1. The A antigen is defined by the terminal sugar molecule _______________, the B antigen is defined by the terminal sugar molecule_____________. Both antigens are built by adding their respective defining terminal sugar molecules to the antigen _______________.

2. The color of the label on the bottle containing the commercial anti-A reagent is ____________, the color of the label on the bottle containing the commercial anti-B reagent is_____________.

3. An individual with the genotype hh se se AB has the ________________ Phenotype.
   a. His forward type would type as Group _________
   b. His reverse type would be consistent with Group _________
   c. Antibody body screen and identification is expected to be ________________
   d. However, the antigen ____________ is not present, which can be demonstrated by the lack of agglutination with this lectin ________________.
   e. Because of the presence of naturally occurring anti-______, this person can only be safely transfused with what type of RBCs?
   f. If his genotype is hh Se Se AB, his phenotype would be known as the ________________.

4. Cold agglutinin disease is most often associated with autoantibodies with specificity against the _______ antigen. Antibodies with this specificity is also associated with the _______________ infection. The _______________ and the _______________ are the most important determinants of the potential hemolytic potential of cold autoantibodies.

5. The acquired B phenotype
   a. Is the result of the defining residue of the A antigen being converted by this enzyme ___________, the resulting residue is _______________, which resembles the B antigen.
   b. Is most often seen in patient with this medical condition______________
   c. A group A person with acquired B would reverse type as ___________, forward type as ________________, and his serum would/would not react with his own RBCs
   d. Name 2 ways the acquired B antigen could be distinguished the “real B “ antigen

6. About ___% Group A individuals can be classified A2. They can make anti-A1 which can give rise to discrepancies between ABO forward and reverse typing.
   a. The anti-A1 is usually clinically significant/insignificant
   b. The lectin _______ can be used to distinguish A1 vs A2. This lectin only binds to A1 RBCs.

7. Match the lectin on the right to the antigen on the left according to their binding specificities
   A1         Ulex europaeus
   N          Dolichos biflorus
8. What do the Se and H gene products have in common? How are they different?

9. Approximately ___% of the population have the Se gene product, which means that the ABO antigen can also be found in _______________(name a bodily fluid).

10. Just to make things even more confusing, what about the Le gene product? How does it differ from the Se and H gene products?

11. What is the most likely Lewis antigen phenotype of adults who have both the Le and Se genes?
   a. Le(a-b-)
   b. Le(a+b-)
   c. Le(a-b+)
   d. Le(a+b+)

12. What is the Lewis antigen phenotype of adults who have the Le and se genes?
   a. Le(a-b-)
   b. Le(a+b-)
   c. Le(a-b+)
   d. Le(a+b+)

13. The Le(a-b-) phenotype is found more commonly in African Americans or Caucasians?

14. True or False
   a. The Le(a+b+) is commonly seen in adults
   b. Pregnant females can be transiently Le(a-b-) phenotypically
   c. Most phenotypically Le(a-b+) individuals will have naturally occurring IgM anti-Lea antibodies
   d. Lewis antibodies can occasionally cause hemolytic disease of the newborn.

15. Enough about the Lewis antigens! Let us talk about the P/P1 antigens;
   a. Anti-P1 can be neutralized by ______________________________
   b. The P antigen is also a receptor for this virus:
   c. Auto-anti-P is implicated in this immune mediated hemolytic disease:______________________________, commonly seen in patients of what age group?________________________. The antibody is ___________(IgG or IgM). The diagnostic test is called the __________________________