PULMONARY EMBOLISM

- Obstruction of a branch of the pulmonary artery by a blood-borne substance.
- 50 – 100k deaths per year in the U.S.
- Mortality rate is high – 15 – 17.5 %
- Can occur with:
  1) A Thrombus.
  2) Air.
  3) Fat.
  4) Amniotic fluid
PULMONARY EMBOLISM

- Almost all arise from DVT.
- PE is often the 1st indication of DVT.
- Risk factors for PE are the same as for DVT:
  - Virchow’s Triad:
    - 1) Venous stasis.
    - 2) Endothelial injury.
    - 3) Hypercoagulability.
PULMONARY EMBOLISM

VENOUS STASIS & ENDOThelial INJURY

- Prolonged bed rest.
- Trauma, surgery.
- Childbirth.
- Hip / femur fractures.
- MI, CHF.
- Spinal cord injury.
PULMONARY EMBOLISM

- Also at increased risk: OCP’s, ERT, pregnancy, esp. pregnancy with surgery- C-section, etc.
- OCP’s and smoking, esp after age 35- 3X the risk as non-users of OCP’s.
PULMONARY EMBOLISM

THE RESULT

1) Mechanical obstruction of the pulmonary circulation.
2) Neurohumoral reflexes $\rightarrow$ vasoconstriction.
3) Bronchoconstriction $\rightarrow$ impaired gas exchange.
4) If massive PE $\rightarrow$ pulmonary hypertension $\rightarrow$ right heart failure; pulmonary infarction (uncommon).
PULMONARY EMBOLISM

MANIFESTATIONS

- **If Mild** → Chest pain, dyspnea, ↑ resp rate. Hypoxemia without hypercapnea.
- **If moderate** → SOB, pleuritic chest pain, apprehension, fever, hemoptysis. Tachycardia, tachypnea that is shallow.
- **If massive** → collapse, shock, hypoxia w/ cyanosis, tachycardia, death.
O2 AND CO2

- **HYPOXIA** - reduced O2 in the tissues. Expressed as PO2 or PaO2.

- **HYPOXEMIA** - reduced O2 in the blood. Expressed as PCO2 or PaCO2.

- **HYPERCAPNEA** = **HYPERCARBIA** = increased / excess CO2 in the blood.
HYPERVENTILATION SYNDROME

- Chapter 34, Pg 806.
- Couldn’t find 4 causes. Most common cause is anxiety. I’m wondering if the syllabus meant hypoventilation.
HYPOVENTILATION

4 CAUSES

1) Depression of the respiratory center (narcotics).
2) Neurologic Disorders affecting the muscles of respiration (Guillan-Barre Syndrome).
3) Disorders of the respiratory muscles (M.D.).
4) Thoracic cage disorders- trauma, scoliosis.
HYPOVENTILATION

**MANIFESTATIONS**

- Increased PCO2.
- ↓ ventilation by ½ → ↑ PCO2 by 2X.
- Hypoxemia- less so- ↑ PCO2 by 2X from 40 mm Hg to 80 mm Hg → ↓O2 from 100 to 60 mm Hg.
- The hypoxia can be reversed by increasing the % of inspired O2.
ELECTROLYTE DISORDERS
REGULATION OF Na+ BALANCE

- Most Na+ is in the ECF, small amount in the ICF
- Pumped out of the cell by Na+/K+ ATP-ase pump.
- Functions to regulate extracellular and intravascular volume. “As Na+ goes, so goes H2O.”
- Along w/ Cl⁻ and HCO₃⁻, accounts for 90-95% of the osmotic pressure in the ECF (not the colloidal osmotic pressure; albumin does this).
- Regulates pH via sodium bicarb.
REGULATION OF Na+ BALANCE

- Eliminated (and reabsorbed via renin-angiotensin-aldosterone) by the kidney.
- Filtered by the glomerulus, and excreted or reabsorbed according to: #1) GFR (reg by the A.N.S.), and #2) aldosterone (causes Na+ reabsorption).
- 10% eliminated via GI tract and skin.
- Lost via: vomiting, perspiration.
REGULATION OF Na+ BALANCE

THE BIG PICTURE

- THE CONTROL OF Na+ IS REGULATED BY CHANGES IN:
  - 1) GFR- GLOMERULAR FILTRATION RATE.
  - 2) ALDOSTERONE SECRETION AND ANGIOTENSIN II.
REGULATION OF WATER BALANCE

BY:

1) THIRST.

2) ADH / VASOPRESSIN.
HYPONATREMIA

- See text.
- Most common form is hypotonic (dilutional) hyponatremia.
- 3 Flavors:
  - 1) Hypervolemic.
  - 2) Euvolemic.
  - 3) Hypervolemic.
HYponatREMIA

HyperVolemIC HYponATREMIA

EuvolemIC hyponATREMIA
- SIADH, psychogenic water intoxication.

HyPovolemIC HYponATREMIA
- Diuretics, sweating, vomiting / diarrhea. H2O is lost along w/ Na+. 
MANIFESTATIONS OF HYPONATREMIA

- Depends on the degree and the rapidity of onset.
- “Cellular swelling.” (Water moves from ECF to ICF compartment)
- Muscle cramps, weakness, fatigue.
- N/V, abdominal cramps.
- CNS: lethargy, headache → motor weakness, confusion → seizures, coma.
HYPERNATREMIA

- Hypernatremia → hypertonicity of the ECF → draws water out of the cells → “cellular dehydration.”
HYPERNATREMIA

- Caused by:

  1) **NET WATER LOSS**: by far the most common; loss of Na+ - containing fluid/water, w/ disproportionate loss of H2O: urine, GI tract, skin, lungs (w/ ↑ resp rate seen in fever) and also from ↓ intake.

  2) **SODIUM GAIN**: infusion (IV, iatrogenic), PO intake.
MANIFESTATIONS OF HYPERNATREMIA

- Again, depends on severity and rapidity of onset.
- Remember, if H20 is lost, other stuff becomes concentrated too, such as the Hct, BUN, etc.
- When mild: decreased urine output, dry mucous membranes, decreased tissue turgor.
- When severe: decrease in DTR’s, agitation, headache → seizures, coma.
REGULATION OF POTASSIUM BALANCE

- The major cation of the ICF.
- 98% is intracellular, as compared to Na+ which is mainly extracellular.
- Filtered in the glomerulus.
- Like Na+, it is reabsorbed (w/ H2O) from the proximal renal tubule; but, unlike Na+, it is also secreted into the distal tubule under the influence of aldosterone. Corticosteroids (cortisol) also have this mineralocorticoid effect.
POTASSIUM

FUNCTIONS

- 1) Osmotic integrity of the cells.
- 2) Acid-base balance.
- 3) Various chemical reactions, glucose $\rightarrow$ glycogen, amino acids $\rightarrow$ proteins.
- 4) Nerve impulses.
- 5) Excitability of cardiac, skeletal, and smooth muscle.
- Sensitive to minor changes in serum K+.
REGULATION OF POTASSIUM BALANCE

REGULATED BY

1) Renal reabsorption (proximal tubule) or excretion (distal tubule via aldosterone).

2) Transcellular shifts from the intracellular to the extracellular compartment. See text.
POTASSIUM AND THE TUBULE

- Under the influence of aldosterone, Na+ is reabsorbed and K+ is secreted via the Na+/K+ ATP-ase pump.
- As well, K+ is exchanged for H+, contributing to the regulation, or disruption, of pH.
- Pg. 767.
HYPOKALEMIA

CAUSED BY

■ 1) Inadequate intake.
■ 2) Excessive loss.
■ 3) Redistribution between the ICF and the ECF.
HYPOKALEMIA

INADEQUATE INTAKE
- The elderly, fad diets, eating disorders.

EXCESSIVE LOSSES
- Urine, stool, skin.
- Diuretics- the most common cause.
- Increased aldosterone (Conn’s Syndrome) and cortisol (Cushing’s), Mg+ deficiency, licorice (inhibits metabolism of cortisol).
- Excessive sweating, burns, vomiting, diarrhea.
HYPOKALEMIA

TRANSCELLULAR SHIFTS

- Primarily drugs: beta agonists: theophylline and albuterol for asthma, pseudoephedrine; also insulin: causes movement of not only glucose but K+ into cells.
MANIFESTATIONS OF HYPOKALEMIA

- When mild- GI Sx’s- anorexia, N/V, atony of GI smooth muscle → constipation, paralytic ileus.
- When moderate- skeletal muscle dysfunction- weakness, fatigue, muscle cramps esp w/ exercise esp in the quadriceps.
- When severe- cardiovascular- arrhythmias, hypotension
HYPERKALEMIA

CAUSED BY

1) DECREASED RENAL ELIMINATION.
2) EXCESSIVE ADMINISTRATION.
3) MOVEMENT FROM ICF INTO ECF.
HYPERKALEMIA

DECREASED RENAL ELIMINATION

- Most common cause of hyperkalemia.
- Renal failure.
- Addison’s disease (aldosterone deficiency).
- ACE inhibitors.

EXCESSIVE ADMINISTRATION

- PO, IV. Oral route unlikely to cause hyperkalemia in the presence of normal renal function unless the patient is also on a potassium-sparing diuretic.
HYPERKALEMIA

MOVEMENT FROM ICF TO ECF

- Examples: Trauma, burns, extreme exercise, seizures, hemolysis as in hemolytic anemia.
- The common thread is that cells are disrupted and the intracellular K+ is released into the serum.
REGULATION OF CALCIUM, PHOSPHATE, AND MAGNESIUM
Frank and Ernest

Pharmacy

IT'S NOT EXACTLY A GENERIC... THE MANUFACTURER JUST PREFERENCES TO REMAIN ANONYMOUS.
CALCIUM

- Absorbed from the GI tract in the presence of vitamin D.
- Stored in bone, 99%; 0.9% in cells, 0.1% in ECF
- Eliminated by the kidney.

3 FORMS:

1) Protein-bound- can not leave the vascular system.
3) Ionized- leaves the vascular space → cellular function (See next slide).
CALCIUM

IONIZED CALCIUM

- CELLULAR FUNCTIONS SUCH AS:
  - Enzymatic reactions, coagulation.
  - Membrane potential for muscle contraction.
  - Release of hormones, neurotransmitters.
CALCIUM (A REVIEW, RIGHT?)

- **VITAMIN D** - required for absorption of Ca++ from the GI tract.
- **PTH** - removes Ca++ from bone during bone remodeling; stimulates reabsorption of Ca++ from the distal tubule.
- **CALCITONIN** - drives Ca++ into bone during remodeling.
CAUSES OF HYPOCALCEMIA

1) IMPAIRED ABILITY TO MOBILIZE Ca++ FROM BONE:
   - HYPOPARATHYROIDISM.
   - HYPOMAGNESEMIA- Mg++ deficiency inhibits PTH release, impairs action of PTH on bone.

2) DECREASED INTAKE OR ABSORPTION OF Ca++: malabsorption; failure to convert Vitamin D to active form in the liver and kidneys- see fig 56-4.
CAUSES OF HYPOCALCEMIA

- 3) Abnormal renal loss - renal failure.
- 4) Increased chelation or protein binding - renders Ca++ in the non-ionized form - ↑ pH, ↑ free fatty acids, alcohol.
- 5) Sequestration - in the pancreas in acute pancreatitis - Ca++ combines with free fatty acids resulting from lipolysis of the pancreas as it self-destructs.
MANIFESTATIONS OF HYPOCALCEMIA

- Increased neuromuscular excitability.
- Sensory and/or motor.
- Paresthesias- tingling, numbness, esp the mouth, hands, and feet.
- Tetany- spasms, esp of the face, hands, and feet. Chvostek’s Sign. Trousseau’s Sign.
- Acute- arrhythmias
- Chronic- skeletal, skin changes.
CAUSES OF HYPERCALCEMIA

- 90% DUE TO INCREASED RESORPTION FROM BONE, DUE TO:
- 1) HYPERPARATHYROIDISM.
- 2) MALIGNANCY.
- OTHERS: prolonged bed-rest, excess Vitamin D, increased absorption, drugs: lithium, thiazide diuretics.
MANIFESTATIONS OF HYPERCALCEMIA

1) DECREASED NEUROMUSCULAR EXCITABILITY- weakness, fatigue, CNS/behavioral changes.

2) CARDIAC FUNCTION- ventricular dysrhythmias.

3) GI- constipation, N / V, pancreatitis from deposition of Ca++ stones in the ducts.

4) RENAL- stones, interference w/ ADH → polyuria, polydipsia.
PHOSPHATE – PO$_4^{2-}$

- **Phosphorus**: Intracellular anion.
- 4$^{th}$ most abundant element behind carbon, nitrogen, and calcium.
- **Involved in:**
  - Bone formation, metabolic processes such as ATP formation, enzymatic reactions, phospholipids of cell membranes, incorporation into nucleic acids, acid-base buffer.
  - 85% in bone. 14% in cells. 1% in the ECF.
PHOSPHATE

- Absorbed through the GI tract, inhibited by calcium, magnesium, aluminum (Maalox = magnesium and aluminum hydroxide).
- Eliminated by the kidney; glomerular filtration, tubular reabsorption.
HYPOPHOSPHATEMIA

CAUSED BY

1) INSUFFICIENT ABSORPTION- glucocorticoids, antacids containing aluminum hydroxide, aluminum carbonate, calcium carbonate (Maalox); alcoholism.

2) INCREASED RENAL EXCRETION- drugs: theophylline, corticosteroids, diuretics.

3) TRANSCOMPARTMENTAL SHIFTS- glucose administration, insulin shift phosphate into cells
MANIFESTATION OF HYPOPHOSPHATEMIA

- The result of decreased ATP production.
- Red and white cell dysfunction.
- Neural manifestations- tremors, hyporeflexia, stupor, seizures, dysphagia, weakness.
- Impaired bone mineralization.
HYPERPHOSPHATEMIA

- **CAUSES**

  1) Failure of the kidneys to excrete phosphate—most common; chronic renal failure.

  2) Redistribution of phosphate from the ICF to the ECF—massive tissue injury, rhabdomyolysis, heat stroke, seizures.

  3) Excessive intake of phosphate—antacids, laxatives, enemas (Fleets Phospho-Soda)
MANIFESTATION OF HYPERPHOSPHATEMIA

- Excess phosphate combines w/ calcium resulting in hypocalcemia.
- As such the manifestations of hyperphosphatemia are those of hypocalcemia.
MAGNESIUM

- 2ND most common intracellular cation, behind Na+.
- 50-60% in bone, 39-49% in cells, 1% in the ECF.
- 20-30% protein-bound.
- Cofactor in enzymatic reactions.
- Essential for: ATP production, replication and transcription of DNA, translation of messenger RNA, Na / K pump, membrane stabilization, nerve conduction, ion transport, calcium channel activity.
MAGNESIUM

- Absorbed from the GI tract, excreted by the kidneys.
HYPMAGNENESEMIA

CAUSES

1) Decreased intake- malnutrition, prolonged parenteral nutrition w/out Mg replacement, malabsorption, diarrhea, NG suction, laxative abuse. Ca++ competes w/ Mg for absorption. Alcoholism. Other electrolyte disorders.

2) Excess renal excretion- DKA, hyperparathyroidism, hyperaldosteronism, diuretics, nephrotoxic drugs.
HYPERMAGNESEMIA

CAUSES

1) RENAL INSUFFICIENCY

2) MAGNESIUM-CONTAINING MEDICATION-antacids, mineral supplements, laxatives.

- Elderly most susceptible due to age-related ↓ in renal function and ↑ use of Mg-containing meds.
HYPERMAGNESEMIA

MANIFESTATIONS

- NEUROMUSCULAR- ↓ release of acetylcholine at the neuromuscular junction → muscle weakness, hyporeflexia, respiratory depression, paralysis.

- CARDIOVASCULAR- blocks the calcium channels → arrhythmias, hypotension, cardiac arrest.
DISORDERS OF RENAL FUNCTION

CHAPTER 35
EXAM ROOM

TAKE ONE OF THESE PILLS ON SUNDAY WHEN I'M OUT OF MY OFFICE.

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POLYCYSTIC KIDNEY DISEASE

- Common.
- Genetic- autosomal recessive and autosomal dominant forms of inheritance.
- Cystic formation within the renal tubule → tubular obstruction, compression of renal blood flow.
- Results in renal failure, hypertension.
- Often associated w/ other anomalies- aneurysms, cysts in the liver, pancreas.
POLYCYSTIC KIDNEY DISEASE

MANIFESTATIONS

- Abdominal, flank pain.
- Hematuria, stone formation.
- Hypertension.
- Palpable abdominal mass; CT, MRI for imaging.
URINARY OBSTRUCTION

CAUSES

- Congenital malformations.
- Calculi.
- Tumors, pregnancy, strictures.
- Benign prostatic hypertrophy (BPH).
- Surgical- hysterectomy, adnexectomy.
URINARY OBSTRUCTION

RESULTS IN

- Hydronephrosis- unilateral or bilateral, depending on location of obstruction.
- Static urine → infection → stone formation.
- If not relieved within days to a week or 2 results in renal failure from backpressure interfering w/ blood flow.
URINARY OBSTRUCTION

MANIFESTATIONS

- W/ ureteral obstruction → Fever, Flank Pain, Ileus.
- Other sites, Sx’s vary acc to the location of obstruction.
Zoo

Infirmary

I can never remember... is it "feed a toad and starve a beaver" or "feed a beaver and starve a toad"?

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