ELECTROLYTE	CAUSE	S/S	INTERVENTION	CONNECTION
HYPERNATREMIA <u>Na+</u> •135-145 mEq/L •90% of the ECF cations •Water follows salt •Helps transmit nerve impulses in nerve fibers and muscle fibers (fire, ie depolarize) •Main regulator is kidneys ADH & Aldosterone play major roles	**Renal disease (unable to excrete)** **Increased Na+ intake** •Hypertonic fluids, fast NS •Sodium bicarbonate •Hypertonic feedings **Increased water loss** • very old (loss of thirst reflex) •very young (dehydrate easily) •Cog impaired (forget to drink) •Diaphoresis •Diabetes insipidus •Hyperventilation •Diarrhea •Mechanical ventilation •Poor water intake •Thirst center injury-hypothalmus •Oral injury	S&S due to ICF dehydration (H2O shifts out of icf –cells shrink) •Thirst •Weakness, restlessness •Disorientation, delusions, hallucinations •Dry, sticky mucus membranes •Flushed skin •Postural hypotension •Increased muscle tone and deep tendon reflexes •Increased body temperature ADVANCED/SEVERE: shock, circ overload, seizures **SALT** **S**kin flushed **A**gitation **L**ow grade Fever **T**hirst	•Hypotonic IV fluids like 0.2% NS •D5W •Diuretics- increase Na+ excretion lower Na+ lvls slowly 1-2 meq/h (cuz otherwise gets too low and pt gets cerebral edema) If cz'd by diabetes insipidus (damage to hypothalmus causes ↑ urine and thirst due to deficient production or secretion of ADH) may give vasopressin (ADH)	•Combines w/ Cl- and HCO3- to reg acid-base balance •Na+ and Cl- together •Influences conc of K+ in body in kidneys if salt is retained then K+ is excreted and vice versa •K+ and H+ compete for exchange w/ Na+ •Aldosterone retains Na+, excretes K+
HYPONATREMIA ratio of Na+ to total body H2O is ↓ Can be caused by •loss of Na+ w/ less of a loss of water •normal Na+ content with an increase in total body water. •Cells swell because blood is hypotonic	**LOSS of Na+** Vomiting or Diarrhea or fistula Diaphoresis, burns Inadequate intake of Na+ •often in conjunction with diuretics Adrenal insufficiency – Addison's •↓aldosterone secretion **DILUTIONAL** hyponatremia- 2ndary to increased total body water SIADH (classic S&S: Na↓, Mental status change) Hyperglycemia Electrolyte-poor IV fluids Tap water enemas NG tube irrigation with tap water Compulsive drinking of water •Psychogenic polydipsia	CEREBRAL EDEMA (S&S: HA, N&V) Headache Confusion Nausea, vomiting Seizures. coma Muscle weakness, muscle cramping	depends on if due to loss of Na+ or dilution of ECF Sodium replacement- oral or IV Water restriction Hypertonic IV 3% NS •Only in emergency, only in ICU. Raising the serum Na+ too rapidly can cause neurological changes or permanent damage. •Lithium and Demecloycine ↓ ADH •If cerebral edema then diuretics	

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HYPERKALEMIA **K= general 3.5-5.0 mEq/L** 1° ICF cation. Kidney is 1° regulator of K+. Most of body's K+ needs met through foods; some meds K+ must be replaced qd. **Control of K+ levels** Renin-angiotens-aldosterone sys Kidneys **Functions of K+** Maintain cell fluid volume Cardiac, skeletal, and smooth muscle contraction Nerve impulse conduction Metabolism of CHO & protein Maintain/correct acid base bal **Foods containing higher levels of K+:** citrus, banana, cantalopes, prunes, raisins, squash, dried beans	In a person with a healthy kidney, usually hyperkalemia does not occur because excess K+ is excreted. Acidosis: excess H+ ions move into the cells. Because cell must maintain electroneutrality, K+ moves out of the cell Increased intake PO or IV including salt substitutes Renal failure Adrenal insufficiency- Addison's ↓aldosterone secretion Potassium sparing diuretics Cell destruction Metabolic acidosis DKA (Lack of insulin)	Muscle cramping or weakness like hypokalemia, but cause is different (from firing so much)? N&V, hyperactive bowel sounds, diarrhea Bradycardia, low blood pressure EKG changes (peaked T waves) Cardiac arrhythmias are the biggest concern. **SUCTION** **S**kelatal muscle weakness **U** wave (ekg) **C**onstipation **T**oxic effects of dig **I**rregular, weak pulse **O**rthostatic hypotention **N**umbness (parathesia)	**Goal: ↑excretion & move K+ → cells** •Treat cause •Limit intake •Kayexaylate-cation exchange resin. Binds w/ K+ and prevents absorption •K+ losing diuretics w/ IV NS •ER tx admin hypertonic D5W w/insulin •Admin HCO3- corrects acidosis •Dialysis •IV calcium gluconate (er tx, temp) (stops cardiac effect of ↑K+) •Serial ekg **C BIG KID** **C**alcium gluconate **B**icarbonate **I**nsulin **G**lucose **K**ayexalate **D**ialysis	•Aldosterone retains Na+, excretes K+ •Need Mg++ for na-k pump •K+ r/t Insulin •K+ and H+ exchange easily across cell membranes
HYPOKALEMIA	Situations that ↑urine will ↑ K+ loss. Insulin levels- ↑insulin causes K+ to shift into cells. Alkalosis: H+ ions move out of the cells, K+ moves in. ↓intake- food or IV fluid Increased excretion- renal, GI, (vomiting diarrhea, NG suction) or sweat Drugs- K+ wasting diuretics, cortisone (steroids) Hyperaldosteronism or Cushing syndrome Epinephrine infusions (shift from ECF to ICF)	Usually seen when K+ < 3.0 If K+ is lost slowly, body adjusts and it takes longer to see S&S Musculoskel- fatigue, weakness Smooth muscle- constipation, distention ?(from not firing) Cardiac muscle- ventricular arrhythmias- irregular pulse, EKG changes (flat T wave, big U wave) Respiratory- hypoventilation Neuro- drowsiness, decreased deep tendon reflex, confusion	**Collaborative Management** Treat cause Increase intake of K+ rich foods K+ replacement either by mouth or IV- IV potassium should be diluted and administered slowly 10-20 mEq per hour. Potassium sparing diuretics Correct Magnesium deficit	

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HYPERCALCEMIA **Calcium reg'd by PTH, presence of vitamin D, and calcitonin.** Absorbed through dietary intake. Stored in the bones. **Total-8.5-10.5 mg/dL** **Ionized-4.5-5.5 mg/dL** **Functions** Forms bones and teeth Role in cell wall permeability Sedative effect on nerve cells Role in contraction of muscles Promotes blood clotting **Regulation of Calcium** With high serum calcium, secretion of PTH is inhibited, and thyrocalcitonin is secreted inhibits reabsorption, inhibits vitamin D release, and increases renal excretion of calcium With low serum calcium, PTH secreted which causes calcium to be resorbed from bone and stimulates vitamin D, which increases absorption of calcium from the intestine.	Increased intake- antacids Decreased excretion- renal failure Increased bone resorption- malignancy, hyperparathyroidism, immobility, fractures, bone metastisis hemoconcentration- dehydration, adrenal insufficiency Acidosis (as ph drops less Ca++ binds to protein)	**excitable tissue less sensitive to normal stimuli, faster clotting times** lethargy, weakness, nausea, vomiting bone pain increased risk of dig toxicity confusion	**Collaborative Management** •treat cause •IV normal saline rapid infusion to increase renal excretion •Low calcium diet •Calcitonin or other meds •Parathyroidectomy if increased calcium due to hyperparathyroidism •Hemodialysis	**Primarily combines with phosphorus to form bones and teeth.** Ca++ and P inverse Binds to albumin. Ionized value drops. If albumin lvl drops then total Ca++ lvl drops. Metabolic acidosis(low pH) ↓ calcium binding to albumin ↑ ionized value Citrate binds to Ca++ Blood transfusion ↓ ionized value
HYPOCALCEMIA	Poor calcium intake Chronic diarrhea- interferes with absorption Renal failure Decreased PTH secretion, increased calcitonin secretion Alkalosis (as ph rises more calcium binds to protein) Increased serum albumin level Multiple blood transfusions- citrate binds with calcium glucocorticoids decrease calcium absorption in the intestine	Tetany- positive Chvostek (face) sign, positive Trousseau (arm) sign Poor blood clotting Weak cardiac contractions Muscle cramps Numbness and tingling of fingers	Oral calcium-calcium carbonate, calcium citrate IV calcium-Calcium chloride, calcium gluconate- give slowly Increase absorption- vitamin D, aluminum hydroxide Decrease nerve and muscle excitability- Magnesium sulfate, diazepam	Giving IV normal saline rapidly will increase renal excretion of calcium.