Electrolyte Repletion, bowel regimen, and other hum-drum Tasks **2024-08**

- **Electrolyte repletion is boring but easy. Following is a guide to how I'd like it to be done (and why):**
  - **hypokalemia (eg K 3.8)**
    - clinical importance
      - for cirrhotics, decreases risk for encephalopathy (low K => ^NH3)
      - low K (eg low 3's) impairs gut motility, promotes constipation/ileus
      - in pt's with HF, cardiomyopathy, hx or risk for arrhythmias, low K is dangerous; it also inhibits the ENAC channel which impairs diuresis, promotes metabolic alkalosis
      - for pt's being diuresed, keeping K up lets you proceed with diuresis without worry
        - if you want to give lasix twice a day, you need to check K/Mg in early afternoon to allow you to get the result back and replete before you give the second dose
    - how to replete
      - check (add on Mg to the lab with the low K) and replete the Mg if <1.9
      - at the same time, start repleting K
      - **rule of thumb: 10meq needed to raise K by 0.1 (unless pt has significant CKD!)**
      - **always use PO unless can't take PO or truly dire hypokalemia** (eg K 2.8 and/or PVC's/NSVT on tele)
      - eg K 3.2 => 80meq => Kdur 40 PO q3 x2 doses (much better than KCl IV x3 runs!)
      - if K was significantly low (eg K <3.5) then repeat Renal/Mg a few hours after you've repleted (it often will need more repleting, esp in refeeding syndrome or diuresing)
  - **hypomagnesemia (eg Mg 1.7)**
    - clinical importance
      - for AWS, low Mg exacerbates AWS as well as increases risk for seizures
      - in pt's with HF, cardiomyopathy, arrhythmias, low Mg is dangerous (in fact, Mg IV can be useful even if Mg is not low in eg Afib with RVR, etc)
      - Low Mg causes Low K which is resistant to repletion
    - how to replete:
      - Mg 1.6-1.9 => Mg 2gm IV (if Mg 1.8-2 and chronic/refractory, then can just go with oral)
      - Mg <=1.5 => Mg 2gm IV q2 x2 and repeat Renal/mg in 6 hrs
  - **hypophosphatemia (eg Phos <2)**
    - clinical importance
      - severe hypophosphatemia (eg low 1's and esp <1) can be fatal or have dire complications (respiratory failure, coma, severe peripheral and CNS damage, shock)
    - if phos and other lytes are dropping markedly then think refeeding syndrome and be aggressive, go with IV phos infusion and repeat Renal/K/Mg labs several times a day (in extreme cases, consider making them NPO!)
    - how to replete
      - Phos 2 is merely billable (not particularly important) so be sure to mention in your notes ("hypophosphatemia") and give oral phos repletion
      - if Phos < 1.7 and falling fast, then bad things may be on the way. Phos 15mmol infusion and repeat Renal/Mg in 6hrs. Note that Phos infusion contains 250cc saline and takes 4hr to run
- **Bowel Regimen**
  - clinical importance
    - constipated people don't eat much, become weaker. As it progresses they may develop abd pain, nausea/vomiting -> aspirations and eventually ileus/PSBO's, other drama
    - adds hours to your day when these things happen, and makes your patient miserable!
  - being in the hospital is constipating. Dry air, mushy food, acute illness, pain, lying in bed all day, electrolyte disturbances and medication side effects
    - almost everyone should be on a bowel regimen from day 1 (eg miralax daily and senna 1-2 tabs qhs). **Add hold parameters** "hold for diarrhea, pt may refuse".

        - **never rely on "PRN constipation"** meds. They will never be given!
    - follow exam (listen to and touch the belly) and act appropriately
    - if you've fallen behind (eg, day 1 on service you will often inherit a number of patients with distended, tender bellies), you will probably need a jump start. Can try dulcolax PO or suppository (which may work faster than PO), but frail/elderly or tenuous patients probably do better with mechanical methods first
        - rectal exam to r/o impaction
        - serial enema until bowels start moving
- **Elevated BP**
    - clinical significance
        - think of this as data more often than a target for therapy!
        - no one in the hospital has accurate BP measurements as far as typical notions of Hypertension are concerned. This isn't the context where you should be thinking "I need to treat to target to prevent cardiomypoathy or CKD"
        - treating inpatient BP's by uptitrating meds is more likely to cause harm than benefit
            - IV hydralazine (never!) -> watershed stroke or AKI; Discharge on new script for chlorthalidone or increased doses of their outpt meds -> AKI or fall with hip fracture a few days after discharge
    - Assess thepatient before thinking about treatment
        - is it elevated? Repeat it yourself and make sure it's real (eg cuff is right size, pt not flexing, etc)
        - think "why is BP elevated"
            - medications: is there a medication that they didn't get (incomplete med rec, off the floor when meds were being given out, etc)? Is a q8 or q12 med being given "TID" or "BID"?
            - pain, anxiety, agitation, etc. Treat these rather than reach for labetolol!
            - is the patient volume overloaded (eg decompenstated CHF or CKD/ESRD)? Maybe needs higher dose of lasix or need to talk to Renal about getting more fluid off at next HD
            - *if* pt doesn't have regular med f/up and you have good evidence that they in fact have uncontrolled HTN at baseline, you can decide whether starting on a good, guideline-directed BP regimen is indicated (in which case, don't over-do the dosing; their BP's are probably not as high at home as they are here!).
        - ***if* the BP is *so* high (eg >180/120) that it is conceivably causing acute harm (hypertensive emergency), then pt likely needs a stat consult to the relevant specialty and should be upgraded to A4/CCU/ICU for IV infusion therapy (eg clevidapine)**
            - **in my experience, HTN emergencies are uncommon, and usually the damage is chronic (needs management of chronic disease) rather than acute**. If chronic, then the patient needs treatment of their underlying problem rather than (or in addition to) a clevi drip
                - eg ischemic stroke is the cause of an acute spike in BP, rather than an acute spike in BP (why would that happen?) is causing a hemorrhagic stroke. Stroke code not Clevi.
                - decompenstated CHF requires decongestion, rather than acute spike in BP leading to CHF. Diurese!
                - progressing CKD (eg CKD 5+) needes intensified BP meds/diuresis, rather an acute spike in BP is causing AKI