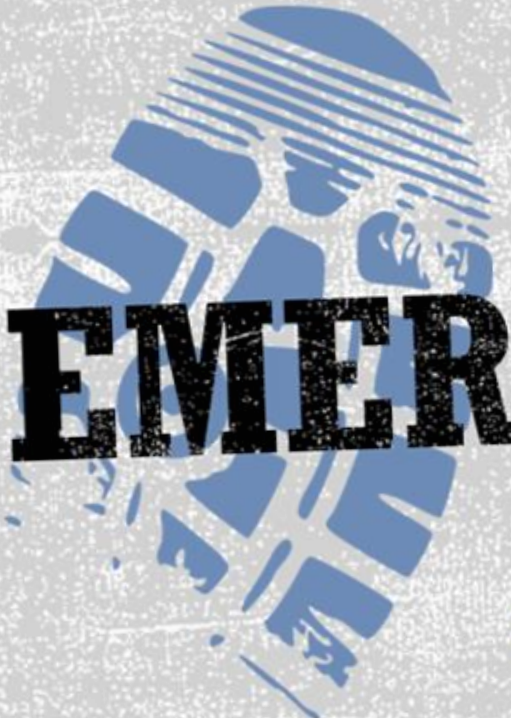
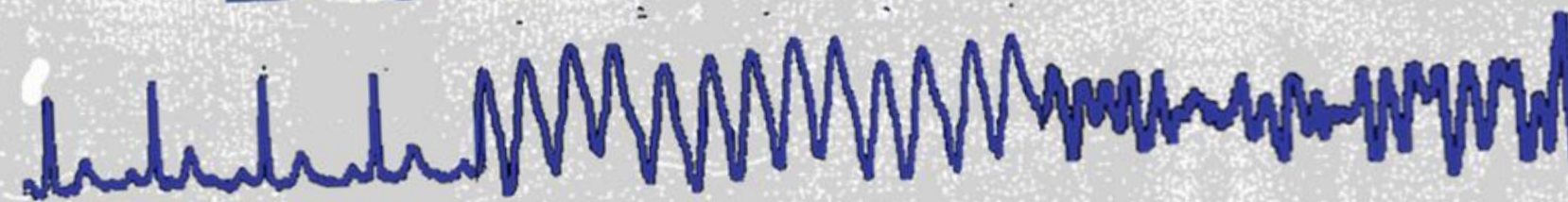




EMERGENCY MEDICINE

BOOT CAMP





ENDOCRINE AND ELECTROLYTE DISORDERS

José A. Rubero, MD, FACEP, FAAEM

Professor

CASE PRESENTATION

- *You're working a shift in the ED when a 23-year-old female with Type 1 Diabetes is brought in by EMS. She has been vomiting, feeling weak, and breathing heavily for the past 12 hours. Her roommate says she ran out of insulin two days ago. On exam, she is tachycardic, tachypneic, and has dry mucous membranes. Her vitals: BP 95/60, HR 120, RR 28, Temp 98.8°F, SpO2 98%.*
- *What's your initial impression?*
- **Key question:** Is this **DKA** or **HHS**, and how do we tell the difference?



CLINICAL FEATURES & DIAGNOSTIC WORKUP

- **Key Symptoms to Ask About:**

- **DKA:** Polyuria, polydipsia, vomiting, abdominal pain, fruity breath, Kussmaul respirations.
- **HHS:** Weakness, confusion, extreme dehydration, blurry vision, lethargy.

- **Physical Exam Clues:**

- **Tachycardia, hypotension** → Dehydration in both DKA & HHS.
- **Deep Kussmaul breathing** → DKA.
- **Altered mental status or coma** → More likely in HHS.



CLINICAL FEATURES & DIAGNOSTIC WORKUP

- **Diagnostic Workup:**

- **Fingerstick Glucose:** Immediate test, usually >250 in DKA and >600 in HHS.
- **ABG or VBG:** Determines acidosis ($\text{pH} < 7.3$ in DKA).
- **Serum Ketones (Beta-hydroxybutyrate):** Present in **DKA**, absent or minimal in **HHS**.
- **Electrolytes:**
 - **Potassium (K^+):** Often normal or high initially but drops rapidly with insulin therapy.
 - **Sodium (Na^+):** Pseudohyponatremia due to hyperglycemia.
- **Serum Osmolality:**
 - >320 mOsm/kg suggests **HHS** (severe dehydration).
- **BUN/Creatinine:** Elevated in both due to dehydration and AKI risk.



PATHOPHYSIOLOGY & DIFFERENCES BETWEEN DKA AND HHS

- **Diabetic Ketoacidosis (DKA) – Common in Type 1 Diabetes**
 - Caused by **absolute insulin deficiency** → Lipolysis → Ketone production → **Metabolic Acidosis**.
 - Key features:
 - **Glucose:** 250-600 mg/dL (not extremely high).
 - **Ketosis & Acidosis:** pH <7.3, HCO₃ <18.
 - **Kussmaul breathing (deep, labored breathing)** to compensate for acidosis.
 - **Fruity breath odor (acetone)** from ketone production.
- **Hyperosmolar Hyperglycemic State (HHS) – Common in Type 2 Diabetes**
 - **Relative insulin deficiency** → Severe hyperglycemia → Profound dehydration → **No significant acidosis**.
 - Key features:
 - **Glucose:** 600-1000+ mg/dL (much higher than DKA).
 - **No significant ketosis or acidosis** (pH >7.3, HCO₃ >18).
 - **Severe dehydration** → AMS, lethargy, even coma.



PATHOPHYSIOLOGY & DIFFERENCES BETWEEN DKA AND HHS

- **Red Flags for Severe Disease:**
 - **Altered mental status** → More common in **HHS** due to profound dehydration.
 - **Severe acidosis (pH <7.0)** → Indicates **severe DKA** and risk of cardiovascular collapse.
 - **Hypotension, tachycardia, dry mucous membranes** → Suggest **severe volume depletion** in both conditions.



MANAGEMENT & TREATMENT

Step 1: IV Fluids (Most Important Initial Therapy!)

- **1-2L of Normal Saline (NS) bolus** over first 1-2 hours.
- **Switch to D5-½NS when glucose <250 mg/dL** to prevent hypoglycemia while continuing insulin therapy.

Step 2: Insulin Therapy

- **IV Regular Insulin (0.1 U/kg bolus, then infusion 0.1 U/kg/hr).**
- **No bolus if K⁺ <3.3 mEq/L** (fix potassium first!).

Step 3: Potassium Replacement

- **If K⁺ <3.3:** Hold insulin, give IV K⁺ replacement.
- **If K⁺ 3.3-5.0:** Give 20-40 mEq/L K⁺ in IV fluids.
- **If K⁺ >5.2:** Monitor closely but don't supplement.



MANAGEMENT & TREATMENT

Step 4: Monitor and Correct Other Deficits

- **Check glucose, pH, and electrolytes every 2-4 hours.**
- **Bicarbonate:** Only for pH <6.9 in severe DKA.
- **Treat the underlying cause** (infection, missed insulin, MI, pancreatitis).

When to Transition to Subcutaneous Insulin?

- **DKA resolved when:**
 - Glucose <200 mg/dL **and**
 - Bicarb >18, pH >7.3, no anion gap.
- **Overlap IV insulin with subcutaneous insulin by 1-2 hours** to prevent rebound ketoacidosis.



SUMMARY & CLINICAL PEARLS

- **DKA = Ketoacidosis & hyperglycemia (usually Type 1 DM). HHS = Severe hyperglycemia & dehydration (usually Type 2 DM).**
- **DKA patients are acidotic (pH <7.3), while HHS patients have higher serum osmolality (>320 mOsm/kg).**
- **IV fluids first, then insulin, and always monitor potassium.**
- **DKA resolves when acidosis clears, not just when glucose normalizes!**



CASE PRESENTATION

- *You're working a shift in the ED when EMS brings in a 52-year-old male with profound weakness, nausea, vomiting, and confusion. His blood pressure is 75/45, HR 110, RR 22, Temp 98.1°F, SpO2 98%. The family reports he has Addison's disease but recently had a gastrointestinal illness and hasn't been able to take his usual medications.*
- *What's your next step?*
- **Key question:** Is this **adrenal crisis**, and how do we treat it immediately?
- *Adrenal crisis is a failure of the adrenal glands to produce enough cortisol, a hormone essential for maintaining blood pressure, glucose, and stress response*



PATHOPHYSIOLOGY & CAUSES

- **Causes of Adrenal Crisis:**
 - **Primary Adrenal Insufficiency (Addison's Disease)** – Autoimmune destruction of adrenal glands.
 - **Secondary Adrenal Insufficiency** – Hypopituitarism leading to low ACTH.
 - **Chronic Steroid Use with Sudden Withdrawal** – Most common cause in the ED.
 - **Acute Stress in Patients with Known Adrenal Insufficiency** – Infections, surgery, trauma, or illness triggering crisis.
- **Why Is Cortisol So Important?**
 - **Maintains blood pressure** (via vascular tone and sodium retention).
 - **Regulates glucose metabolism** (prevents hypoglycemia).
 - **Suppresses excessive immune response** (prevents inflammatory overdrive).
 - **Helps the body respond to stress.**



PATHOPHYSIOLOGY & CAUSES

- **Red Flags for Adrenal Crisis:**
 - **Hypotension refractory to fluids** → Think adrenal crisis.
 - **History of steroid use with acute illness** → Risk for adrenal suppression.
 - **Severe fatigue, nausea, vomiting, and confusion** → Suggests cortisol deficiency.



CLINICAL FEATURES & DIAGNOSTIC WORKUP

- *Adrenal crisis is often missed because it mimics sepsis or other shock states. Here's how to recognize it.*
- **Key Symptoms:**
 - **Fatigue, Weakness, Confusion** – Due to lack of cortisol.
 - **Hypotension & Shock** – Refractory to IV fluids alone.
 - **GI Symptoms:** Nausea, vomiting, abdominal pain (mimicking acute abdomen).
 - **Hypoglycemia** – Due to impaired gluconeogenesis.
- **Physical Exam Findings:**
 - **Hypotension** – Unresponsive to fluids.
 - **Hyperpigmentation (in Primary Adrenal Insufficiency)** – Due to excess ACTH stimulating melanin.
 - **Dehydration** – Due to aldosterone deficiency (causing sodium loss).



CLINICAL FEATURES & DIAGNOSTIC WORKUP

- **Diagnostic Workup (Don't Delay Treatment!)**
 - **Serum Cortisol & ACTH** – Low cortisol confirms adrenal insufficiency.
 - **Electrolytes:**
 - **Hyponatremia & Hyperkalemia** – Classic in primary adrenal insufficiency (due to low aldosterone).
 - **Hypoglycemia** – Often present in adrenal crisis.
 - **ACTH Stimulation Test** (if diagnosis unclear) – Measures cortisol response to synthetic ACTH.
 - **Renin & Aldosterone Levels** – Helps distinguish primary vs. secondary adrenal insufficiency.



MANAGEMENT & TREATMENT

- *“Once you suspect adrenal crisis, treat immediately—don’t wait for labs!”*
- **Step 1: Immediate IV Hydrocortisone**
 - **Hydrocortisone 100 mg IV bolus, then 50 mg IV q6h or 200 mg continuous infusion.**
 - If hydrocortisone unavailable, use **Dexamethasone 4 mg IV** (does not interfere with cortisol testing).
- **Step 2: IV Fluids for Shock**
 - **Bolus 1-2L Normal Saline (NS) or D5NS** (corrects dehydration & sodium loss).
- **Step 3: Treat Hypoglycemia & Electrolyte Imbalances**
 - **Hypoglycemia → IV Dextrose (D50 push if symptomatic).**
 - **Hyperkalemia → Monitor, may resolve with steroids & fluids.**
- **Step 4: Identify & Treat Underlying Cause**
 - **Infection?** Give IV antibiotics.
 - **Recent steroid withdrawal?** Reinstate maintenance steroids.



SUMMARY & CLINICAL PEARLS

- **Adrenal crisis is a life-threatening condition caused by cortisol deficiency—always suspect it in shock patients with a history of steroid use or adrenal disease.**
- **Hypotension refractory to fluids + Hyponatremia + Hyperkalemia + Hypoglycemia → Think adrenal crisis!**
- **Immediate treatment: IV Hydrocortisone + IV Fluids + Correct hypoglycemia.**
- **Don't wait for labs—treat first if adrenal crisis is suspected!**



CASE PRESENTATION

- **Thyroid Storm Case:**

A 42-year-old female with a history of Graves' disease presents with high fever, tachycardia, confusion, and profuse sweating. She stopped taking her antithyroid medications two weeks ago. BP 140/90, HR 140, Temp 103.1°F

- *What's your first concern?*

- **Myxedema Coma Case:**

A 65-year-old male with a history of hypothyroidism is brought in with confusion, bradycardia, and hypothermia. BP 90/60, HR 48, Temp 94.2°F. He ran out of his levothyroxine two months ago.

- *What are you worried about?*



DIFFERENTIAL DIAGNOSIS

- For Thyroid Storm:
 - Infections (sepsis, pneumonia)
 - Pheochromocytoma (excess catecholamines)
 - Cocaine toxicity
 - Malignant hyperthermia
- For Myxedema Coma:
 - Sepsis
 - Adrenal insufficiency
 - Stroke
 - Hypothermia from environmental exposure



CLINICAL FEATURES & DIAGNOSTIC WORKUP

- **Thyroid Storm Symptoms:**
 - Hyperthermia (Fever $>103^{\circ}\text{F}$)
 - Tachycardia $\pm$ Atrial Fibrillation
 - Hypertension progressing to shock
 - Altered mental status (agitation, delirium)
 - Tremors, nausea, vomiting
- **Myxedema Coma Symptoms:**
 - Hypothermia ($<95^{\circ}\text{F}$)
 - Bradycardia & Hypotension
 - Respiratory depression & CO_2 retention
 - Altered mental status (coma, lethargy)
 - Delayed reflexes, puffy face, dry skin



CLINICAL FEATURES & DIAGNOSTIC WORKUP

- **Key Labs for Both:**
 - **TSH, T3, T4:**
 - *Thyroid Storm:* TSH ↓, T3/T4 ↑
 - *Myxedema Coma:* TSH ↑, T3/T4 ↓
 - **CBC, Blood Cultures:** Rule out infection.
 - **Cortisol:** Consider adrenal insufficiency.
 - **Arterial Blood Gas (ABG):** Check for respiratory failure



MANAGEMENT & TREATMENT

- **Thyroid Storm Management:**
 - **Beta-blockers (Propranolol)** → Controls heart rate.
 - **Thionamides (PTU or Methimazole)** → Blocks new thyroid hormone synthesis.
 - **Iodine (Lugol's Solution)** → Given 1 hour after thionamides to inhibit release.
 - **Steroids (Hydrocortisone 100mg IV)** → Prevents adrenal insufficiency.
 - **Supportive Care:** Fluids, cooling measures, electrolyte correction.
- **Myxedema Coma Management:**
 - IV Levothyroxine (T4) ± Liothyronine (T3)
 - Hydrocortisone 100mg IV (If adrenal insufficiency suspected)
 - Passive rewarming (warm blankets, NOT aggressive rewarming)
 - IV Fluids ± Pressors for hypotension
 - Ventilatory support if respiratory depression present



SUMMARY & CLINICAL PEARLS

- Thyroid Storm = Overactive thyroid → Fever, tachycardia, altered mental status.
- Myxedema Coma = Hypothyroidism → Hypothermia, bradycardia, respiratory depression.
- Treat thyroid storm with beta-blockers, PTU, iodine, and steroids.
- Treat myxedema coma with IV thyroid hormone and supportive care.
- Don't forget to check for adrenal insufficiency in both conditions!



ENDOCRINE EMERGENCIES

- DKA/HHS
- Thyroid storm
- Myxedema coma
- Adrenal crisis
- Electrolyte emergencies



PRESENTATIONS

- Altered mental status
- Weakness
- Polyuria/polydipsia
- Arrhythmia
- Seizures



FORMULAS YOU GOTTA KNOW

- **Anion Gap = $| \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-) |$**
- **Osmol Gap = $(2 \times \text{Na}^+) + \text{Glucose}/18 + \text{BUN}/2.8$**
- **Winter's Formula = $1.5 \times \text{HCO}_3 + 8 = \text{expected CO}_2$**
- **Corrected calcium**
 - $[\text{Ca}] + (4 - [\text{albumin}])(0.8)$
- **Corrected sodium (if Glc > 200 mg/dL)**
 - for every 100 mg/dL of Glc, add 1.6 to Na
- **Free water**
 - $(0.6)(\text{body weight})[(\text{[Na]}/145) - 145]$



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



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



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. Salicylates



CASE

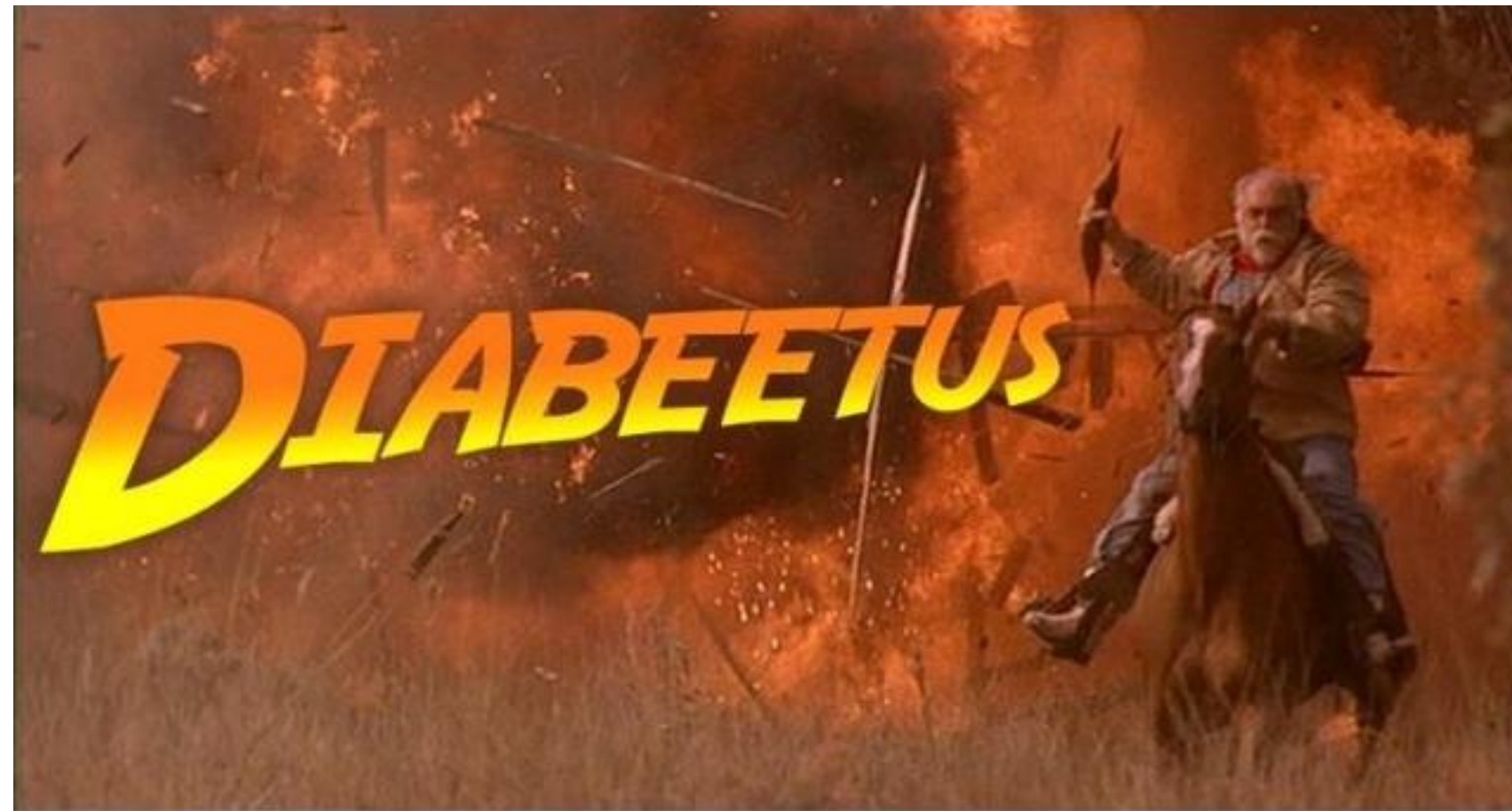
- 13yo male presents with nausea, vomiting, and diffuse abdominal pain. Has been having cold for the past week and started having symptoms this morning. Hx/o noncompliance with his medications.



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 β - cell destruction results in insulin deficiency
- 5% mortality
- Often associated with other autoimmune disorders: thyroid's, Addison's, celiac's, vitiligo



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.

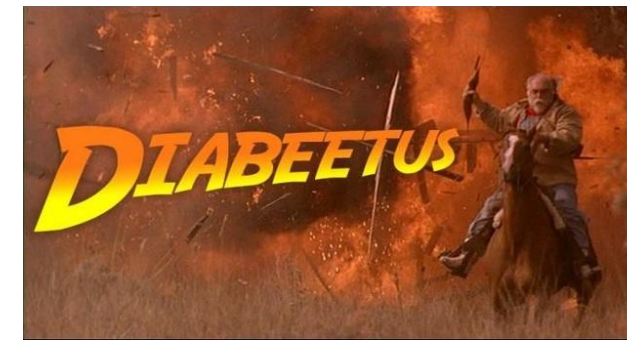


DIABETES MELLITUS

- **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



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

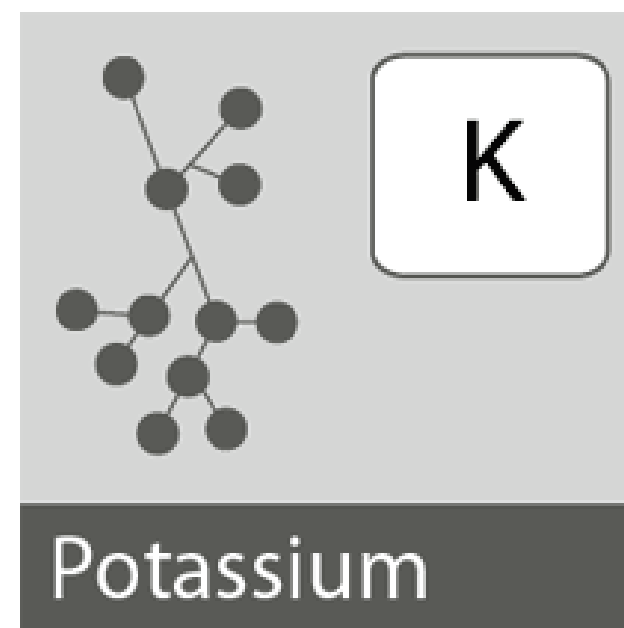
- **Electrolyte changes**
 - **Glucose**
 - Higher concentrations if dehydrated
 - **Metabolic acidosis**
 - Increased anion gap
 - Ketone production
 - Acetone, acetoacetate and BHBT (β -hydroxybutyrate)
 - **Serum sodium**
 - Decreased due to fluid movement into intravascular space
 - Formula?
 - if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



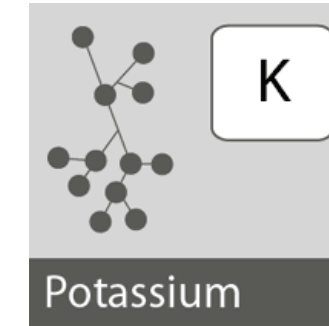
DKA

- **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





DKA TREATMENT



- FLUIDS! May require up to 6-8 L NS
- INSULIN: **BOLUS or not** = 0.1 units/kg; **DRIP** = 0.1 units/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



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 CO_2
 - 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.



Serum osmolality HIGH

pH normal

Glucose 1123

TSH normal



Urine NO ketones, 3+ glucose



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



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





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

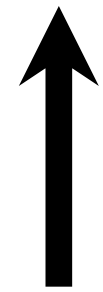




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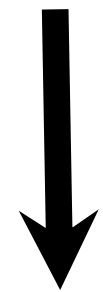


RESPIRATORY OR METABOLIC?



CO₂

Respiratory



CO₂

Metabolic



JUST A REVIEW!

- Metabolic acidosis
 - $\text{PaCO}_2 = \text{last two numbers in pH (if pH} > 7)$
 - $\text{PaCO}_2 = 1.5 [\text{HCO}_3] + 8 (\pm 2)$
 - normal anion gap/hyperchloremic
 - occurs from a loss of HCO_3
 - 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



NON ANION GAP METABOLIC ACIDOSIS

- Hyperchloremic metabolic acidosis
- Caused by
 - Loss of HCO_3
 - 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: NH_4Cl
 - Hypoaldosteronism
- The loss of HCO_3 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 $\text{HCO}_3^- > 45$, 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 mineralocorticoids: licorice, fludrocortisone
 - Compensatory reduction in alveolar ventilation
 - Posthypercapnia – if chronic



RESPIRATORY ACIDOSIS

- Acute
 - HCO_3^- increases 1 mEq/L for every 10 mmHg increase in pCO_2
 - pH drops 0.08 for every 10 mmHg increase of pCO_2
- Chronic
 - HCO_3^- increases 4 mEq/L for every 10 mmHg in pCO_2



RESPIRATORY ALKALOSIS

- Increased minute ventilation
- Acute
 - HCO_3^- drops 1 to 3.5 meq/L for every 10 mmHg drop in pCO_2
 - 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)
 - HCO_3^- drops 2 to 5 meq/L for every 10 mmHg drop in pCO_2
 - 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



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



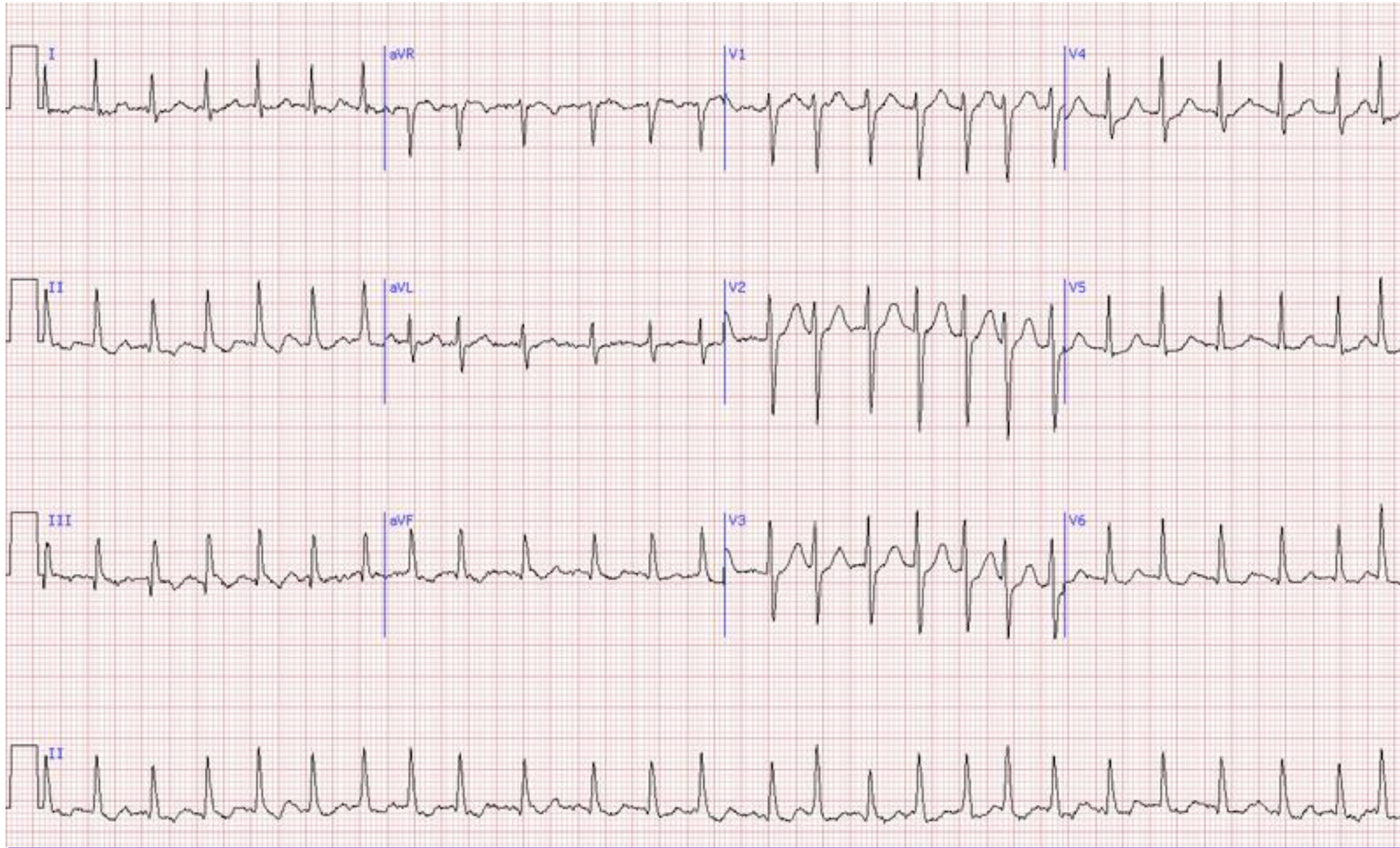
- Clinical characteristics of myxedema coma include all of the following except:
- a. Pseudomyotonic reflexes
- b. Hypothermia
- c. Hypotension
- d. Alopecia
- e. Tachycardia



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.





Temp 104F

HR 145



BP 156 / 60

Delirium

Hx/o Grave's dx

Denies any drug abuse



THYROID STORM

Beta Blockers (propranolol)



PTU



1 hr

Iodide



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.

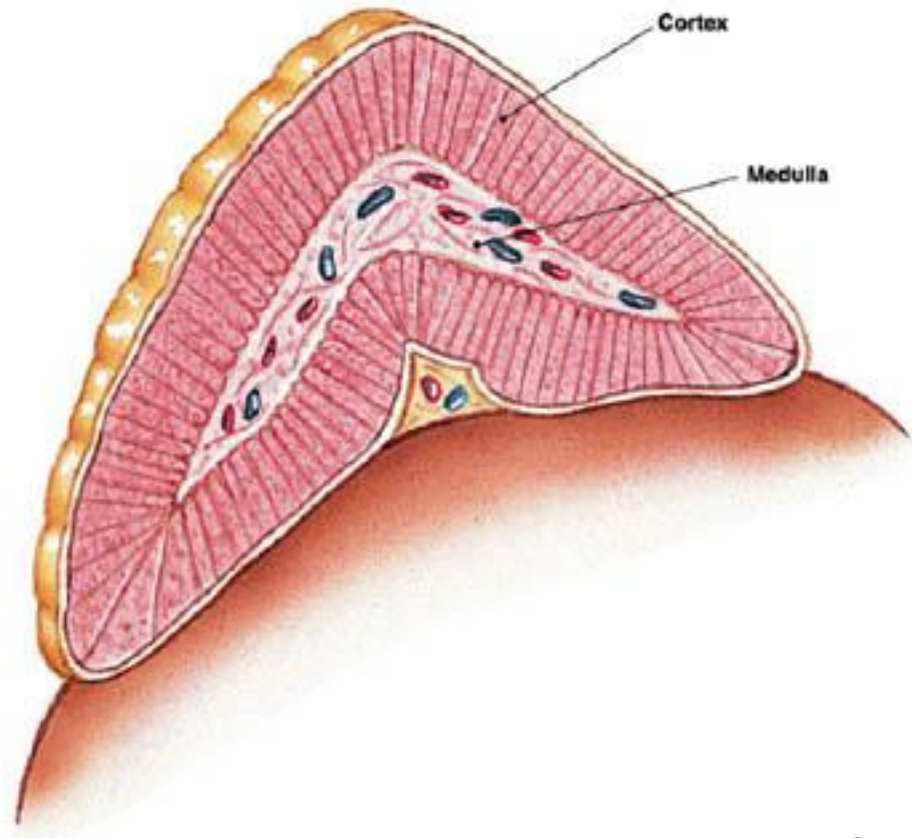


CASE

- 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.







Glucocorticoid (CORTISOL)



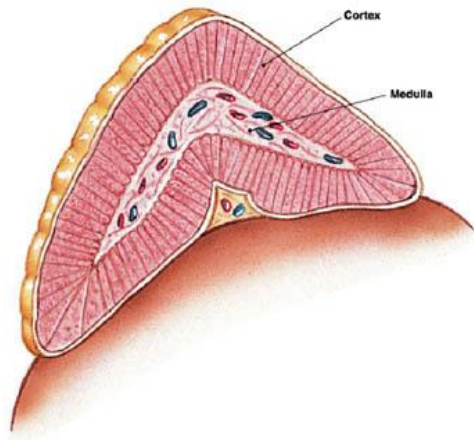
Adrenal insufficiency

Mineralocorticoid (aldosterone)



Hyponatremia / Hyperkalemia

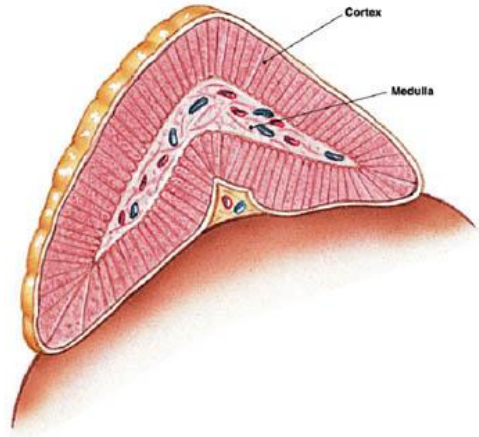




ADRENAL CRISIS

- 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)



DKA MANAGEMENT SEQUENCE

- Fluids first
- Check potassium
- Start insulin
- Monitor anion gap



ENDOCRINE POLL

- First step in DKA?
- A. Insulin
- B. Fluids
- C. Bicarbonate
- D. Potassium



ELECTROLYTE DISTURBANCES

- Hyperkalemia ECG changes
- Hyponatremia correction rate
- Hypercalcemia crisis
- Magnesium disorders



TEACHING PEARL

- Electrolytes can kill faster than infections.



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.



DIALYSIS PATIENT CODE

=

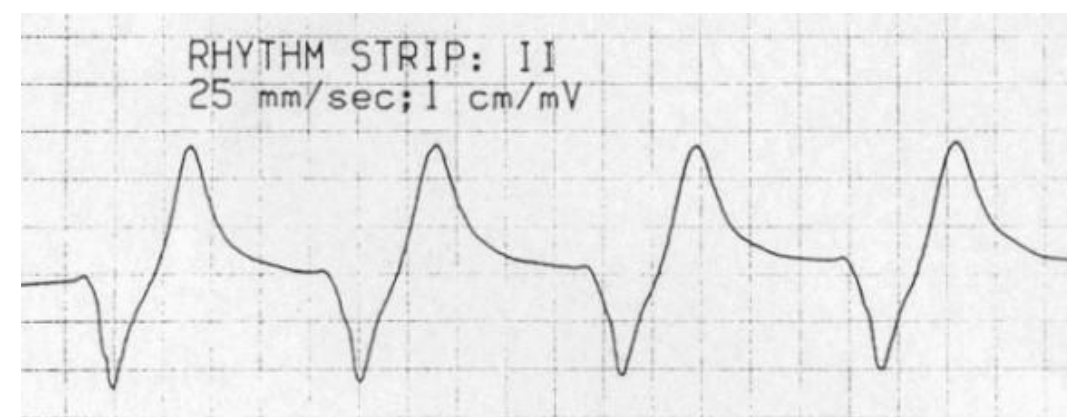
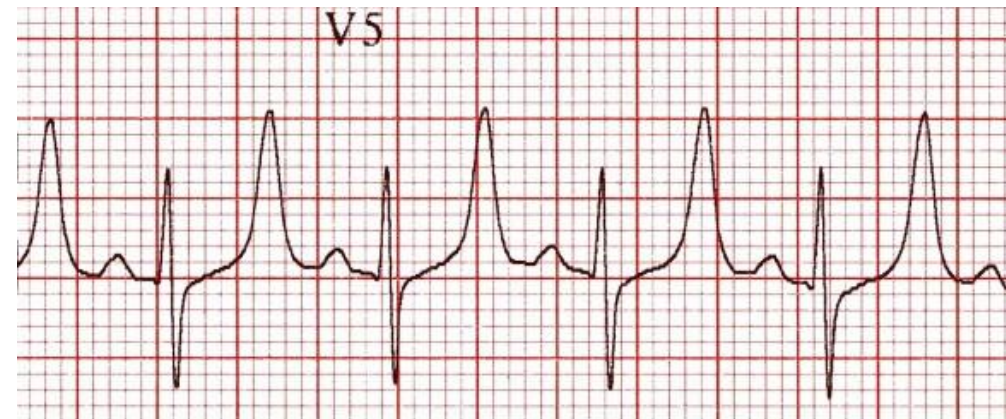
HYPERKALEMIA

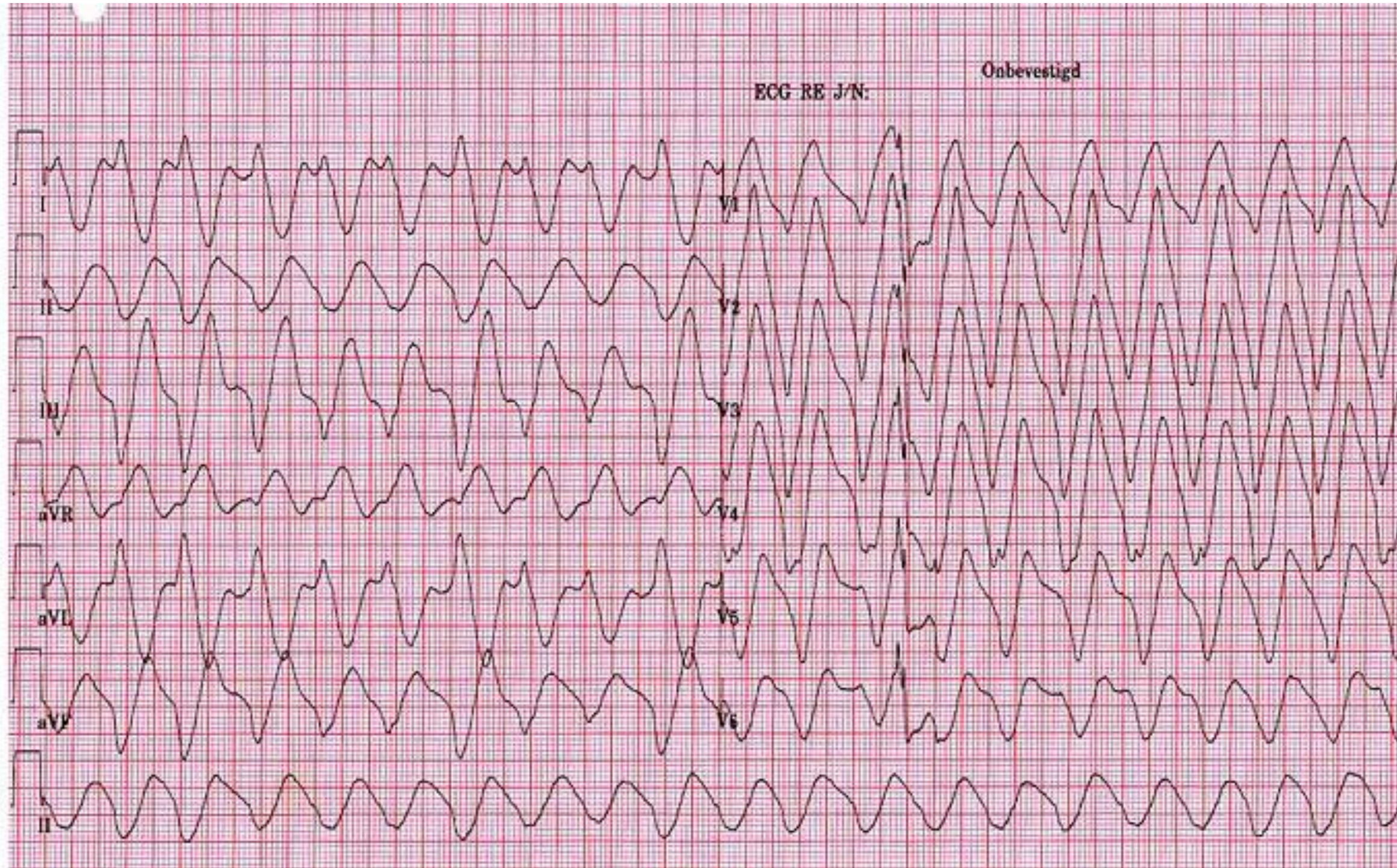
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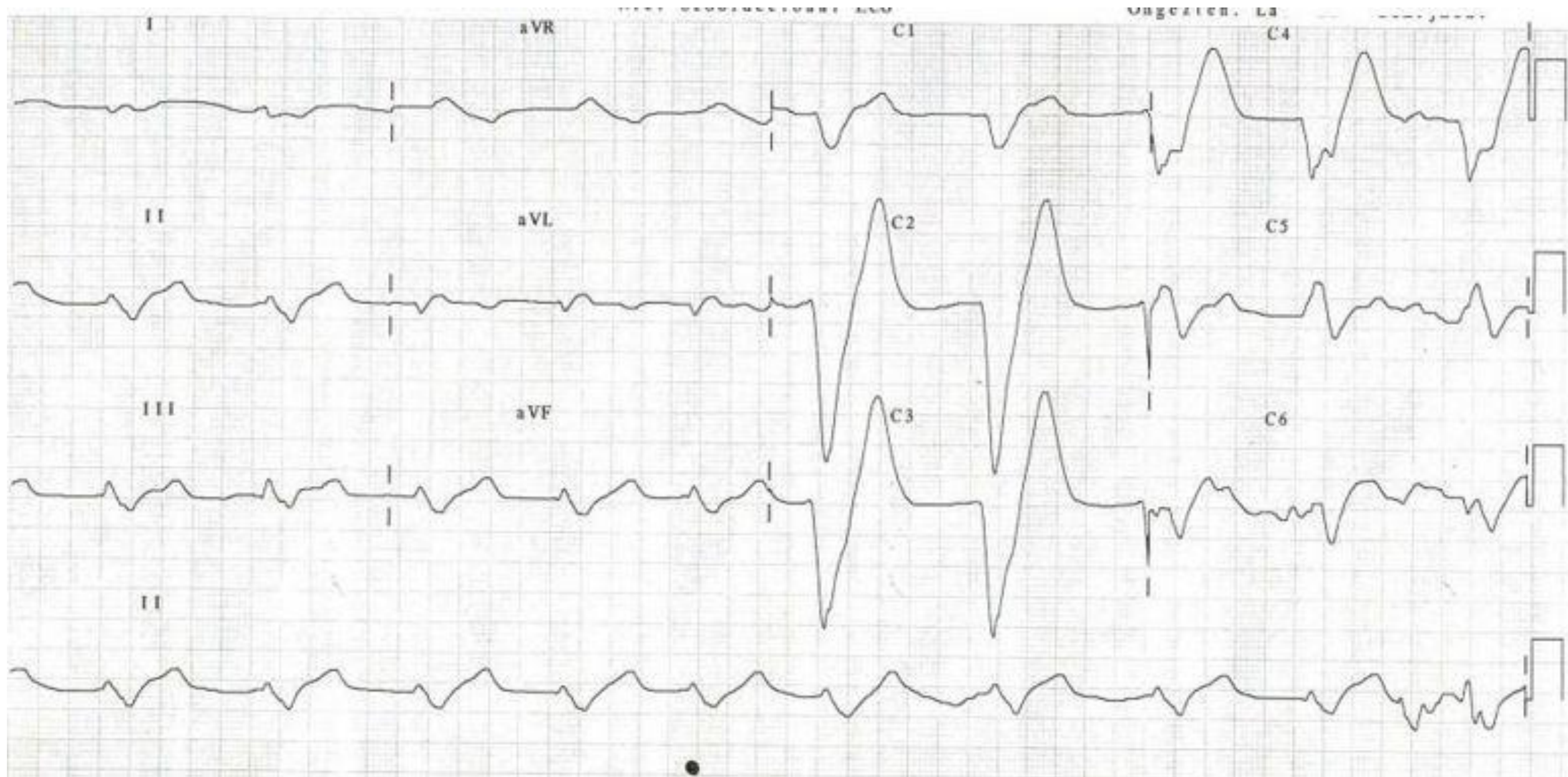
CALCIUM / BICARB

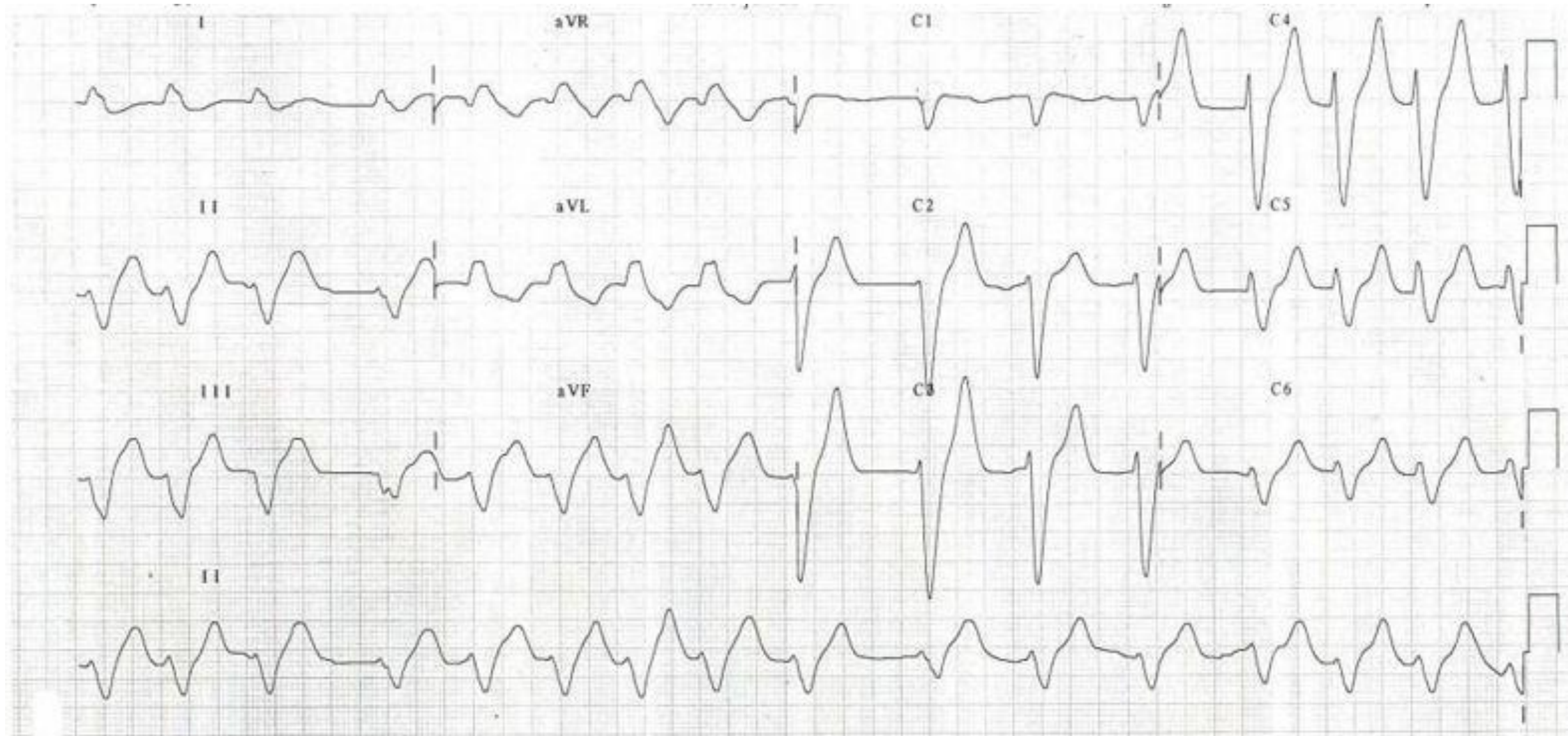


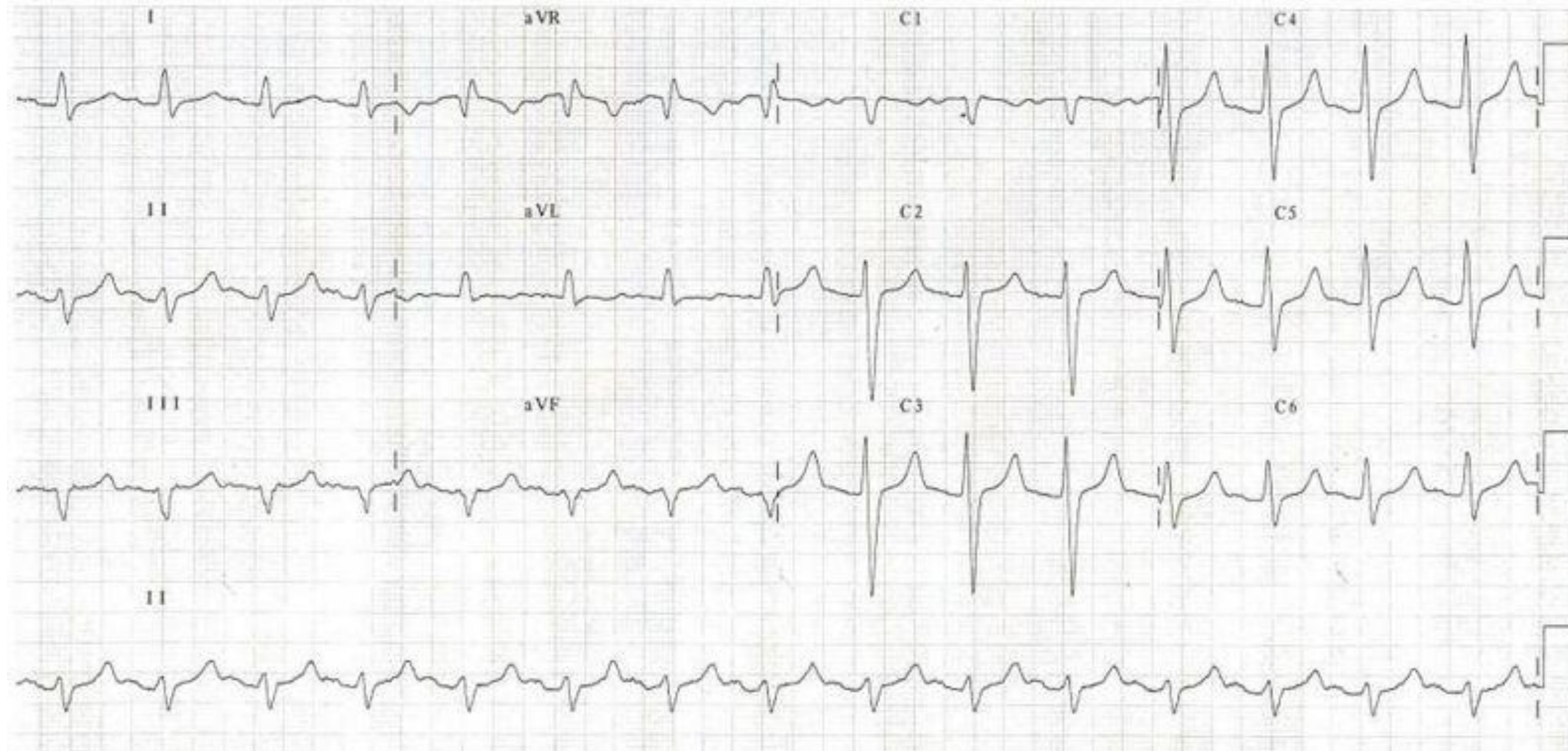
HYPERKALEMIA











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. NaHCO_3 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, β -agonists, and aldosterone by cellular reuptake
- Hypercalcemia
- **Familial periodic paralysis**: weakness, paralysis, family hx. DTRs present.
- Can be associated with $\downarrow\text{Mg}^{+2}$



HYPOKALEMIA

- Symptoms and signs
 - Muscular weakness/cramps, fatigue, constipation, flaccid paralysis, hyporeflexia, hypercapnia, 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



HR 74 PVC 0 RESP 18 T1 37.8

I 1 mV

aVR

V1

V4

II

aVL

V2

V5

III

aVF

V3

V6

II

12-Lead ECG (EASI Derived) Bandwidth: 0.50-20.0 Hz

22/06/03 18:56:41

10 mm/mV 25.0 mm/s



- 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



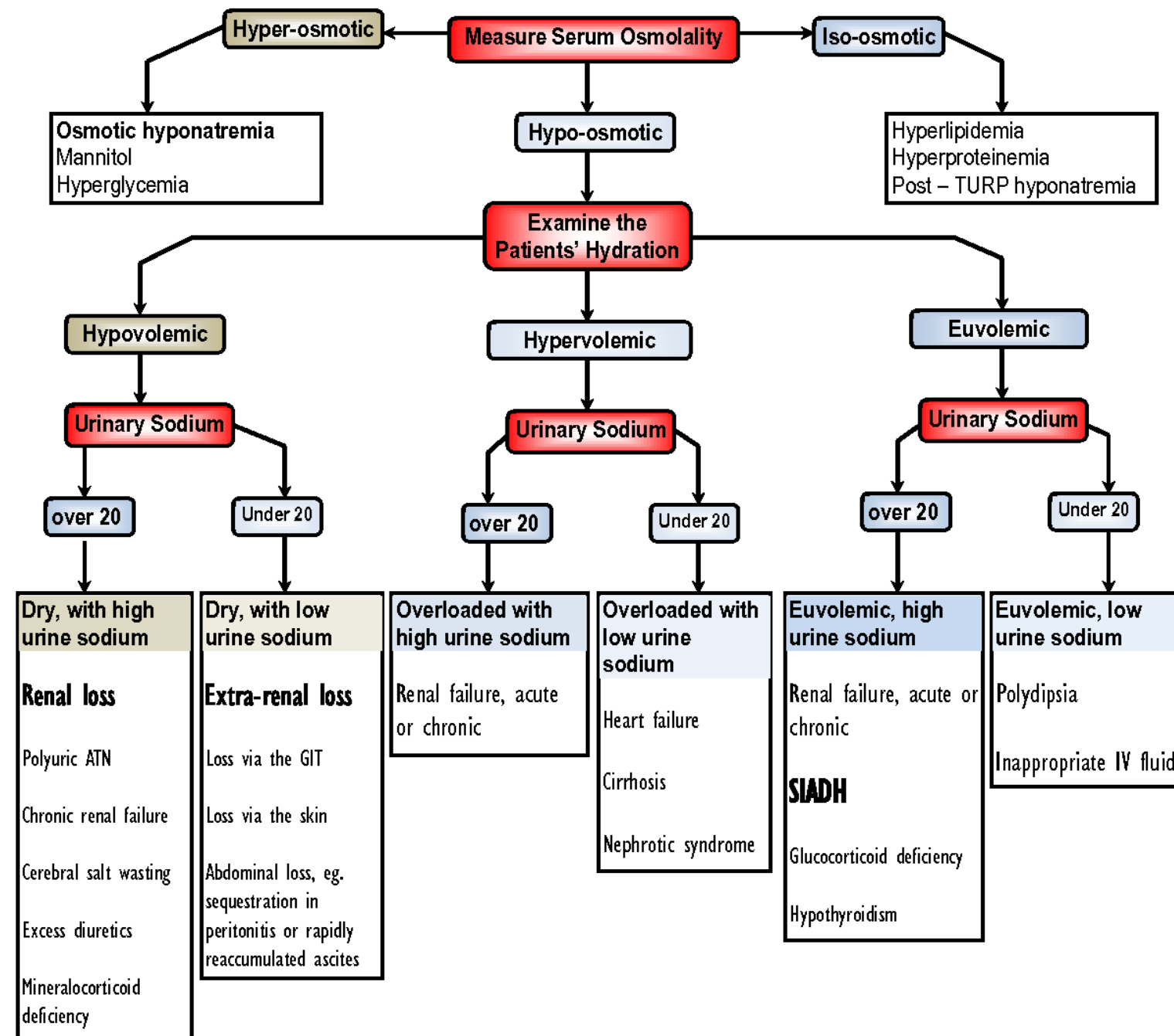
HYPONATREMIA

- **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.



HYPONATREMIA

Hypovolemic



Sodium loss from V/D
diuretics (**HCTZ**)

Hypervolemic



CHF, cirrhosis, nephrotic
syndrome

Euvolemic



Psychogenic polydipsia
SIADH



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



HYPERTONIC SALINE?

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



HYPERNATREMIA

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



HYPERCALCEMIA

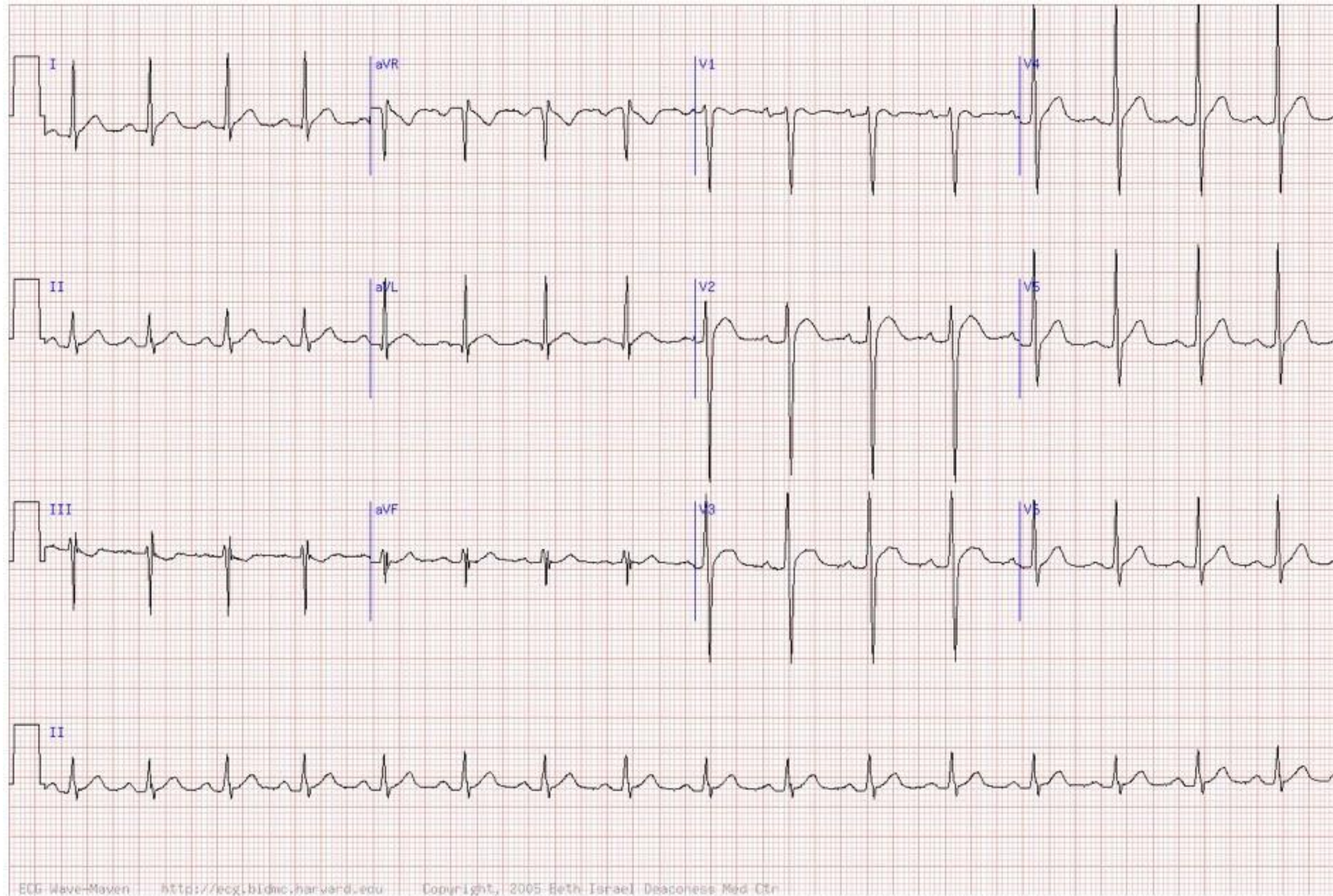
- Stones, Bones, Thrones, Groans, Psychiatric Overtones
- MCC: hyperparathyroidism, **cancer**
- AMS, confusion, Polyuria and constipation
- EKG
 - Shortened QT
 - Ventricular extrasystole
 - Idioventricular rhythm
- Tx: **HYDRATION**
 - LOOP DIURETIC, **NOT HCTZ** (increases)
 - Biphosphonate (pamidronate)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



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





HYPOCALCEMIA

- Causes:
 - Hypoparathyroidism
 - Hypomagnesimia (EtOH)
 - Hyperphosphatemia
 - Rhabdomyolysis
 - Massive blood transfusion
 - ARF/CRF due to defect in the conversion 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

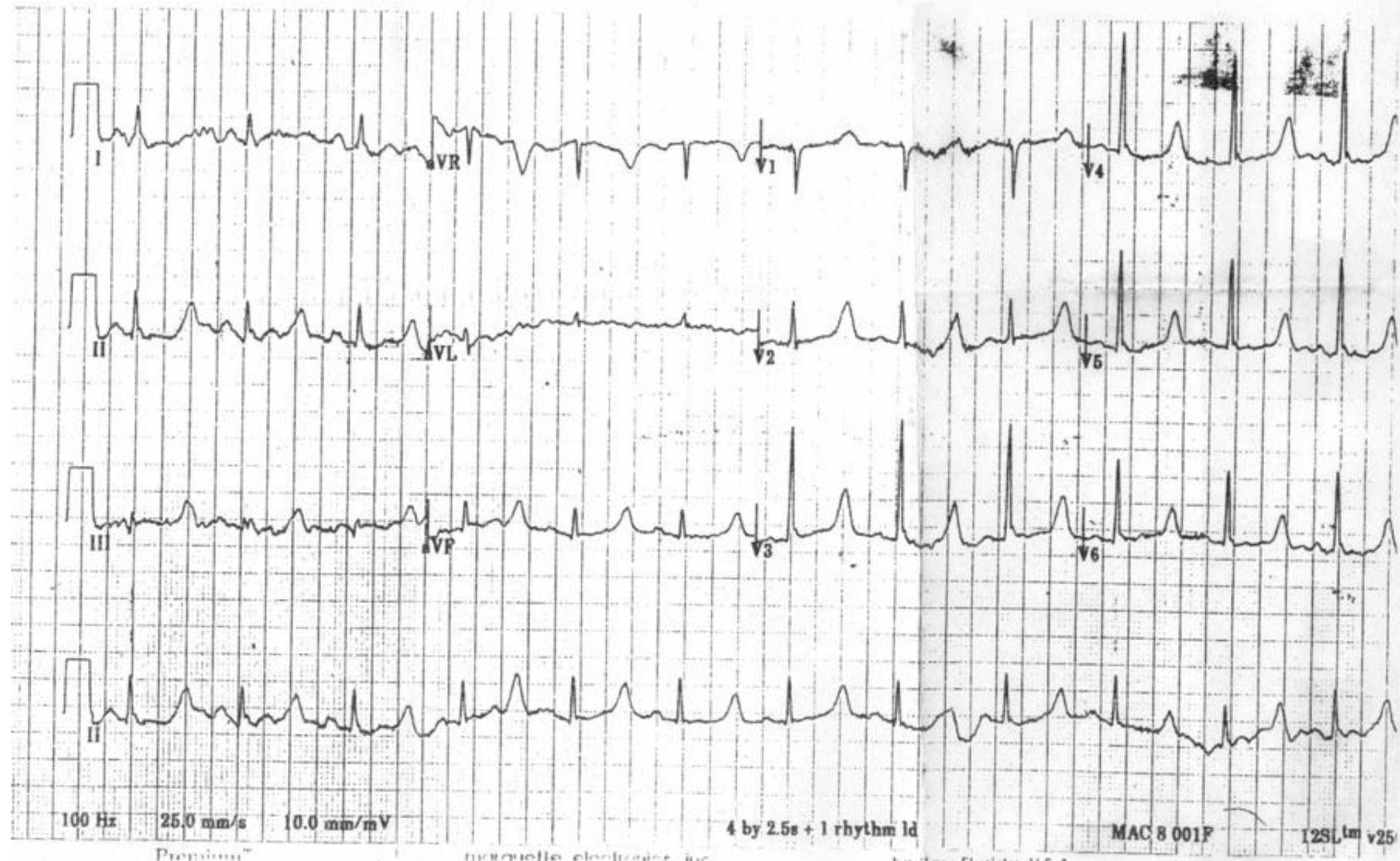
- EKG
 - QT prolongation
 - ST prolongation
- Treatment
 - Thiazide diuretics
 - Ca gluconate
 - Correction of Mg^{+2}



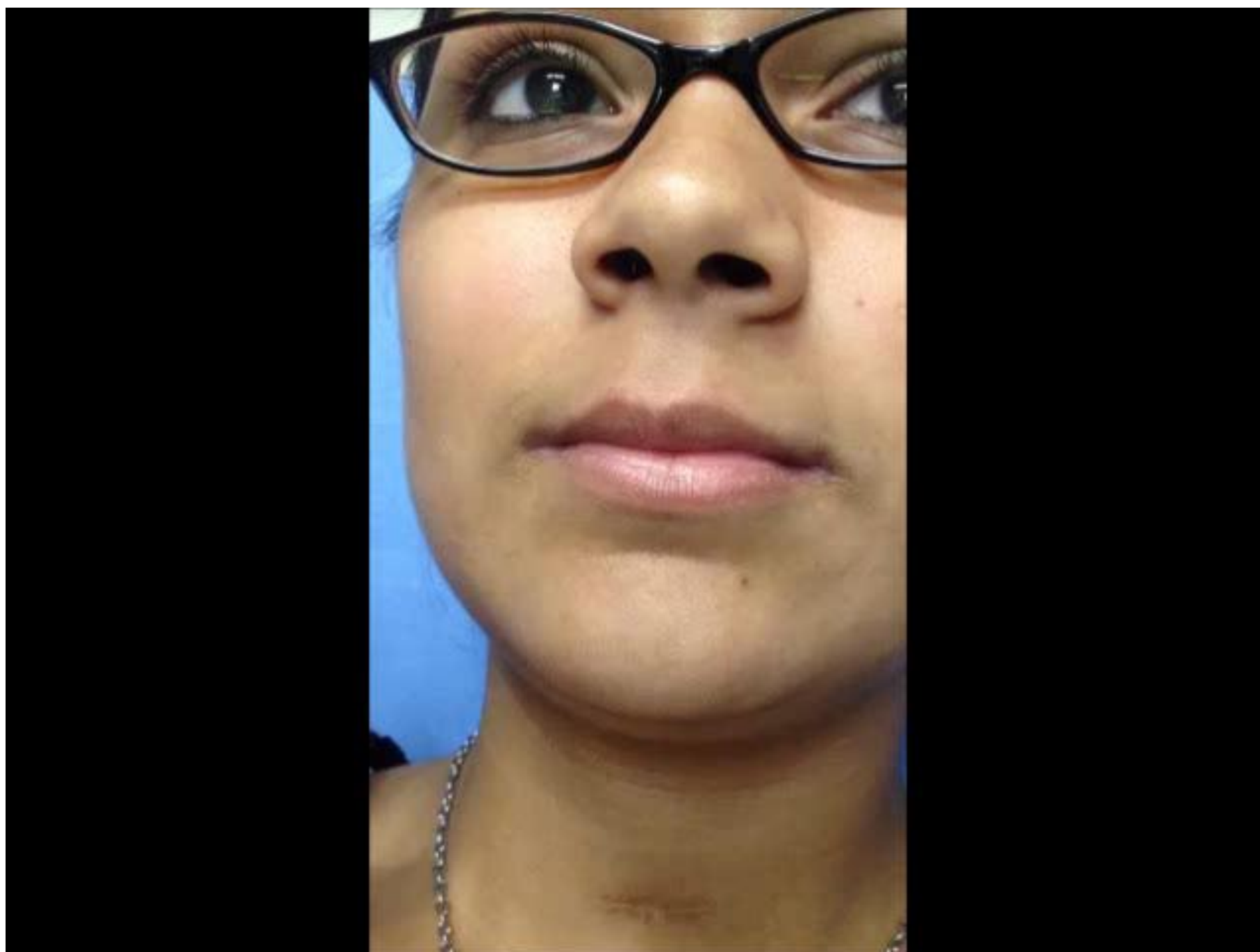
Male Caucasian

Vent. rate 78 bpm
PR interval 168 ms
QRS duration 82 ms
QT/QTc 586/668 ms
P-R-T axes 34 41 64

Normal sinus : im with occasi
Prolonged QT
Abnormal ECG







HYPOMAGNESEMIA

- **Malnutrition**, alcoholism, DKA
 - Hypokalemia, hyponatremia, hypocalcemia
 - Renal wasting
 - Diuretics, gentamycin, cisplatin, amphotericin 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

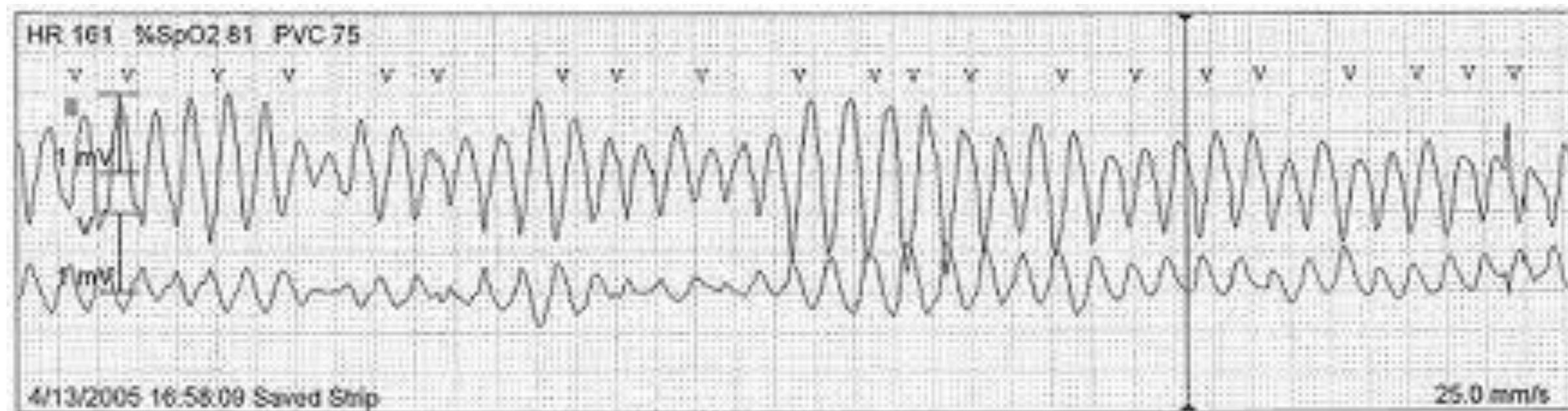
- EKG

- Prolonged QT
- Peaked T
- Depressed ST
- Torsades de pointe

- Treatment

- IV/PO supplement
- Correction of electrolytes





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



QT PROLONGATION

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

