMPERATURE IS 94°F RECTALLY, AND HER RR IS 12 BREATHS PER MINUTE. ON EXAMINATION, THE PATIENT IS OVERWEIGHT, HER SKIN IS DRY, AND YOU NOTICE PERIORBITAL NONPITTING EDEMA. ON NEUROLOGIC EXAMINATION, THE PATIENT DOES NOT RESPOND TO STIMULATION.
WHICH OF THE FOLLOWING IS THE MOST LIKELY DIAGNOSIS?

- a. Apathetic thyrotoxicosis
- b. Myxedema coma
- c. Graves disease
- d. Acute stroke
- e. Schizophrenia

- Clinical characteristics of myxedema coma include all of the following except:
- a. Pseudomyotonic reflexes
- b. Hypothermia
- c. Hypotension
- d. Alopecia
- e. Tachycardia

CASE

- 58yo female presents for medical clearance for psych evaluation. Found in the streets naked yelling to strangers that the end is near. Brought in by EMS in restraints.

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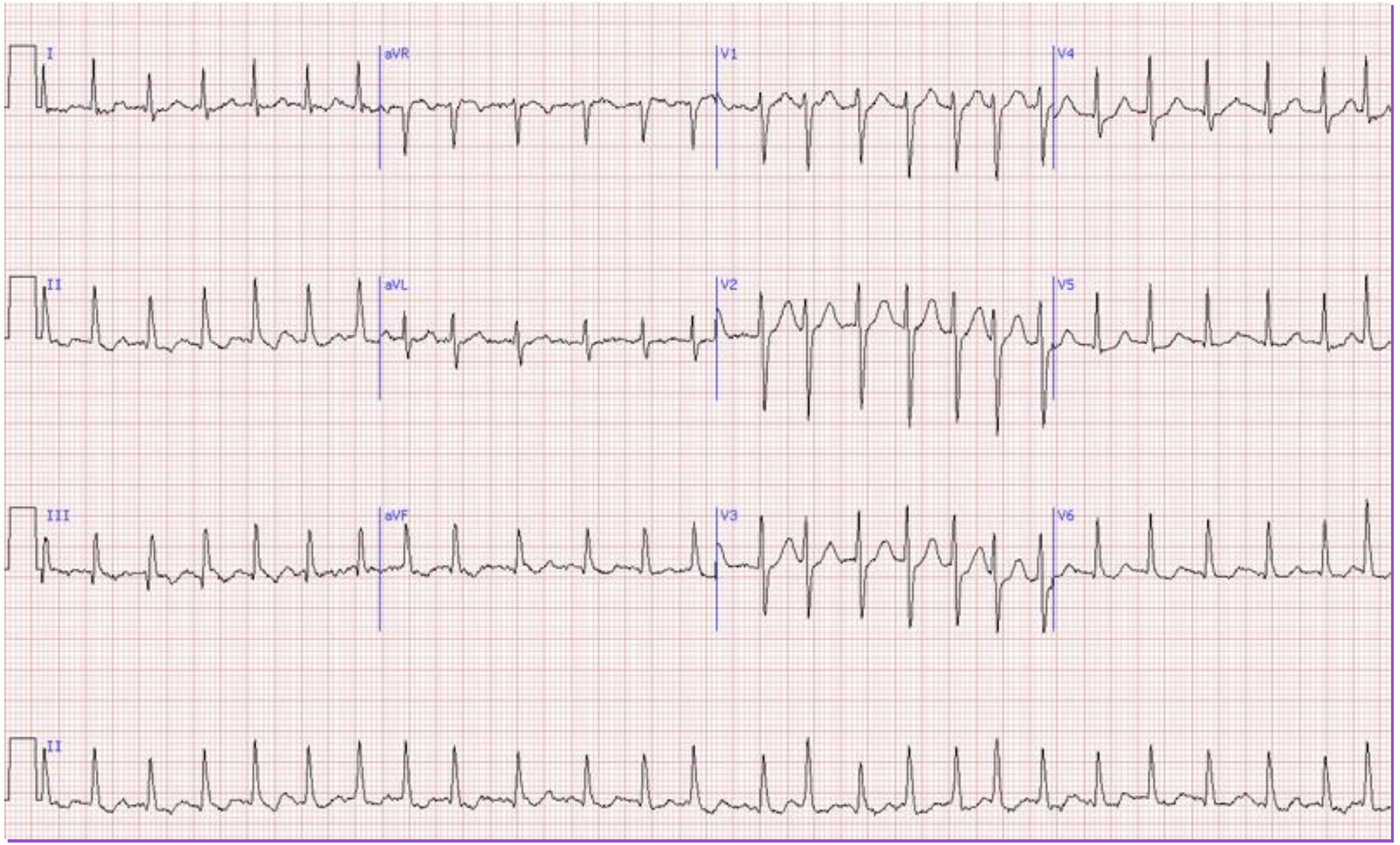

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Temp 104F

HR 145

BP 156 / 60

Delirium

Denies any drug abuse

Hx/o Grave’s dx

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THYROID STORM

Beta Blockers (propranolol)

PTU

1 hr

Iodide

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THYROID STORM

- **Graves Dx** (anti-TSH receptor antibodies) with stress event (**INFX**, radiocontrast agent, stroke, PE, radioactive iodide)
- Fever, AMS, psychiatric exam, tachycardia
- Unstable tx: **Beta-blockers** (Propanolol - C/I with asthma, CHF, COPD)
- **PTU, then IODIDE** (inhibits STORAGE release). Consider steroids
- Inhibits peripheral conversion: propanolol, PTU, **dexamethasone**. Don't give aspirin, sedatives
- **Apathetic thyrotoxicosis**: elderly hyperthyroidism vitally stable. Depression, lethargy, excessive weight loss. Afib/CHF

A 39-YEAR-OLD WOMAN IS BROUGHT INTO THE ED BY HER FAMILY WHO STATES THAT SHE HAS HAD 4 DAYS OF DIARRHEA AND HAS NOW STARTED ACTING "CRAZY" WITH MOOD SWINGS AND CONFUSION. THE FAMILY STATES THAT SHE USUALLY TAKES A MEDICATION FOR A PROBLEM WITH HER NECK. HER BP IS 130/45 MM HG, HR IS 140 BEATS PER MINUTE, TEMPERATURE IS 101.5°F, AND HER RESPIRATORY RATE (RR) IS 22 BREATHS PER MINUTE. AN ELECTROCARDIOGRAM (ECG) REVEALS ATRIAL FIBRILLATION WITH A NORMAL QRS COMPLEX. AFTER YOU ADDRESS THE AIRWAY, BREATHING, AND CIRCULATION (ABCS), WHICH OF THE FOLLOWING IS THE MOST APPROPRIATE NEXT STEP IN MANAGEMENT?

- a. Administer 2 ampules of bicarbonate to treat for tricyclic antidepressant overdose.
- b. Administer chlordiazepoxide, thiamine, and folate.
- c. Administer ceftriaxone and prepare for a lumbar puncture.
- d. Administer propranolol, propylthiouracil (PTU) then wait an hour to give Lugol iodine solution.
- e. Administer ciprofloxacin and give a 2-L bolus of normal saline for treatment of dehydration secondary to infectious diarrhea.

CASE

- 80yo male presents due to altered mental status. Found at home by family. Appears very ill. Vitals unstable. HR 145, BP 64/40, afebrile. Recently discharged from hospital 2 weeks ago for COPD exacerbation. Unable to fill medications in the past week.

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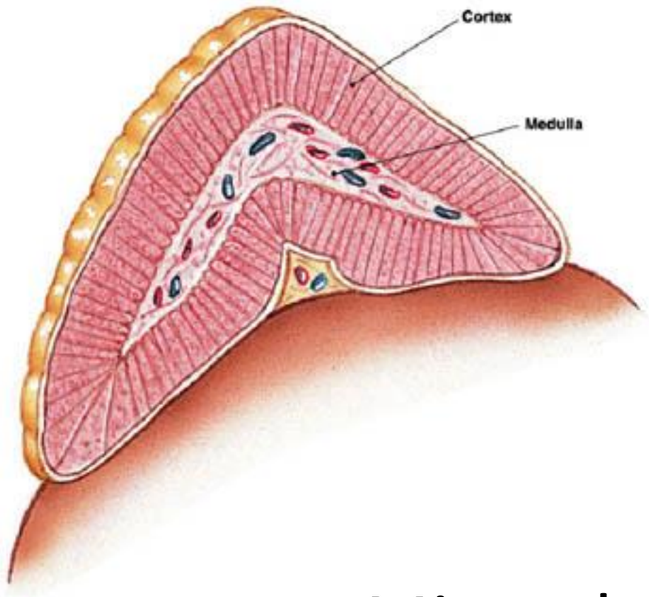

Glucocorticoid (CORTISOL)

↓

Adrenal insufficiency

Mineralocorticoid (aldosterone)

↓

Hyponatremia / Hyperkalemia

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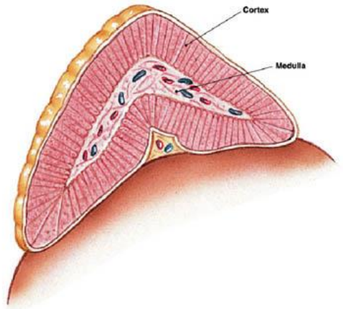
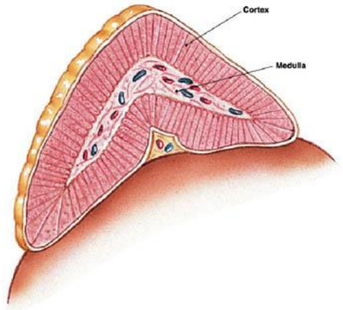


ADRENAL CRISIS

- Causes: PRIMARY: **autoimmune adrenalitis**, idiopathic, INFX (MCC infx worldwide is TB, HIV in US). SECONDARY: iatrogenic due to chronic steroid use
- Addison's Disease
- Unresponsive to IV fluids or pressors
- N/V, abd pain, HYPOGLYCEMIC
- **Hyponatremia** / Hyperkalemia - aldosterone needs be affected
- Waterhouse Friderichsen syndrome: crisis from adrenal hemorrhage in meningococcemia



ADRENAL CRISIS

- Treatment
- IV FLUIDS - D5NS to replace glucose
- **Hydrocortisone** - 100mg q6-8hr - better for vascular tone, though affects cosyntropin test
- **Dexamethasone** 6-8mg - DOES NOT affect cosyntropin test. No mineralocorticoid effects

DISORDERS OF THE ADRENAL GLAND

- Hyperadrenalism (Cushing's Syndrome)
  - Pathophysiology
    - Often due to abnormalities in the anterior pituitary or adrenal cortex.
    - May also be due to steroid therapy for nonendocrine conditions such as COPD or asthma.
    - Long-term cortisol elevation causes many changes.
      - Atherosclerosis, diabetes, hypertension
      - Increased response to catecholamines
      - Hypokalemia and susceptibility to infection

DISORDERS OF THE ADRENAL GLAND

- Assessment & Management
  - Support ABCs.
  - Use caution when establishing IV access.
  - IVF's
  - Decadron suppression test

A 53-YEAR-OLD WOMAN IS BROUGHT TO THE ED BY HER HUSBAND, WHO STATES THAT SHE IS FEELING VERY WEAK OVER THE LAST 2 DAYS, IS NAUSEATED, AND VOMITING AT LEAST THREE TIMES. THE HUSBAND STATES THAT HIS WIFE WAS TAKING A HIGH DOSE MEDICATION FOR HER JOINT PAIN BUT RAN OUT OF HER PILLS LAST WEEK. HER VITAL SIGNS ARE BP OF 90/50 MM HG, HR 87 BEATS PER MINUTE, RR 16 BREATHS PER MINUTE, AND TEMPERATURE 98.1°F. YOU PLACE HER ON THE MONITOR, BEGIN IV FLUIDS, AND SEND HER BLOOD TO THE LABORATORY. THIRTY MINUTES LATER THE METABOLIC PANEL RESULTS ARE BACK AND REVEAL THE FOLLOWING:

NA+ 126 MEQ/L K+ 5 MEQ/L CL− 99 MEQ/L HCO3 21 MEQ/L BUN 24 MG/DL CREATININE 1.6 MG/DL

GLUCOSE 69 MG/DL CA+ 11 MEQ/L

WHAT IS THE MOST LIKELY DIAGNOSIS?

- a. Myxedema coma
- b. Thyroid storm
- c. Hyperaldosteronism
- d. Adrenal insufficiency
- e. Diabetic ketoacidosis (DKA)

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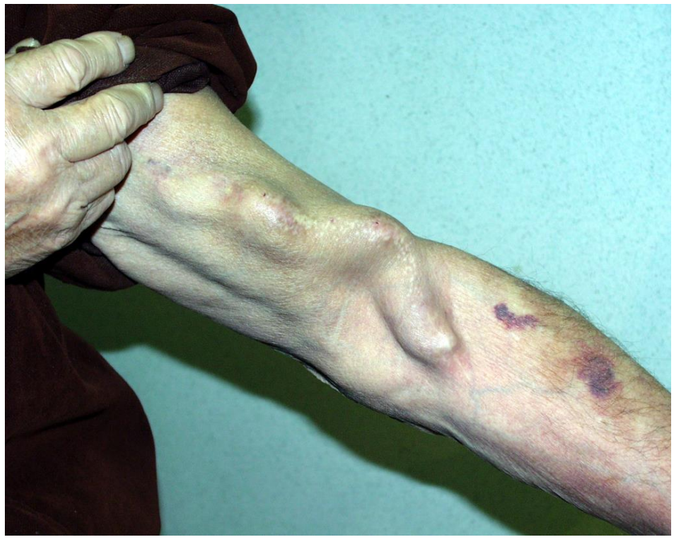
CASE

- EMS arrives with CPR in process. 60 yo male noncompliant with medications and appointments for the past week noted to collapse at home. EMS has shocked patient multiple times without any change in rhythm or pulse.



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DIALYSIS PATIENT CODE

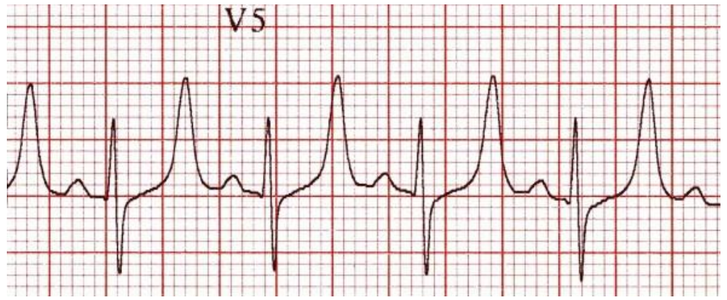
=

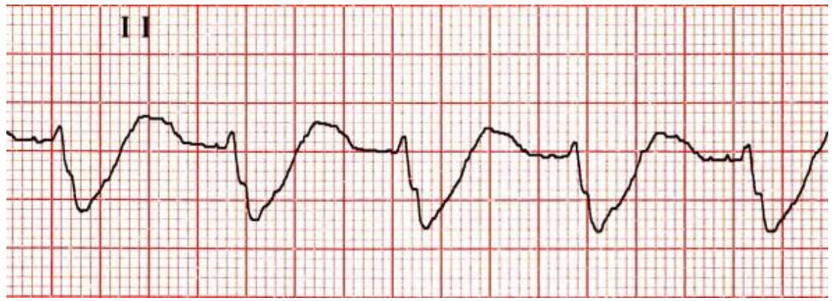
HYPERKALEMIA

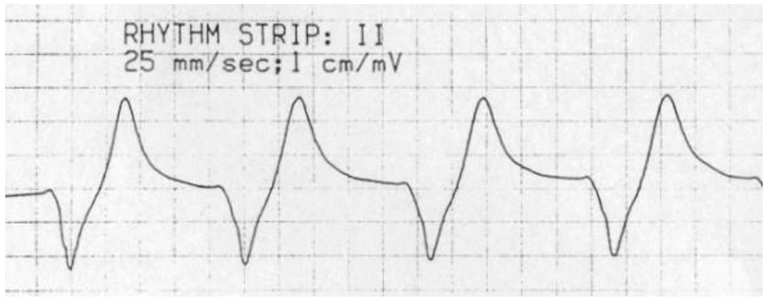
=

CALCIUM / BICARB

HYPERKALEMIA







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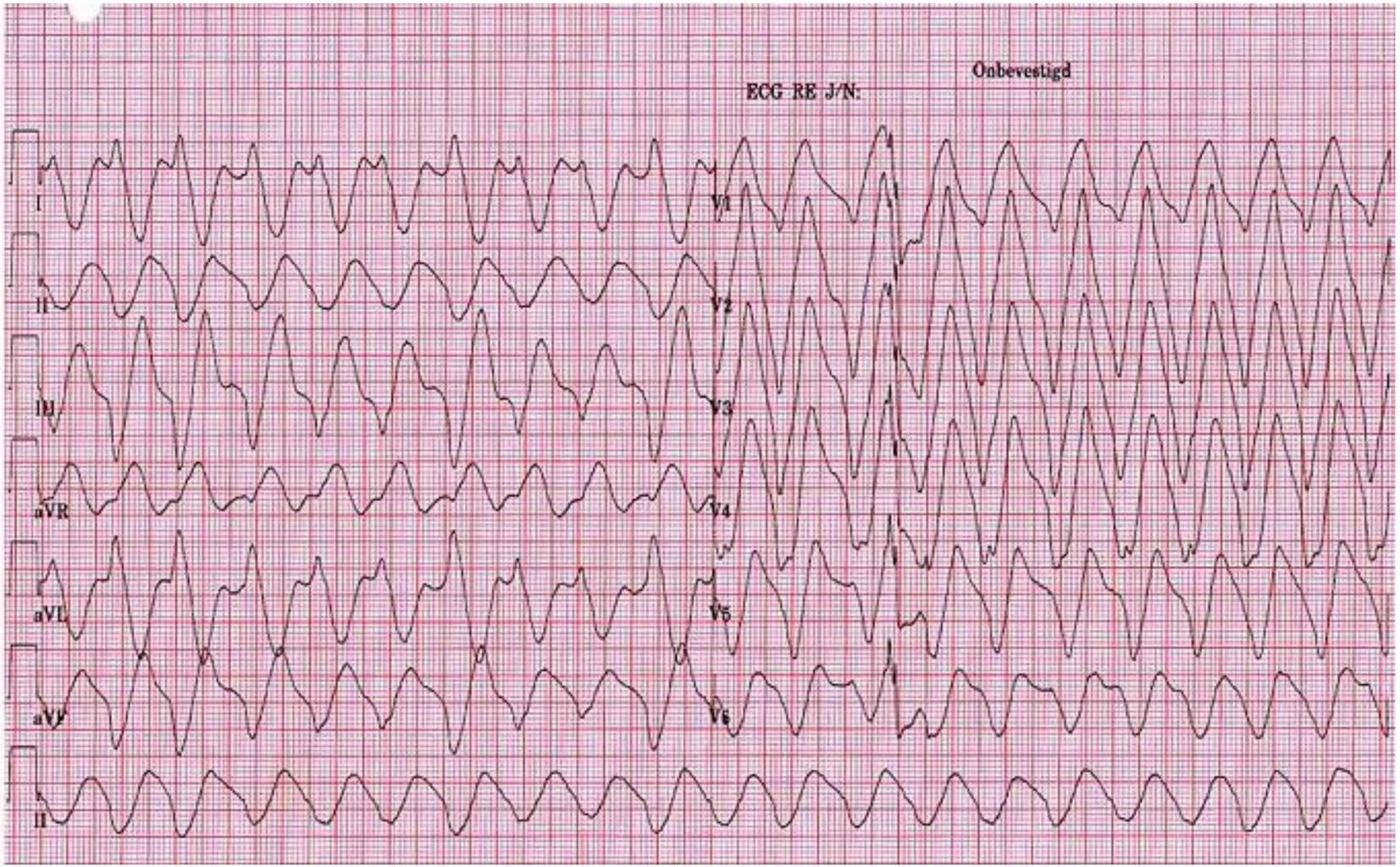

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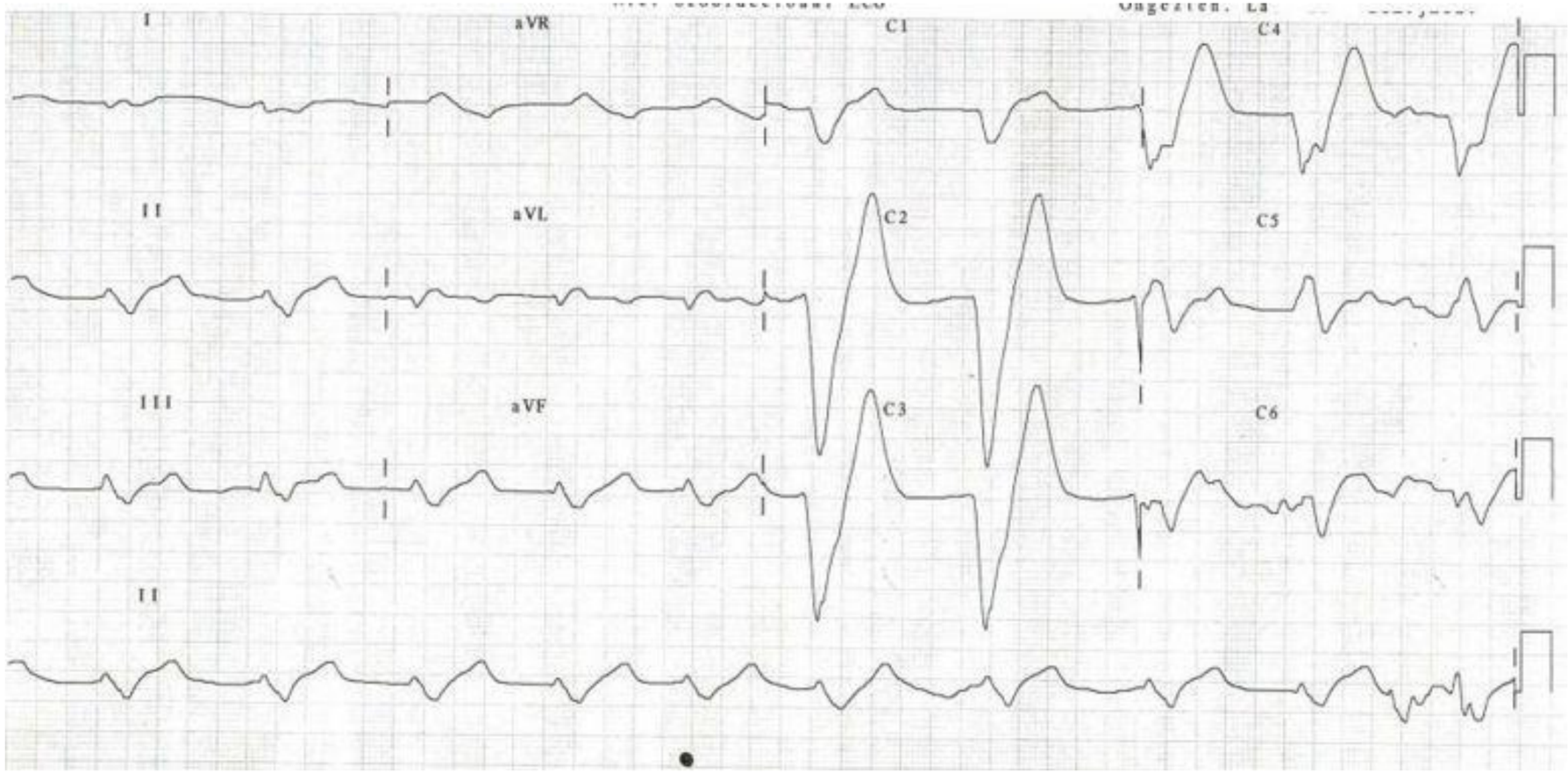

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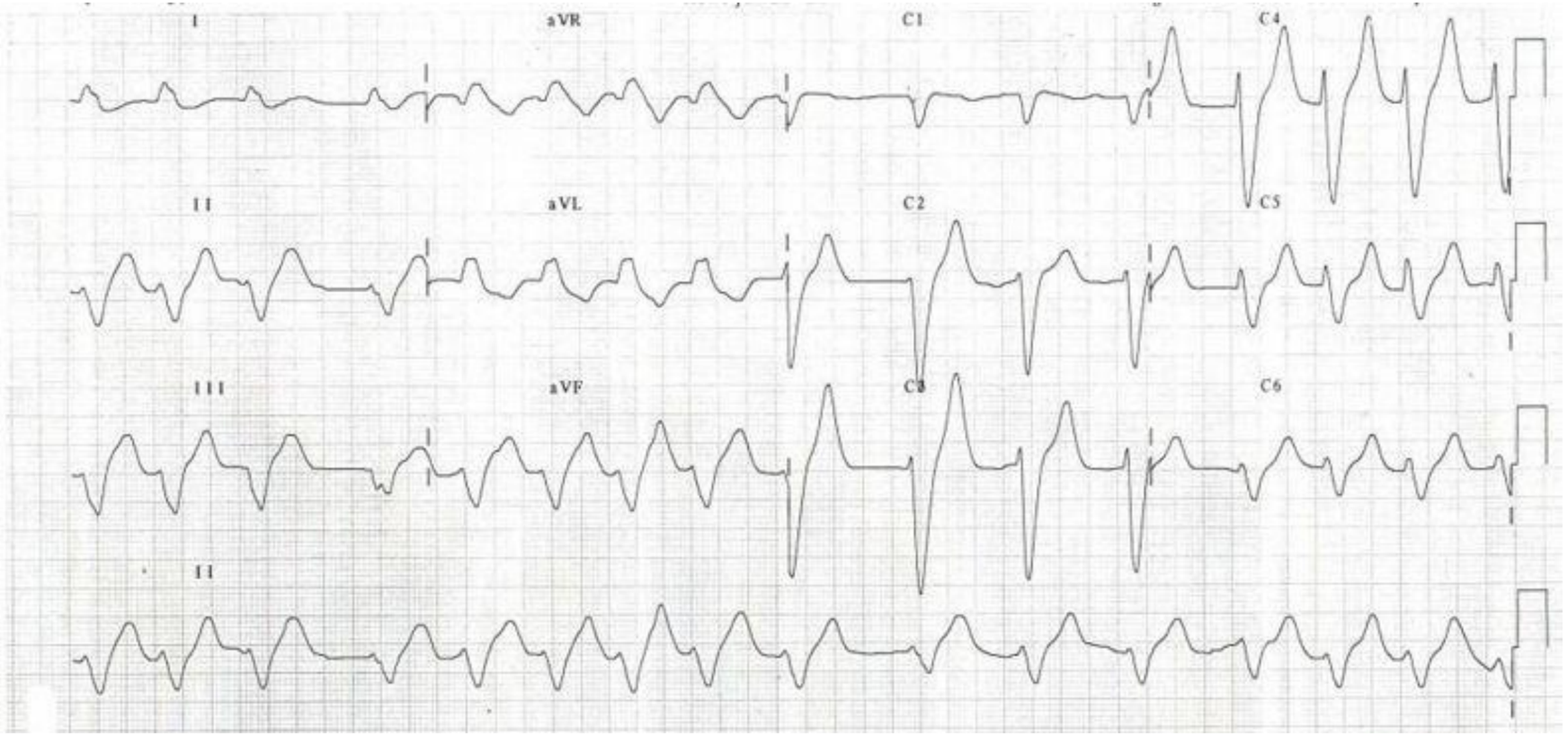

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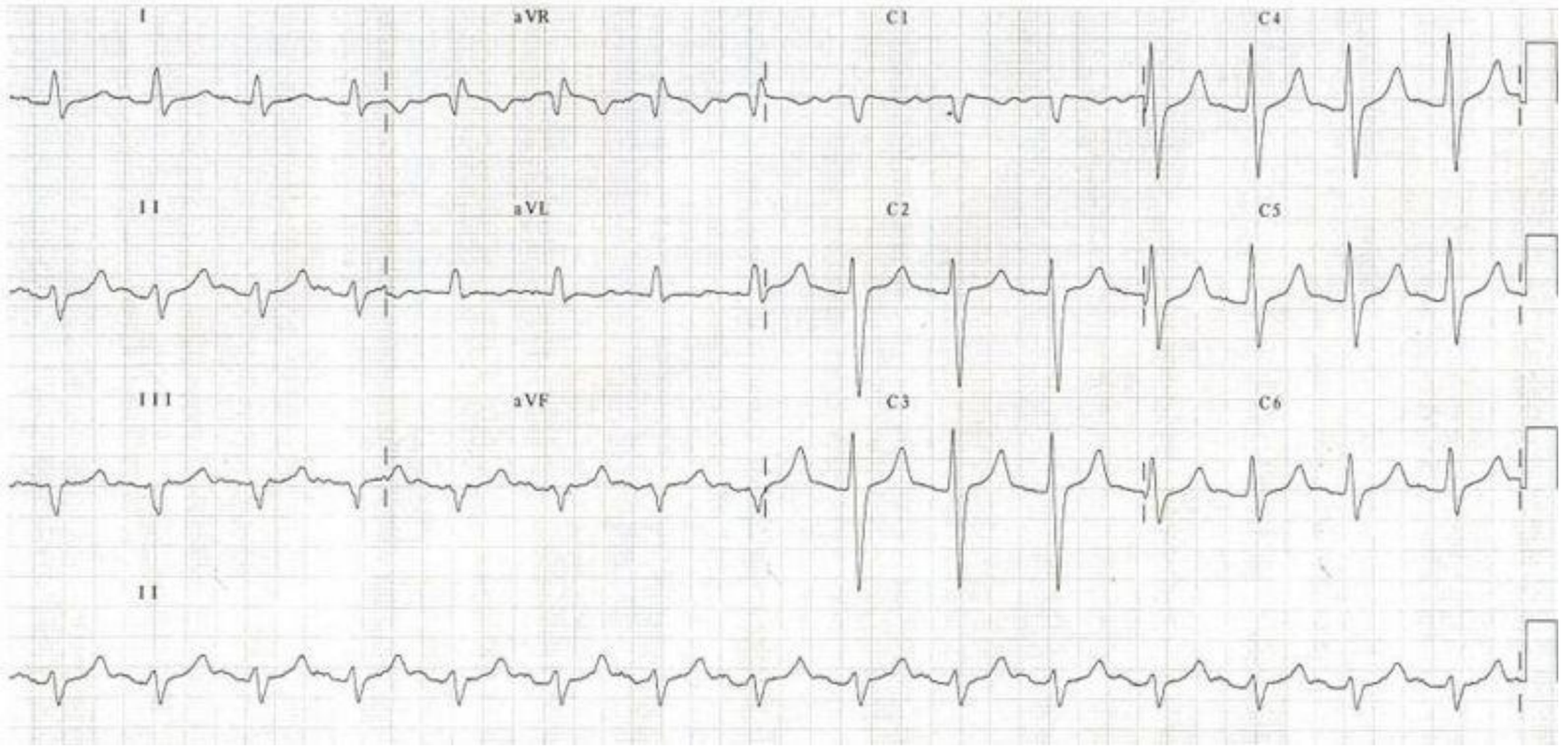

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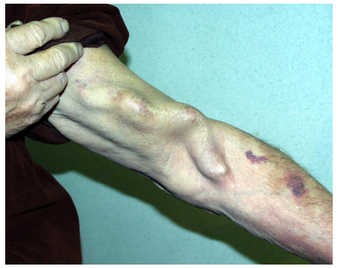
HYPERKALEMIA



- MCC elevated potassium on labs: **Lab error**
- Causes: renal failure, meds (ACE, NSAIDs, TMP/SMX), hemolysis
- Calcium gluconate / chloride - membrane stabilizer, no effect on potassium level
- **Bicarb: onset 5-10 min**
- Insulin / Glucose: onset 30 min
- **Albuterol**: quickest shift
- Dialysis / Kayexalate

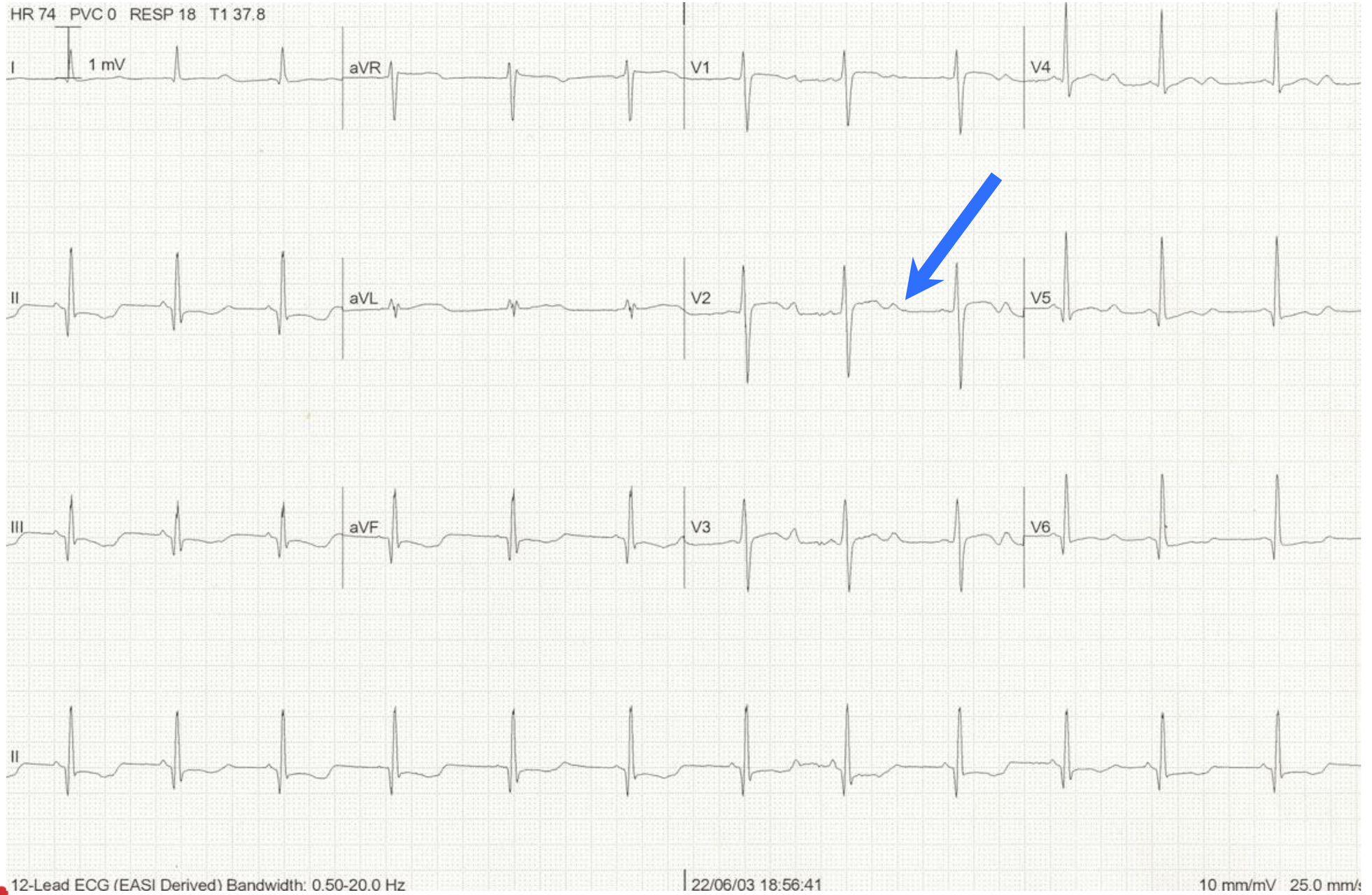
1. Which of the following treatments are not used for hyperkalemia?
    a. IV calcium gluconate
    b. Regular insulin
    c. Dextrose
    d. NaHCO3 80 mEq
    e. Magnesium

HYPOKALEMIA

- Due to **GI losses**: vomiting (metabolic alkalosis), diarrhea, laxatives, fistulas (hypokalemic, hyperchloremic, normal anion gap metabolic acidosis)
- Due to **Renal losses**: diuretics, RTA 1
- Gentamycin, hyperaldosterone
- Alkalosis, insulin, ß-agonists, and aldosterone by cellular reuptake
- Hypercalcemia
- **Familial periodic paralysis**: weakness, paralysis, family hx. DTRs present.
- Can be associated with ↓Mg+2

HYPOKALEMIA

- Symptoms and signs
  - Muscular weakness/cramps, fatigue, constipation, flaccid paralysis, hyporeflexia, hypercabnia, tetany, rhabdomyolysis
- EKG: **U-waves**, flattened T-waves, **Prolonged QT**
- Can cause adynamic ileus
- 100 mEq of potassium raises serum K by 1 mEq/L



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- Causes of hypokalemia include all of the following except:
- a. Steroid therapy
- b. Renal tubular acidosis
- c. Addison's disease
- d. Diuretics
- e. Diarrhea

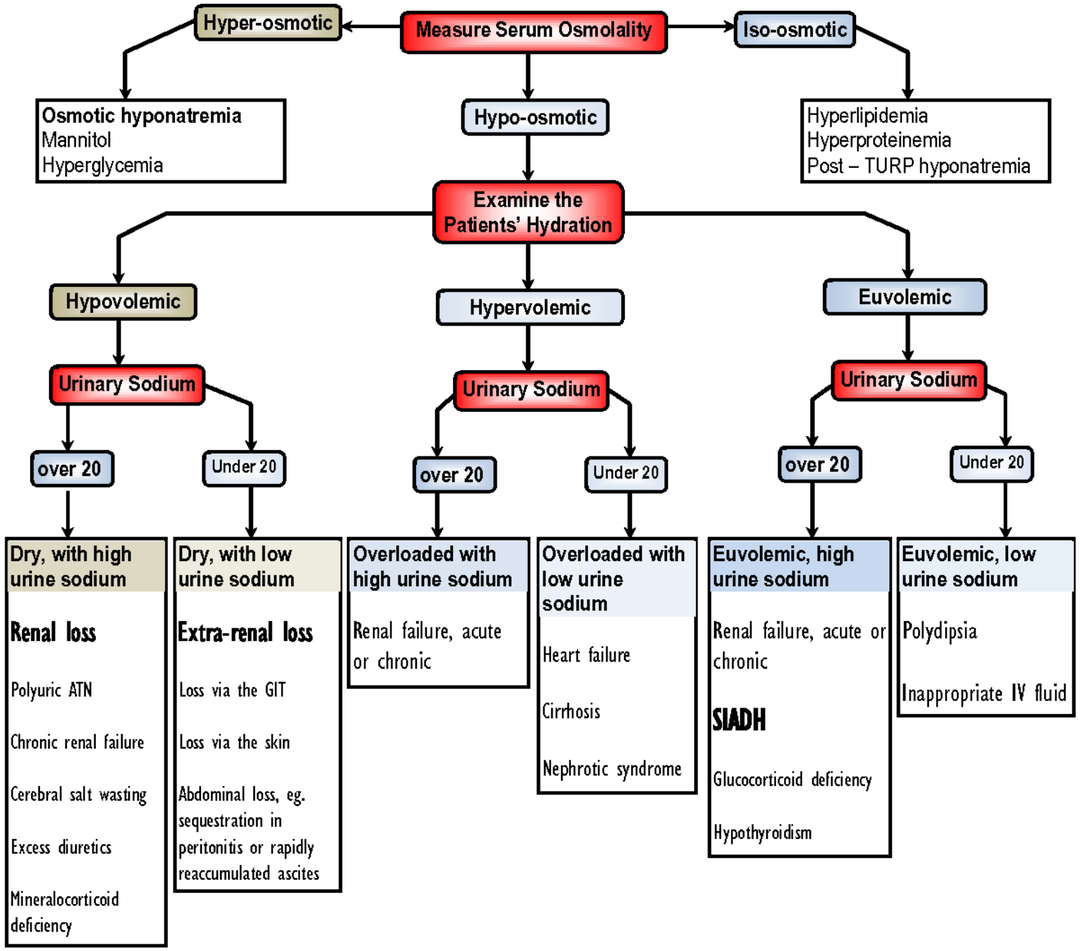
ELECTROLYTE ABNORMALITIES

HYPONATREMIA

- **MCC electrolyte abnormality**
- Pseudohyponatremia in hyperglycemia
- Medication causes: HCTZ, SSRIs, ecstasy
- 3 types

Hyponatremia: classification

An algorithm for investigations of hyponatremia:



How much sodium should there be?

Sodium deficit = 0.6 x body weight x (desired concentration -current concentration)

In a 100 kg man with a sodium of 120mmol, when it ought to be 140mmol, the deficit is 1200mmol.
That's about 8.5 litres of normal saline.

From "Basic Assessment and Support in Intensive Care" by Gomersall et all, as well as "The Washington Manual of Critical Care" by Kollef et al, chapter 23, "Renal and Electrolyte Disorders" by Schrier and this eMedicine article

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HYPONATREMIA

Hypovolemic	→	Sodium loss from V/D diuretics (**HCTZ**)
Hypervolemic	→	CHF, cirrhosis, nephrotic syndrome
Euvolemic	→	Psychogenic polydipsia SIADH

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SIADH

- Confusion, headache, seizures
- Antidiuretic = holds on to water
- Causes: **lung cancer**, CNS lesions
- Urine osmolality less than expected (**concentrated**)
- Diagnosis of exclusion (thyroid, adrenal, renal dx)

WHICH OF THE FOLLOWING CHARACTERIZES SIADH?

- a. Urine osmolality < 100 mOsmo/L
- b. Hyponatremia
- c. Urine Na < 20
- d. EtOH increases release of ADH
- e. Morphine decreases release of ADH

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HYPERTONIC SALINE?

- Severe symptoms (**seizures / coma**)
- Tx hyponatremia SLOWLY or else…
- **Osmotic Demyelination Syndrome**
- Treat < 0.6mEq/L/hr
- Consider crash-cart bicarb (11% hypertonic)

HYPERNATREMIA

- Reduced water intake
- Diabetes insipidus: decreased ADH (central or renal)
- AMS, seizures, intracranial hemorrhage
- Free water deficit: 0.6 x wt x (sodium/140 - 1)
- Tx with isotonic over a few days
- Too quick: **cerebral edema**

HYPERCALCEMIA

- Stones, Bones, Thrones, Groans, Psychiatric Overtones
- MCC: hyperparathyroidism, **cancer**
- AMS, confusion, Polyuria and constipation
- EKG
  - Shortened QT
  - Ventricular extrasystole
  - Idioventricular rhythm
- Tx: **HYDRATION**
  - LOOP DIURETIC, **NOT HCTZ** (increases)
  - Biphosphonate (pamidronate)
    - Tx for CA related
  - Calcitonin

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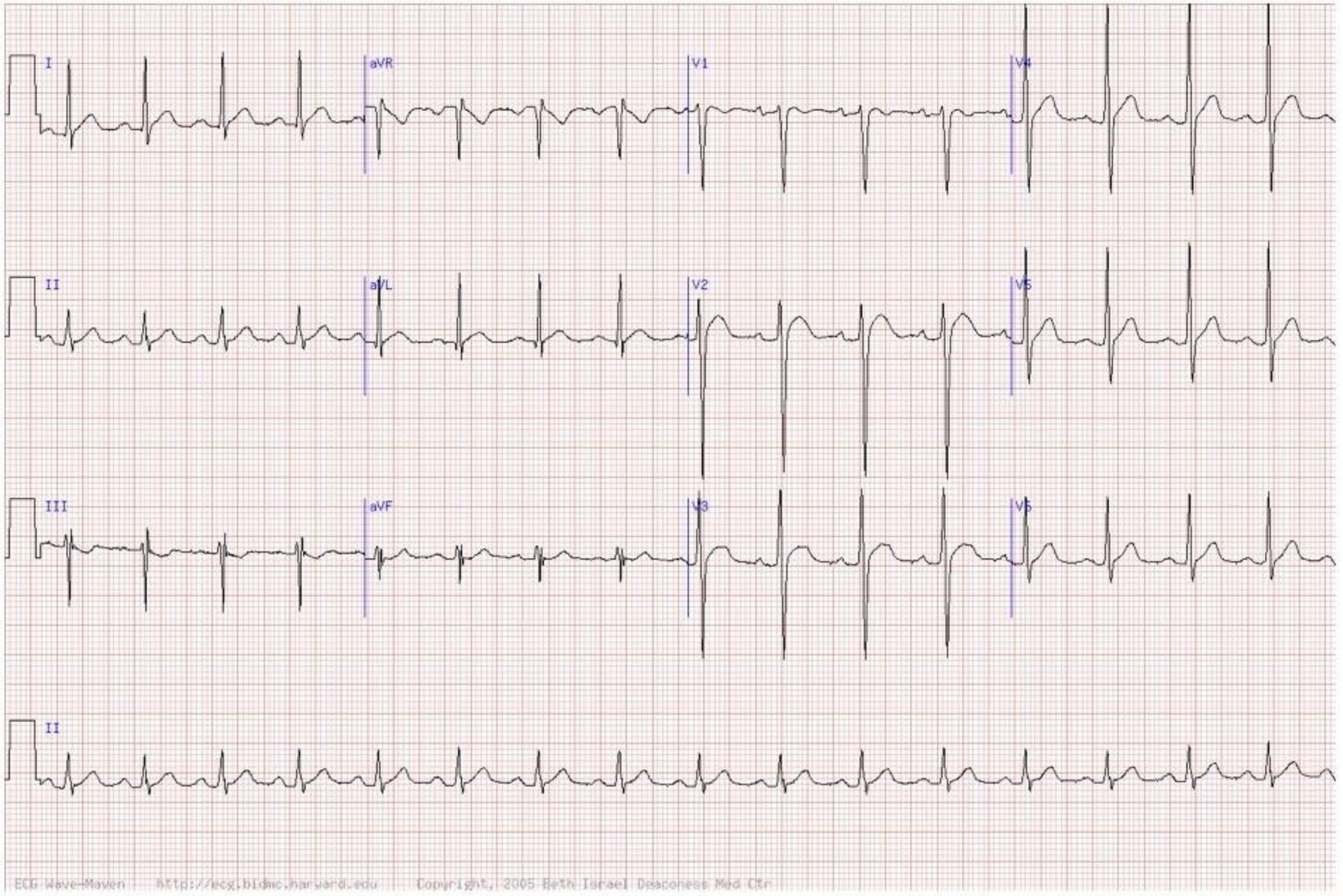
HYPERCALCEMIA

- Causes (**CHIMPAZEES**)
  - Ca supplement
  - Hyperparathyroidism / Hypophosphatemia
  - Iatrogenic (thiazides)
  - Milk-alkali syndrome (ingestion)
  - Paget's disease
  - Addison / acromegaly
  - Neoplasia / metastasis (breast CA, MM, lung CA, lymphoma)
  - Zollinger-Ellison
  - Excessive vitamin A
  - Excessive vitamin D
  - Sarcoidosis



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HYPOCALCEMIA

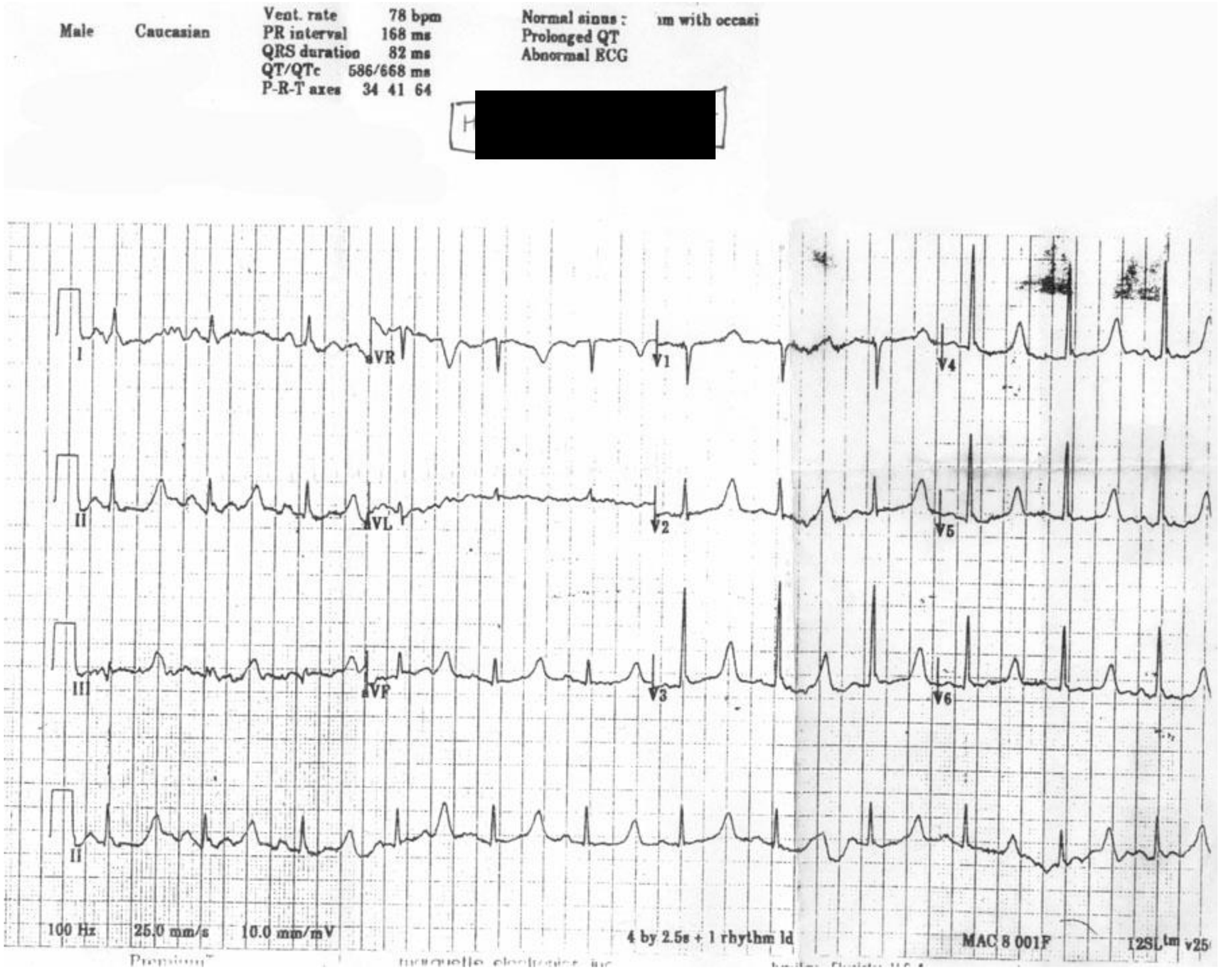
- Causes:
  - Hypoparathyroidism
  - Hypomagnesimia (EtOH)
  - Hyperphosphatemia
  - Rhabdomyolysis
  - Massive blood transfusion
  - ARF/CRF due to defect in the convertion of 1-OH-D3 to 1,25-(OH)2-D3
- Low albumin will cause falsely low calcium on lab; **ionized calcium** the important one
- Prolonged QT, perioral parathesias
- **Chvostek's sign**: facial twitch
- **Trousseau's sign**: BP twitch

HYPOCALCEMIA

- Symptoms and signs
  - Increases excitation of nerve and muscle cells causing cramps, tetany
  - Laryngospasm w/ stridor
  - Convulsions
  - Paresthesia of lips and extremities; hyperreflexia; dystonia
  - Chvostek's sign (contraction of face)
  - Trousseau's sign (carpal spasm)

HYPOCALCEMIA

- EKG
  - QT prolongation
  - ST prolongation
- Treatment
  - Thiazide diuretics
  - Ca gluconate
  - Correction of Mg^{+2}



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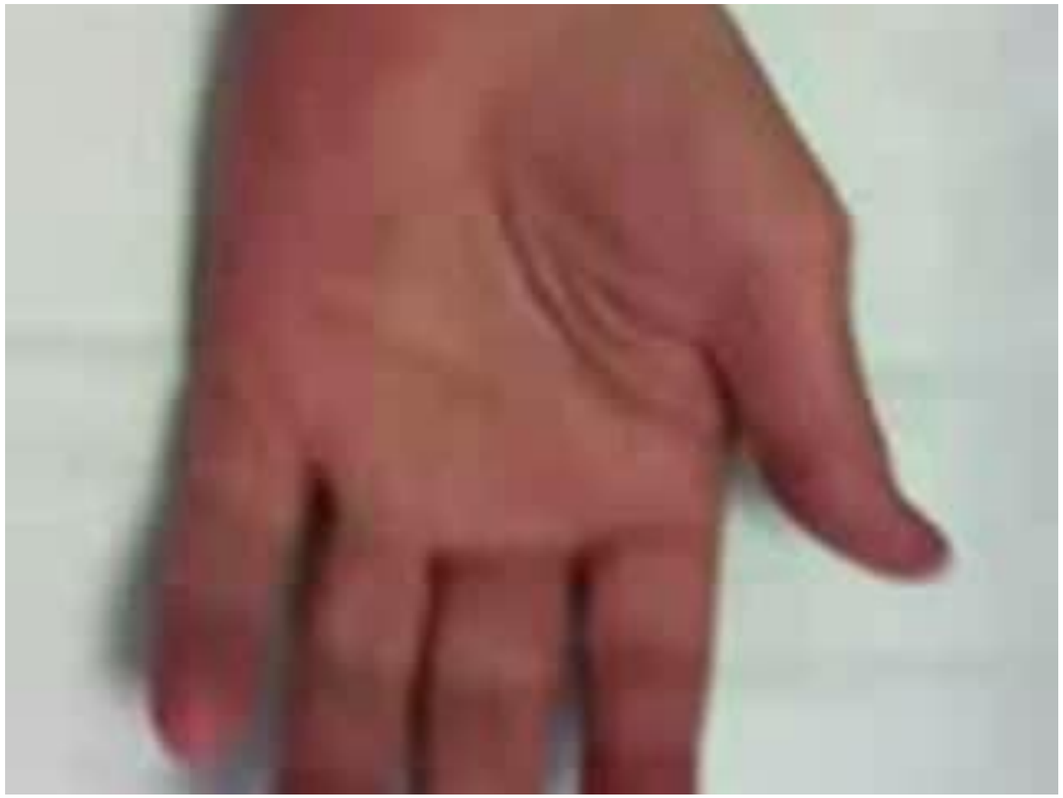

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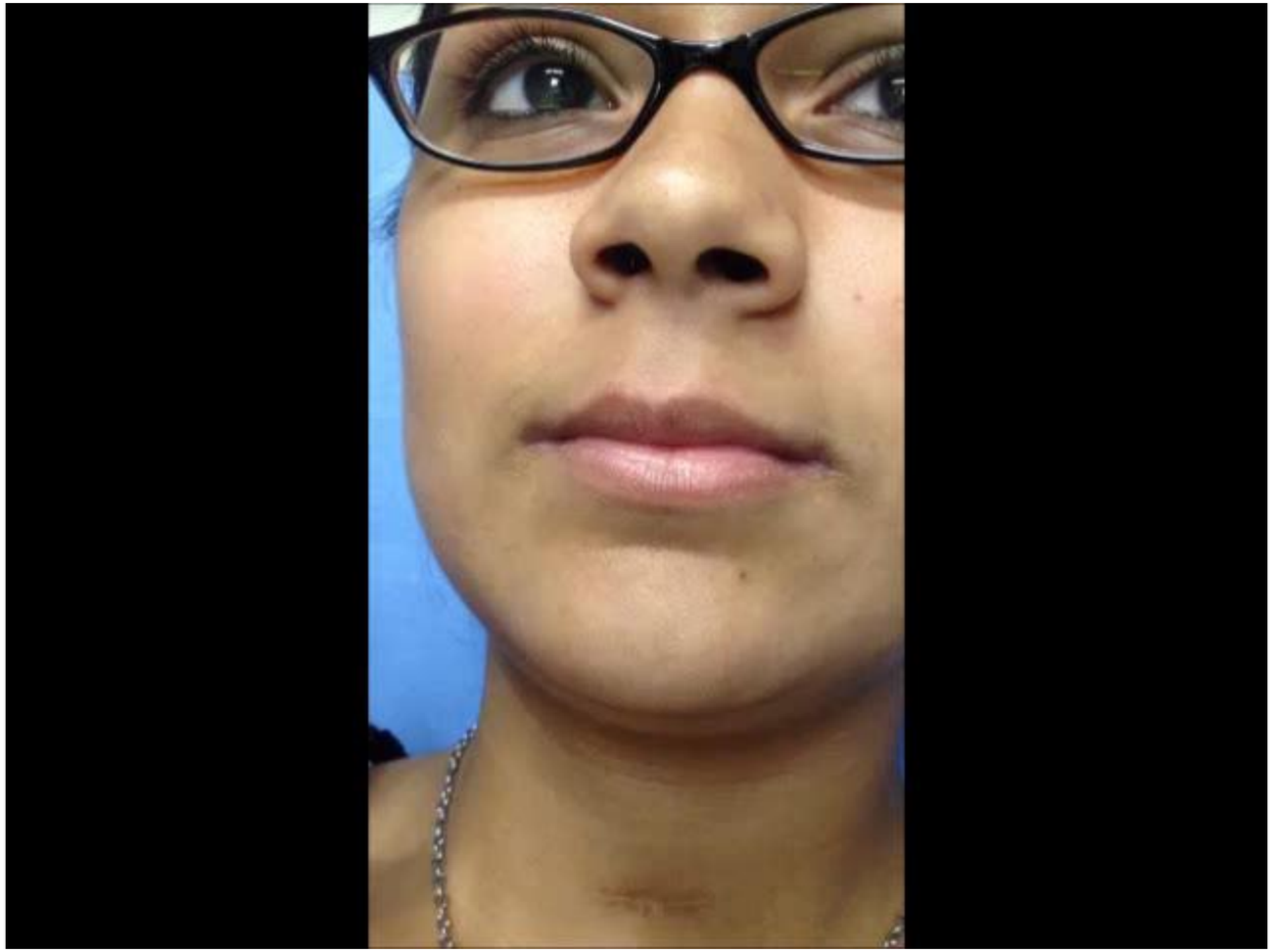
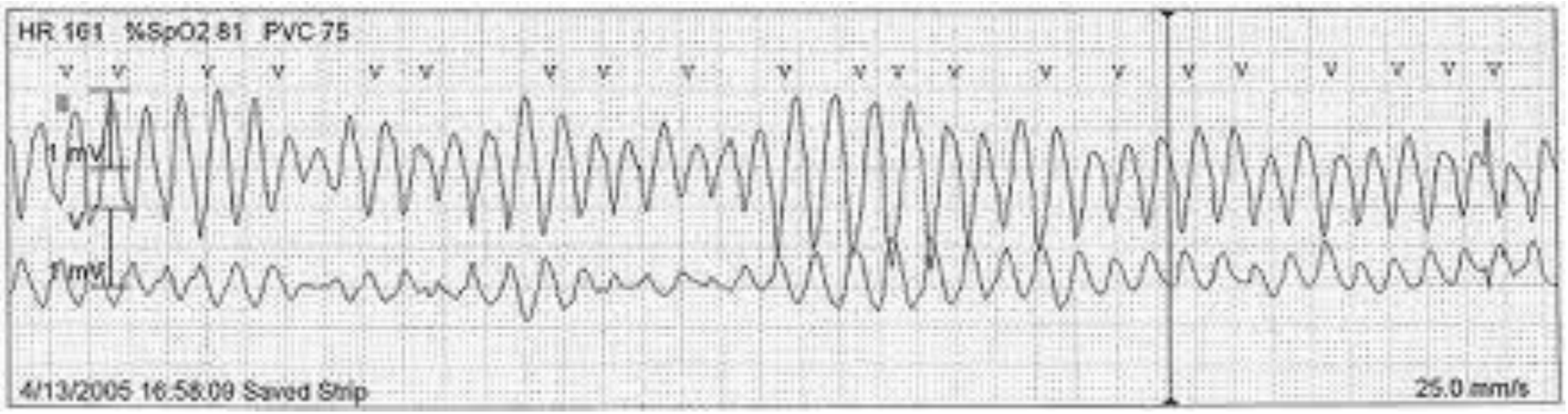


HYPOMAGNESEMIA

- **Malnutrition**, alcoholism, DKA
  - Hypokalemia, hyponatremia, hypocalcemia
  - Renal wasting
  - Diuretics, gentamycin, cisplatin, ampho B
  - Renal distribution
  - Alcohol withdrawal, insulin administration, hungry bone syndrome, malnutrition, burns
- Looks like hypokalemia / hypocalcemia - think with **Chvostek sign**
- Look for low potassium / calcium as well
- Mag sulfate if severe

HYPOMAGNESEMIA

- Symptoms and sign
  - Weakness, muscle cramps, tremor
  - Neuromuscular, CNS hyperirritability (jerking, nystagmus, Babinski)
  - Confusion, same as hypocalcemia
- EKG
  - Prolongued QT
  - Peaked T
  - Depressed ST
  - Torsades de pointe
- Treatment
  - IV/PO supplement
  - Correction of electrolytes



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HYPERMAGNESEMIA

- Renal failure, iatrogenic
  - Mg containing laxatives or antacids
  - MgSO4 infusion during eclampsia
  - Renal insufficiency
- Weakness, **HYPOREFLEXIA**
- Confusion
- Respiratory failure….death
- Hypotension
- Tx: Calcium / Dialysis / Loops

PHOSPHORUS

- Hypo: malnutrition/diuretics, have weakness. Look for in **DKA**
- Hyper: **RENAL FAILURE**, tumor lysis syndrome. Tx with dialysis or aluminum phosphate binders (aluminum hydroxide)

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HYPOPHOSPHATEMIA

- Causes
  - Alcoholic patients due to poor intake, respiratory alkalosis
  - Vitamin D deficiency, malnutrition
  - Hypercalcemia
  - RTA1
  - Hyperparathyroidism
  - Hypomagnesium
  - Sepsis

HYPOPHOSPHATEMIA

- Symptoms and sign
  - acute hemolytic anemia, platelet dysfunction
  - rhabdomyolyisis, encephalopathy, cardiomyopathy
  - respiratory insufficiency due to diaphragm dysfunction
  - profound muscle weakness
- Treatment
  - underlying condition
  - IV/PO supplement
  - correction of magnesium

HYPERPHOSPHATEMIA

- Causes
  - Acute tubular necrosis (drugs)
  - IV solutions
  - **Tumor lysis**
  - Hypoparathyroidism
  - Renal failure; rhabdo

HYPERPHOSPHATEMIA

- Symptoms and signs are depending of the underlying condition
- Treatment
  - treat underlying condition
  - calcium gluconate
  - antacids

QT PROLONGATION

- HYPOKALEMIA
- HYPOCALCEMIA
- HYPOMAGNESEMIA
- **HYPERPHOSPHATEMIA**