Case-based Approach to Blood Work Interpretation

This will be an interactive session. Cases will be used as starting points for greater discussion on general ways to approach blood work, common issues or often-forgotten principles

1. General ways to approach blood work
    a. Biggest outlier
    b. Patterns
    c. Differentials for analytes and see where they overlap
    d. Once you have a "working diagnosis" go back to the blood work and see if it explains all the findings
2. Common issues
    a. <u>Anemia interpretation</u>
        i. Determine severity of anemia.
            1. <u>General</u> guidelines for dogs and cats (for this class; degree is often patient/situation-dependent):
                a. Mild – Dogs ≥ 33%; Cats ≥ 26%
                b. Moderate – Dogs ≥ 24%; Cats ≥ 23%
                c. Marked – Dogs ≤ 23%; Cats ≤ 23%
        ii. Evaluate **plasma or serum protein** (as well as albumin & globulin which comprise the total protein).
            1. When low (especially if albumin & globulin low together), it suggests blood loss
            2. Normal total protein level argues against blood loss
        iii. **Regenerative status** (number of reticulocytes)
            1. Regenerative ➔ Blood loss or Hemolysis
            2. Non-regenerative ➔ Decreased production (although sometimes cats lie)
            3. Of course, not horses!
            4. It could be pre-regenerative (takes 2-3 days to see response following cause of anemia)
        iv. Patient information
            1. Age – young animals have lower PCVs (mild) and may have a decreased MCV (foals)
            2. Breed – any possibility of an inherited disease?
            3. Questions to ask:
                a. Duration of clinical signs, periodic or persistent signs, recent trauma or surgery, indoors vs.outdoors, tick/flea preventative, diet (commercial or prepared), exposure to chemicals/toxic agents, indiscriminate eater (pennies, paint), current medications
        v. RBC morphology
            1. Look for evidence of: regeneration, iron deficiency, hemic parasites, evidence of oxidative injury,immune-mediated RBC

targeting (see further along in notes for more information)

vi. RBC indices
    1. If normal, may suggest anemia of chronic disease or not be relevant
    2. Macrocytic, hypochromic + reticulocytosis = regenerative (but does not indicate cause)
    3. Microcytic, hypochromic = iron deficiency
    4. Elevated MCHC – **may** support intravascular hemolysis (this is not an etiology though)

vii. Serum bilirubin
    1. Increases can be due to hemolysis (although hyperbilirubinemia can also develop secondary toblocked bile canaliculi or bile ducts - cholestasis, anorexia in horses, inflammation in cats)

viii. BUN, creatinine, USG
    1. Non-regenerative anemia + azotemia + isosthenuria (USG 1.008-1.012) supports a lack of EPO dueto renal failure; azotemia, however does <u>not</u> necessarily equal renal failure (see section 2)

b. <u>Interpretation of azotemia and USG</u>

c. Impaired renal concentrating ability (unrelated to renal disease)

There are some diseases/conditions that impair renal concentrating ability but are unrelated to decreased numbers of functioning nephrons. These include:

i. Hypoadrenocorticism (Addison's disease)
    1. Lack of aldosterone ➔ loss of NaCl + H_2O
        a. Dehydration results, as can azotemia (decreased GFR from dehydration)
        b. Inadequately concentrated USG (from a lack of aldosterone)
        c. Support – look for a lack of a stress leukogram, hypercalcemia, low/normalglucose, etc

ii. Medullary washout, i.e. due to decreased medullary tonicity
    1. Prolonged hyponatremia and hypochloridemia
        a. Causes: Hypoadrenocorticism, use of loop diuretics, prolonged diuresis (fluidtherapy, osmotic diuresis, psychogenic polydipsia)
    2. Low urea (low BUN)
        a. Causes: Hepatic failure, portosystemic shunt

iii. Diabetes insipidus (either central where ADH not produced or nephrogenic where distal renal tubules unable to respond to ADH)
    1. Nephrogenic causes – hypercalcemia, E¡.coli. infections (UTI, pyometra), hypercalcemia, steroid administration, prolonged hypokalemia, certain drugs, avariety of others

2. Central – idiopathic, injury, neoplasia, inflammation, etc

iv. Solute diuresis
    1. Too much solute increases the flow rate and decreases reabsorption (along withother mechanisms
        a. Diabetes mellitus, mannitol administration
        b. Can lead to medullary washout