FVE HYPERVOLEMIA (ISOTONIC)	CAUSE	S/S	INTERVENTION	TEACHING
excessive fluid in ECF compartment osmolarity remains normal only ECF expanded –no fluid shift Due to fluid overload or diminished homeostatic mechanisms diff. between sys & dia.	poorly controlled IV therapy, renal failure, long-term corticosteroid therapy Tube feeding w/ lots of oral meds (flushing) **Risk factors:** heart failure, renal failure, cirrhosis of liver **Contributing factors:** excessive dietary sodium or sodium-containing IV solutions **PERFUSION ASSESSMENT** Fluid Excess pulse rate ↑ pulse quality bounding peripheral pulses full BP ↑ pulse pressure ↓ central venous pressure ↑ distended neck and hand veins engorged venous varicosities S3 gallop – indicates Hf edema	Clinical signs req immediate action Dyspnea Crackles Tachypnea Bounding rapid pulse Hypertension Distended neck veins Edema (if edema severe it's call anasarca) Ventricular gallop Clammy skin Early ↑ wt ↑ BP ↑ breath effort progress to moist breaty sounds **OXYGENATION ASSESSMENT** ↑rate shallow respirations dyspnea increases with exertion or in the supine position moist crackles present on auscultation **NUTRITION/ELIMINATION** ↑ motility Weight gain Anorexia Nausea & vomiting ↑ urine output nocturia **COGNITION/NEURAL** altered level of consciousness headache visual disturbances skeletal muscle weakness paresthesias	**Fluid Excess** Diuretics **Medical management:** directed at cause, restriction of fluids and sodium, administration of diuretics **Nursing Management:** I&O and daily weights; assess lung sounds, edema, other symptoms; monitor responses to medications- diuretics 1.Promote adherence to fluid restrictions, patient teaching related to sodium and fluid restrictions 2.Monitor, avoid sources of excessive sodium, including medications 3.Promote rest 4.Semi-Fowler's position for orthopnea 5.Skin care, positioning/turning	•avoid salt if indicated •monitor a daily wt •monitor daily fluid intake and limit if indicated
FLUID VOLUME EXCESS HYPOTONIC OVERHYDRATION •↓ osmolarity of ECF • ↑ hydrostatic pressure • excessive fluid moves into the ICF space • all body fluid compartments expand	CHF, poorly controlled IV therapy, irrigation of wounds and body cavities w/ hypotonic solutions, SIADH Tap water enemas	polyuria, diarrhea, nonpitting edema, cardiac dysrhythmias associated with electrolyte dilution, projectile vomiting Cerebral edema	**Loss of solutes** tx with hypertonic fluid	

CONDITION/DISEASE PROCESS	FLUID AND ELEC IMBALANCE
AIDS F&E result from underlying infections or cancers that invade lungs, bone marrow, brain. F&E also effected by Tx's and/or malnutrition (mostly r/t GI effects).	**yponatremia** common – adrenal insufficiency, V&D **Hyperkalemia**- rx trimethoprim (Primsol) impairs renal excretion **Hypokalemia**- persistent vomiting, renal loss **Hypocalcemia**- poor absorption of vit D **Metabolic alkalosis –** persistent vomiting, renal conservation of H+ to compensate – more K+ lost **Hypovolemia Isotonic FVD –** • V&D, • fever- increased metabolic waste which requires fluid for excretion. Also c hypercapnia so extra fluid loss from lungs (more dehydration then isotonic • Decreased fluid intake (dehydration) • Weight loss and subsequent loss of fluid (isotonic I believe)
ACUTE RENAL FAILURE Affects nephron. Kidneys lose ability to excrete fluid, electrolytes, waste, and acid-base products. Kidneys can produce urine in normal amounts or adequate concentration Leads to • anemia, • hypertension, • uremia (build up in blood of stuff normally eliminated in urine, produces a severe toxic condition and usually occurs in severe kidney disease) • Osteodystrophy (defective ossification of bone usually assoc w/ disturbed calcium & phosphorus metab) **Czd by** **intrarenal**: kidney damage (aminoglycosides, NSAIDs transfusion rxn, postpartum eclampsia , heavy metals, nephrotoxins, trauma) **prerenal**: ↓ renal perfusion -Heart Failure, hypovolemia, renal vascular obstruction, peripheral vasodialation, serious CVD, trauma, severe vasoconstriction) **Postrenal**: back up- prostatitis, obstruction, trauma **ARF has 3 phases:** **Oliguric- Anuric phase (1-2 wks):** ↓GFR, ↓Urine, ↑BUN and Creatinine = uremia **Diuretic Phase (about 10 days):** gradual ↑ urine and BUN stabilization **Recovery Phase (few months):** begins when F&E start to stabilize	**Hyperkalemia –** occurs w/ oliguria or anuria cuz K+ not excreted. ↑K+ can als czd by additional stressors : infxn, gi bleed, trauma, sx. Can worsen w/ metabc acidosis (K+ moves from ICF to ECF). **Hypocalcemia** - ↓activation of vit D by kidneys so ↓ gi absorb. Pt at high risk tetany and seizures. HypoCa+ can cause hyperparathyroidism (constant excret PTH to combat imbalance can cause PTgland to hypertrophy). And you'll get hyperphosphatemia because Ca+ and P are inverse, which will intensify hypoC **Hyperphosphatemia** – kidneys can't excrete **Hyper Mg++** - retain Mg++ from ↓ GFR or damage to tubules. Or from externa sources – laxatives, antacids, iv fluids, hyperailimentation (excessive nutrients) **Hyponatremia** – retention from ↓GFR and damaged tubules, metabolic acidos **Metabolic acidosis - common** Kidneys lose ability to secrete H+ Kidneys fail to hold onto **Metabolic alkalosis – rare** Excessive admin of HCO3 to correct acidosis **Hypervolemia** Body can't excrete excess fluid and electrolytes (isotonic) Fluid intake exceeds output (overhydration) **Hypovolemia** Diuretic phase - ↑urine not matched by replacement

CONDITION/DISEASE PROCESS	FLUID AND ELEC IMBALANCE
BURNS **<u>Imbalances result from:</u>** • alterations in skin integrity and internal body membranes • effect of heat (both source of burn and metabolic) on body water • cellular destruction resulting in water and solute losses • type and severity of imbalances depends on burn type and depth, and amt of body surface area (BSA) , and burn phase • czd: thermal, electrical, mechanical, radiation (therapy or sunburn), chem. **<u>3 burn phases</u>** **Fluid accum phase (36-48h):** • vascular and cellular loss of fluid to interstitial space – third space shift • edema • circulation compromised **Fluid-remobilization phase (diuresis phase) (> 48h):** • fluid shifted back to vascular space • ↓edema, ↑ blood flow, ↑ urine **Convalescent phase** • Healing of burn wound • Major fluid shifts resolved	**yperkalemia** – during fluid accum phase K+ released from destrd J cells → vascular space HypoK+ during fluid remobilization phase K+ shifts from ECF to cells **Hypocalcemia** – Ca+ moves from vascular → damaged tissues (blood clotting) **Hyponatremia** – ↑ loss of Na+ and H2O from cells. Na+ trapped in edema flu during fluid accum phase. Na+ also lost during fluid remobilization phase. Hypernatremia – aggressive use of hypertonic Na+ fluid replacement **Metabolic acidosis –** fixed acids from damaged tissues accum **Respiratory acidosis–** inhalation burns (inadequate ventilation) **Hypervolemia** Occurs during fluid remobilization phase Fluid shifts back to vascular Poss excessive iv fluid admin **Hypovolemia** Czd by 3rd space fluid shift 10% plasma volume lost into tissue w/ severe burn ↓skin integrity/ability to prevent water loss Potential for blood loss
CIRRHOSIS • Chronic • Destruction and subsequent fibrotic regeneration of hepatic cells (widespread) • Liver structure and vasculature altered • Portal hypertension and subsequent seepage of blood (containing lots of protein so H2O pulled from intestinal surface along w/ it) to peritoneal cavity (ascites) • Arterial volume ↓ so kidneys respond as if underperfused • Edema also results bcuz of increased pressure on vena cava from enlarged liver and ascitic fluid and from hypoalbumeria Cz'd by various hepatitis's, toxins, alcoholism, sarcoidosis (chronic disease, unknown cause, characterized by formation of nodules resembling true tubercles especially in the lymph nodes, lungs, bones, and skin—see *Boeck's disease),* chronic inflammatory bowel disease	**Hyperkalemia** – potassium sparing diuretics or K+ supplements **Hypokalemia** – renal losses of K+ and/or hyperaldosteronism **Hypocalcemia** – inadequate storage of vit d in liver so can't absorb Ca++ **Hyper Mg++** - loss through vomiting **Hyponatremia** – more H2O retained the Na+. May result from ↑ADH secretio **Respiratory acidosis -** Ascites ↓s ventilation – CO2 ↑ **Respiratory Alkalosis** ↑ammonia stims hyperventilation - ↓ CO2 **Metabolic alkalosis –** Associated w/ K+ sparing diuretics Possible vomiting and ng suction **Hypervolemia** Excess of total body fluid (edema and ascities) H2O and Na+ retention (but relatively more H2O)