1. While on rounds in the medical unit you come upon a 47 y.o patient (normal renal function) with CAP that does not appear to be responding well to Augmentin 750mg BID. The attending physician wants to change the dosing regimen in order to maximize the pharmacodynamic characteristics of the drug. You suggest:

a) Increasing the interval to QD – this would, in theory, decrease the time above the MIC
b) Decreasing the interval to TID – maximize T>MIC for beta-lactams by giving the drug more often
c) Leaving the regimen the same – if not responding, a change in therapy should be made
d) Decreasing the dosage to 500mg – this would decrease the peak concentration and the AUC, neither of which is related to the most important parameter for beta-lactams (T>MIC)
e) None of the above.

2. During an overnight shift, a patient presents to the emergency room with abdominal pain and diarrhea. While on rounds the following day, the team finds out that the patient had eaten some leftover stew for dinner, and according to the microbiology lab, the pathogen is Gram-positive upon staining and has produced a double zone of beta hemolysis (like a target) on the agar plate. You suspect:

a) C. perfringens – hallmark double zone of beta hemolysis; common in food poisoning
b) Nocardia – not associated with food poisoning; no beta hemolysis
c) C. difficile – not associated with food poisoning; no beta hemolysis
d) P. aeruginosa – Gram negative; not associated with food poisoning; no beta hemolysis
e) None of the above.

3. A first year resident consults you regarding a neonatal sepsis case. The microbiology lab has informed her that the pathogen is a Streptococcus. The only other pieces of information given were that the area around the colonies was completely clear and the organism was PYR negative. You suspect:

a) S. pneumoniae – alpha hemolytic; not typical in neonatal sepsis
b) Strep Group B – S. agalactiae
c) S. aureus – staphylococcus
d) Streptococcus, Group A – PYR positive; not typical in neonatal sepsis
e) None of the above.

4. Resistance to vancomycin often occurs due to target site modification. The normal binding site of vancomycin is D-ala D-ala and vancomycin can no longer bind when the peptide is changed to D-ala D-lac OR ser.

***Note: This question is referring to vancomycin resistance in general. Please remember the differences in mechanisms for VISA and VRSA.

5. Draw a line between the microorganism and the identifying microbiological charactereristic: (Each pair must be correct to receive any credit):

- Neisseria gonorrhea -------------- intracellular G(-) diplococci
- Streptococcus pneumoniae--------- lancet-shaped pairs
- Haemophilus influenzae --------- TINY G(-) rods