Vesalius SCALpel™ : Fluids and electrolytes

Volume

water adult M 60% of body wt. (70Kg=42L), F 50%, decreases with age; ICW 40%, ECW:
- 16% interstitial, 4% plasma (2.8L), 3% trans-cellular (gut)
- 60% tot (40 intracell, 20 extra {15 interstit, 5 IV})

daily intake: 2.5L water, 50-150mEq Na (1-2mEq/kg/d), 50-80 meq K (0.5 mEq/d)
daily water out: 800cc insensible, 500 urine, 200 fecal
adult maintenance: D5 1/4% NS, 100cc/h + 20meq KCl: 96meq Na, 146 Cl, 50 K (~1meq/Kg/d), 125g Glu
normal saline has higher chloride (154) than LR (110)
- predisposes to hyperchloremic metabolic acidosis
- compensatory respiratory alkalosis requires higher minute volume, make weaning more difficult

ECF deficit: GI: vomiting, NG, diarrhea, fistula; burns, peritonitis, intestinal obstruction, fever; decreased level of consciousness -> coma, ileus, decreased pulse pressure, tachycardia, hypotension, oliguria, loss of skin turgor
ECF XS: iatrogenic, renal failure; circulatory overload, increased CVP, pulmonary edema, gallop, peripheral edema -> anasarca
fluid shifts primarily reflect Na concentration: plasma osm.= 2[Na]+(glu/18)+(BUN/2.8); nl 290-310
ECF vol deficit and hyponatremia from free water and isotonic fluid loss
ECF vol deficit and hypernatremia: mannitol use, diabetic acidosis (osmotic clearance from glucosuria)
ECS XS and hyponatremia: renal failure being treated with hypotonic fluid
ECS XS and hypernatremia: XS Na intake
creatinine clearance: normal 90-140mL/min
fractional excretion of Na (FENa): < 1% probably pre-renal, > 1% renal, > 4% post-renal/obstructive
BUN/Cr ratio: BUN rises more than creatinine in dehydration, both rise together in renal disease
- normal ratio 10-20:1; higher indicates dehydration

aldosterone: secretion stimulated by hypovolemia, hyponatremia, hyperkalemia; receptors in right atrium (hypovolemia), juxtaglomerular apparatus
- hypovolemia decreased renal perfusion, stimulates JG to secrete renin, renin cleaves angiotensin to angiotensin I to (angiotensin converting enzyme) angiotensin II which stimulates aldosterone secretion from adrenal cortex resulting in Na and water retention and K loss

ADH/vasopressin: from posterior pituitary responds to intracranial osmoreceptors to hyperosmolar plasma, right atrium hypovolemia receptors
- osmolar effect: increases renal collecting tubule permeability to water resulting in resorption of free water (no effect on Na reabsorption)
- pressor effect: vasoconstriction

Electrolytes

normal values:
- cations:
  - Na^+ 135-145 (~140)
  - K^+ 3.5-5
  - Ca^{++} total 8.9-10.3 (~10), ionized 4.6-5.1 (~5)
  - Mg^+ 2
- anions:
  - Cl^- 97-110 (~100)
  - HCO_3^- 20-29
  - PO_4^- 3.5
  - SO_4^-

- total protein 6-8.4
- organic acids
- BUN 8-25
- Cr 0.6-1.5

normal serum osmolarity 280-300
kidney exchanges Na^+ for K^+ and H^+ in distal tubule under control of aldosterone
- kidney can conserve almost all Na filtered

mixed volume and concentration most common abnormality, usually iatrogenic
hypernatremia: restless, delerium (cerebral edema), tachycardia, dry mucous membr., oliguria, fever
- etiologies: **diabetes insipidus**, head trauma, hypothalamic injury, lack of ADH/vasopressin, thiazides, lithium, hypercalcemia, polyuria, excessive $NaHCO_3$ during code
- goal to keep ICP <20, CPP >60-70
- cautious replacement of free water defecit (FWD):
  - FWD=total body water (KgX0.6)-actual body water (TBW X {140/serum Na^+})
    - e.g. 70Kg wt, 42L normal total body water X (140ideal/156actual Na) = 37.7 actual total body water, 42-37.7 = 4.3L free water deficit
  - replace half 1st 24h; replace ADH w desmopressin/DDAVP (parenteral, intranasal)

hyponatremia (<136mmol/L, Na^+ <120):
- asymptomatic unless precipitous change, then muscle twitching, convulsions
- most hypoosmolar, water retention more likely than Na loss: hypovolemia
- stimulates ADH secretion-> water retention, secondary volume expansion, dilution
- cerebral salt wasting (**CSW**) in trauma:
  - brain naturetic protein (BNP) causes increased renal loss of Na
  - dilute high volume urine
  - decreased circulating volume, elevated BUN/Cr, K normal or elevated
  - hypo-osmolarity results in brain edema, seizures
  - treat with Na and volume

syndrome of inappropriate ADH (**SIADH**) in trauma:
ADH secretion despite hypo-osmolality (vs normal secretion in response to hyperosmolality and hypovolemia)
hyponatremia with normal volume which differentiates it from CSW
absence of peripheral edema, dehydration (in the absence of adrenal, thyroid or renal dysfunction
normal volume and Hct, low BUN, normal K^+
very concentrated low volume urine
treat with water restriction
effects of mild hyponatremia: headache, nausea, vomiting, muscle cramps, lethargy, restless, disoriented, depressed reflexes,
effects severe/sudden drop: cerebral edema, increased ICP -> hyperreflexia, convulsion, seizure, coma, brain stem herniation, death, Cushing response (hypertension, brady), oliguria, aneuria
treatment: NS volume resucitation, water restriction
kidney corrects water overload
3% normal saline slowly if severe

hyperkalemia: (98% intracellular), small change in concentration affects heart
acidosis shifts H^+ into cell, K^+ from cell to serum
causes: renal insufficiency, increased intake, tissue breakdown (crush, mesenteric ischemia), decreased aldosterone
pseudohyperkalemia: hemolysis, extreme leukocytosis, thrombocytosis
effects: nausea, vomiting, abdominal pain, weakness, paralysis, paresthesias, peaked T, wide QRS, V-fib, asystole
treatment: D/C intake of K, calcium gluconate, glucose/insulin, $NaHCO_3$, diuretics (K wasting), dialysis

hypokalemia:
causes GI loss, diuretics, alkalosis, renal tubular acidosis, diuresis, corticosteroids, inadequate intake, VIPoma, hypomagnesemia (renal K^+ loss, high-normal Mg better K homeostasis)
effects: weakness, flat T, ST depression, arrhthymias, ileus, constipation, metabolic alkalosis, predisposition to Dig tox
treatment: PO preferred (80-120meq/d); IV 10-40 meq/h centrally; EKG monitor, serial [K], slow cellular repletion, if Mg low give 30meq K/8g $MgSO_4$/d

hypermagnesemia (>2.2meq/L):
causes: renal insufficiency, antacid use, lithium, bone metastases, hypothyroid
effects: symptoms above 4meq/L, weakness, loss of deep tendon reflexes (Mg used in pre-eclampsia to decrease hyperreflexia), flaccid paralysis, hypotension, nausea, vomiting, bradycardia, torsades des pointes (V-tach with long Q-T)
treatment: Ca++ (100-200mg IV), dialysis

hypomagnesemia
causes: vomiting, diarrhea, diuretics, malnutrition, aminoglycosides, inadequate intake, malabsorption (inflammatory bowel disease), alcoholism, renal loss/wasting, association with K and Ca imbalances
refractory hypokalemia must replace Mg^+

effects: paresthesias, weakness, tremor, psychosis, dig. toxicity, arrhythmias (torsades des pointes)(renal K^+ loss), anorexia, vomiting

treatment: $MgSO_4$ IV (not PO, cathartic), monitor [Mg^+], monitor BP (drops if overshoot)

hypercalcemia (>10.4mg/dl)

causes: hyperpara, malignancy (bone mets or primary bone tumor), vit A, D toxicity, immobility (mild increase Ca^{++}), milk-alkalai syndrome, sarcoidosis

effects: fatigue, weakness, confusion, polydypsia/polyuria, headache, heart block, bradycardia, nephrolithiasis, hypovolemia (increased water loss by renal attempt to clear Ca^{++})

treatment: treat the primary cause, NS rehydration, lasix after rehydration, corticosteroids, mithramycin, bisphosphonates, calcitonin, dialysis if renal insufficiency

mithramycin: antineoplastic antibiotic inhibits RNA synthesis in osteoclasts; Ca^{++} levels start falling after 12h; IV infusion for very high Ca^{++}

hypocalcemia:

causes: hypoparathyroid, pancreatitis (saponification), hypomagnesemia, renal insufficiency, D deficit, massive transfusion (citrate Ca^{++} binding)

effects: tingling, paresthesias, tetany, laryngospasm, hypotension, seizures, prolonged Q-T, arrhythmias, Chvostek's, Trousseau's

treatment: PO Ca^{++}, D, IV Ca gluconate/Cl, monitor [Ca], replete Mg (almost always associated with Mg deficiency), replete PO_4

hyperphosphatemia:

phosphate main intracellular anion

causes: renal insufficiency, laxative abuse, hypovolemia, hypoparathyroidism, thyrotoxicosis, thyroid storm

effects: related to associated hypocalcemia treatment: hydrate, dialysis, Aluminum antacids

hypophosphatemia:

GI causes: malabsorption, diarrhea, alcoholism, antacids

renal causes: diuretics, hyperparathyroidism

redistribution: glucose/insulin infusion, respiratory alkalosis, TPN

effect: weakness, coma, osteomalacia, decreased 2,3 DPG shifts oxyhemoglobin curve to left, harder to release O_2, hemolysis (membrane instability)

treatment: Na^+ or KPO_4 IV (PO causes diarrhea), replace Ca,

selenium deficiency: myalgia, cardiomyopathy

Acid/base

management of large amount of endogenous H^+ produced daily

acidosis: H^+ moves into cell, K^+ moves out

depends on carbonic acid breakdown: $H+HCO_3 <-> H2CO_3 <-> H_20+CO_2$

resp acidosis: pH <7.35, pCO_2> 45, decreased resp drive, mechanical causes (rib fx)

10mm rise in $PaCO_2$ decreases pH 0.08

resp alkalosis: pH >7.45, pCO_2 <35, iatrogenic (vent high minute vol)

metabolic acidosis: pH <7.36, HCO_3 <22 (PCO_2 drops 1mm for each 1meq drop in HCO_3)

Two reasons for metabolic acidosis

1. HCO_3 loss (non-anion gap): diarrhea, pancreatic fistula, renal tubular disease, carbonic anhydrase diuretic (acetazolamide)
2. lactic acidemia (anion gap): cardiogenic shock, sepsis, hypovolemia, low flow, ischemia (mesenteric), ketoacidosis, renal failure
   A. hypoxic: hypoperfusion, pulmonary disease, anemia, carbon monoxide
   B. non-hypoxic: liver failure, renal failure, cancer, strenuous exercise, ethanol toxicity, low thiamine

non-anion gap metabolic acidosis (normal anion gap):

loss of HCO_3^-, reciprocal increase Cl^-

causes: renal tubular acidosis, Addison's, diarrhea, GI/pancreatic fistula, ureterosigmoidostomy, hypoaldosterone

anion gap (Na-{Cl+HCO_3}) met acidosis (elevated anion gap): normal anion gap 8-12mmol/L

metabolic increase in H^+, HCO_3 loss, no increase in Cl^-

causes of increase: renal failure, diabetic ketoacidosis, lactic acidosis, ASA, ethylene glycol (antifreeze) ingestion, methanol ingestion

metabolic alkalosis: pH >7.44, HCO_3>27; compensated by respiratory acidosis, increase PCO_2

10meq rise in HCO_3 raises pH 0.15

saline responsive causes: vomiting, NG output, diuretics, milk-alkalai syndrome, lactate infusion, citrate infusion; treat with NaCL+K

saline unresponsive causes: corticosteroids, renal artery stenosis, profound hypokalemia, hypomagnesemia; stop steroids, correct electrolytes, acetazolaminde/diamox-> clear base load