EQUINE BLOOD TRANSFUSION - WHAT YOU NEED TO KNOW TO GET THE JOB DONE.
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1. Blood products
   a. Whole blood
      o No commercial blood banks for horses
      o Almost all blood is collected from donor immediately before use
   b. Plasma
      o Usually acquired frozen from a commercial source
      o Uses:
         o failure of passive transfer
         o colloid support in FOALS (need too much volume for adults, $$$)
         o replacement of coag factors (fresh-frozen (FFP) only)
      o “Specialty” plasmas (commercially available) come from animals hyper-immunized against specific pathogens:
         o Hi-Gamm: high IgM, for FPT in foals
         o J-5: *E. coli* cell wall (endotoxemia)
         o anti-botulism
         o anti-Rhodococcus
         o anti-West Nile Virus
   c. Washed RBC
      o Rarely used, produced at point-of-care
      o ideal for foals with *Neonatal isoerythrolysis*
   d. Packed RBC
      o Rarely used, produced at point-of-care
      o Ideal for animals with hemolysis (such as *Red Maple Toxicity*) that do not need extra volume (plasma)
   e. Platelets
      o Rarely used, produced at point-of-care
      o Can only be stored at room temperature (up to 3 days) as refrigeration causes platelets to become non-functional

2. Equine blood types
   a. seven blood groups: A, C, D, K, P, Q, and U
   b. each *group* can have multiple *factors*. For example, horses with blood group A can have factors a, b, c, d, e, f, g. These factors are surface markers on the RBC.
      o So each horse has a blood type such as Qa or Pd…
      o 400,000 combinations!
   c. Antibodies
      o Anti-RBC antibodies are typically acquired by exposure (usually pregnancy - see *Neonatal isoerythrolysis*)
      o anti-Ca antibodies may naturally occur, and cause mild agglutination reactions, not usually clinically *significant in vivo*
As horses age, they seem to develop more allo-antibodies, possibly due to cross-reaction with ingested protein, exposed by diet? (Dr. Sean Owens, personal communication)

3. Cross-matching

Horses have minimal naturally occurring anti-RBC antibodies and often do not make RBC antibodies until they have been administered blood multiple times, transfusions can usually be performed **without crossmatch** if necessary, as long as you are careful to watch for transfusion reactions and get informed consent from the owner.

a. Major crossmatch - same as small animal, human  
   - Donor erythrocytes are washed and incubated with recipient serum
b. Minor crossmatch crossmatch - same as small animal, human  
   - Donor serum is incubated with washed recipient erythrocytes
c. Types of crossmatch reaction  
   - Agglutination  
     - caused by agglutinins
   - Hemolysis  
     - caused by hemolysins
     - detected after adding complement to the reaction mixture
     - technically quite difficult, usually only performed in large hospitals/labs
d. Storing blood samples for crossmatches.  
   - As many large hospitals maintain a blood donor herd, it would make sense to store samples from these donors in the lab so that crossmatches can be performed without re-phlebotomizing the horses each time.
   - ***Unfortunately, a study that we did at New Bolton in 2011 demonstrates that crossmatch results are not stable on aliquots of blood stored for as little as a week.***

4. Blood donor selection, blood collection  
   a. Ideally Qa and Aa antigen and antibody negative  
      - Usually Standardbreds and Quarterhorses  
      - “universal donor”?  
   b. Usually large, quiet gelding (or maiden mare), PCV >35%  
   c. Needs to be UTD on vaccines, anthelmintics, and especially testing for blood borne disease (i.e. Coggin’s negative).  
      - Aseptically place a 14g (or even 10g) catheter ideally going up the neck, and connect to blood collection device.
      - Remove up to 15-18 ml/kg (**so approx 8-10L for a 500kg horse**)
      - Replace with 10-20L of crystalloid.
      - Can usually collect every 3 weeks or so.
   d. Collection systems
5. Equine blood product administration
   a. Normal blood volume for an adult horse is ~8% of bodyweight (80ml/kg)
   b. Blood deficit can be calculated using an intuitive formula:

   \[(\text{normal PCV} - \text{current PCV})/\text{normal PCV} \times 0.08 \times \text{bodyweight (kg)}\]

   o However, many horses who have had *acute hemorrhage* will still have a normal PCV; typically the plasma protein will drop before the PCV
   o Assuming donor blood is ~ PCV 40%, every 2.2 ml of whole blood/kg body weight will increase the PCV by 1%

c. Indications for whole blood
   o Anemia with clinical signs
     o tachycardia (HR>60 bpm for an adult)
     o colic (low GI perfusion)
     o dullness, depression, anorexia
     o usually occur when PCV <12-15% if acute loss, may get as low as 9-10% with chronic anemia before signs are seen

d. Transfusion technique
   o Same as small animals, humans
   o Replace transfer set every 4 liters
   o Monitor for signs of reaction (increased rectal temperature, piloerection, sweating, tachycardia, colic, diarrhea)
     o TPR every 2 minutes for first 30 minutes, then every 15 minutes until transfusion is complete
   o Treat reactions by stopping transfusion, giving crystalloid fluids and epinephrine if necessary (1-5ml of 1:1,000 epinephrine SQ or 1-2ml IV in an average 450kg horse)

e. Transfused RBC lifespan
   o Normal RBC lifespan is 150 days
   o **Half-life of 20-30 days donated RBCs**
     o too many blood-types for “perfect” matching
     o takes 5 days to mount an immune response
     o **Bone marrow response is essential**
   o ~50 days for autologous re-transfused blood

6. Blood storage
   a. Equine blood is usually collected at the time of use and not banked.
   b. Can be stored for 28 days, but red cell half-life drops a little.
     o CPDA-1 best, glass bottles worst for storage
     o Use same storage techniques as small animal
Diseases requiring blood transfusion

A. Neonatal isoerythrolysis

- Neonatal foal develops severe intravascular hemolysis due to colostral antibodies
- Most common in *multiparous* Standardbreds and mules

1. Pathophysiology
   a. Mare is bred to stallion with different blood type
   b. She becomes sensitized to foal antigens in utero
   c. She makes antibodies against foal’s red cells
   d. Foal ingests colostrum with anti-RBC antibodies
   e. Type II hypersensitivity reaction → severe hemolytic anemia

   - Qa antigen and Aa antigen are responsible for most cases
     a. Therefore mares that are negative for Qa and/or Aa antigens are at highest risk for having an NI foal.
       o ~19% of Thoroughbreds and ~17% of Standardbreds
     b. Occasionally other groups (Dc, Ua, Ab and Pa)
     c. Mules (horse dam, donkey sire) have very high incidence of NI because of DONKEY RBC ANTIGEN; therefore 100% of matings are incompatible.

<table>
<thead>
<tr>
<th>Breed</th>
<th>Qa(-)</th>
<th>Aa(-)</th>
<th>Incidence of NI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoroughbred</td>
<td>39%</td>
<td>15%</td>
<td>0.05%</td>
</tr>
<tr>
<td>Standardbred</td>
<td>100%</td>
<td>44%</td>
<td>2%</td>
</tr>
<tr>
<td>mule</td>
<td>N/A</td>
<td>N/A</td>
<td>8-10%</td>
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2. Clinical signs
   a. NORMAL AT BIRTH
   b. Hemolysis becomes clinically apparent from 12 hours to 5 days post-birth foal
   c. Dull, lethargic
   d. Tachycardic
   e. Icteric
   f. Variable hemoglobinemia, hemoglobinuria, anemia

3. Treatment and Prevention
   a. Transfuse if necessary (tachycardia, dullness, PCV <10-15%)
      o Whose blood?
         1. WASHED dam’s RBC
         2. whole blood from an unrelated gelding
   b. Prevent by:
      o Not re-breeding to stallion with incompatible blood type
Cross-matching mare and stallion during late pregnancy to see if mare is producing antibodies

Performing a jaundice foal agglutination test, which is a stall-side cross-match that looks for agglutination between mare colostrum or serum and foal blood.

1. If any of these are positive, withhold colostrum from foal (can be difficult!)

B. Red maple toxicosis

Ingestion of wilted leaves and branches from *Acer rubrum* (Red maple)

a. Aka swamp of soft maple
b. Eastern USA
   i. Typically seen in summer and autumn
   ii. After winds or storms (blow downs)
   iii. After trees have been felled
   iv. Yard mulch and trimmings have been dumped in the field.

1. Pathophysiology
   o Toxic principle gallic acid?
   o Toxic dose 1.5-3 gm/kg body weight (0.7-1.5 kg for 450-kg horse)
   o Causes severe intra- and extravascular hemolysis and methemoglobinemia

2. Clinical signs
   o Dull, lethargic
   o Tachycardia
   o Pale or muddy brown (methemoglobin) mucous membranes
   o Massive anemia, hemoglobinemia, hemoglobinuria
   o Death (~60% mortality)

3. Treatment
   o Transfuse if necessary (tachycardia, dullness, PCV <10-15%)
     • any matched donor, unmatched if first transfusion and crossmatching is unavailable
   o Crystalloids (cautiously) for diuresis (hemoglobin=nephrotoxin)
   o Activated charcoal to early cases? Usually too late for affected horses.

C. Hemorrhage

i. During nasal/sinus surgery
   o because this is anticipated, some people bank autologous blood 1-4 weeks before surgery

ii. Post-castration

iii. Uterine artery rupture, other obstetric

iv. Guttural pouch mycosis

v. Thrombocytopenia

vi. Trauma
D. Immune-mediated hemolytic anemia
   i. Usually hapten-mediated
      o penicillin, other drugs
      o neoplasia
      o Streptococcal infection
      o anything else!
   ii. Diagnose using Coomb’s test, direct immunofluorescence (DIF) flow cytometry
   iii. Treat with immunosuppression (corticosteroids) and ideally remove the inciting cause.